DISEASES OF THE

INTESTINES

AND

PERITONEUM

BY

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EDITED, WITH ADDITIONS

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PREFACE.

THE excellence of the series of monographs issued under the editorship of Professor Nothnagel has been recognized by all who are sufficiently familiar with German to read these works, and the series has found a not inconsiderable proportion of its distribution in this and other English-speaking countries. I have so often heard regret expressed by those whose lack of familiarity with German kept these works beyond their reach, that I was glad of the opportunity to assist in the bringing out of an English edition. It was especially gratifying to find that the prominent specialists who were invited to co-operate by editing separate volumes were as interested as myself in the matter of publication of an English edition. These editors have been requested to make such additions to the original articles as seem necessary to them to bring the articles fully up to date and at the same time to adapt them thoroughly to the American or English reader. The names of the editors alone suffice to assure the profession that in the additions there will be preserved the same high standard of excellence that has been so conspicuous a feature in the original German articles.

In all cases the German author has been consulted with regard to the publication of this edition of his work, and has given specific consent. In one case only it was unfortunately necessary to substitute for the translation of the German article an entirely new one by an American author, on account of a previous arrangement of the German author to issue a translation of his article separately from this series. With this exception the Nothnagel series will be presented intact.

ALFRED STENGEL.

EDITOR'S PREFACE.

WHILE highly appreciating the opportunity of editing the American translation of Professor Nothnagel's *Diseases of the Intestines and Peritoneum*, I have, throughout, felt the responsibility of making additions to this authoritative work. The translation was made from the second edition, which appeared in 1903, and it has not always been easy to be quite certain whether facts and views which I have added had been duly weighed by Professor Nothnagel and purposely excluded. The additions are inclosed in brackets, and include fresh sections on Intestinal Sand, Sprue, Ulcerative Colitis, Idiopathic Dilatation of the Colon, and numerous interpolations in most of the sections, especially those on Intestinal Ulcers and New Growths, Peritonitis, and Appendicitis. I am indebted to Mr. D'Arcy Power for his kindness in reading and making additions to the section on Intussusception—a subject which he has specially made his own.

H. D. ROLLESTON.

LONDON, April, 1904.

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OUR knowledge of intestinal digestion is deficient and is not consistent with our knowledge of normal physiology, and still less as regards pathologic perversions. Much less is known about the chemic functions of the intestine than about these functions of the stomach. This is mainly due to difficulty in obtaining the contents of the intestines (chyme) in a satisfactory state for chemic analysis, but, in addition, the complicated nature of the chemic processes that go on in the intestine makes the solution of the problem far from easy. While the stomach-tube makes it an easy matter to examine the stomach-contents, it is only in those comparatively rare cases of intestinal fistula that an opportunity is provided of investigating in a satisfactory manner the chemic processes in the intestines during life. The feces give no information whatever about the processes that go on in the upper portions of the intestine. It is surprising, therefore, to learn that careful examinations of the intestinal contents were made long before similar examinations of the stomach-contents were undertaken. As early as 1662 Regnier de Graaf made the first experimental intestinal fistula in animals, whereas the first examination of the stomach-contents was not made until 1752, by Réaumur.

In the following paragraphs an attempt will be made to give a condensed summary of the present state of our knowledge of intestinal digestion.

The chyme which passes from the stomach into the intestine is subjected to the action of both organized and unorganized ferments. The latter are the products of the living cells of the glandular appendages of the intestine [pancreas, liver] or of the small glandular tubules in the mucosa of the intestine; the former are represented by certain micro-organisms that cause fermentation of carbohydrates and of proteids (putrefaction).

These complicated chemic processes can be studied both with the aid of artificial intestinal fistulas and by observing the changes that are brought about in the different classes of food-stuffs by the action of the various intestinal secretions. The latter method gives more instructive results, and will therefore be considered first.

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The Pancreatic Juice.—The pancreas is the most important of the above-mentioned glands. It is found in all mammals, while glands possessing similar functions to the pancreas are present in all the articulata. The albumin-freeing property of the pancreatic juice was discovered in 1836 by Purkinje and Pappenheim. Claude Bernard, in 1846, performed the first fundamental experiments with pancreatic secretion. Bidder and Schmidt, Corvisart, W. Kühne, Bernstein, and Heidenhain amplified these experiments and obtained results which are accepted at the present time.

The secretion of pancreatic juice, as was originally shown by Bernard and Bernstein, is dependent on digestion. [The introduction of 0.4 per cent. HCl into the duodenum or jejunum induces a flow of pancreatic juice which is not reflex, since it occurs after section of all nervous connections. It is due to direct excitation of the cells of the pancreas by a body provisionally called secretin (Starling and Baylis). This body is formed in the mucous membrane of the duodenum and jejunum and reaches the pancreas by the blood-stream. The acid splits off " secretin " from a precursor " presecretin," which is present in relatively large amounts in the mucous membrane of the duodenum.—Ed.]

No definite statement can be made as regards the quantity of pancreatic juice excreted in twenty-four hours. This is due to the fact that the amount secreted varies in temporary and in permanent fistulas. Dogs with a temporary fistula excrete from 2.5 to 5 grams per kilo in the twenty-four hours, while dogs with a permanent fistula have been known to excrete twenty times as much. Great variations have also been observed in the composition of the secretion. It is a clear, colorless, very alkaline fluid, sticky and odorless, and contains a sufficient amount of albumin to become solid on boiling. The percentage of solids varies as much as the total quantity, and may be from 1.5 to 11.5 per cent. The percentage of organic substances fluctuates from 0.6 to 0.8 per cent. Zawadsky, who had an opportunity of analyzing the composition of normal human pancreatic juice (in a case of pancreatic fistula following removal of a tumor), obtained the following results : Water, 86.4 per cent. ; solids, 13.59 per cent. ; organic matter, 13.25 per cent. ; inorganic matter, 0.34 per cent. Of the organic matter, 9.2 parts were proteid material and 0.83 part could be extracted with alcohol. [In a fistula of two and a half years' duration following drainage of a pancreatic cyst in a girl aged nineteen years, the fluid contained 99.32 per cent. water and 0.68 per cent. solids ; and since it had practically no tryptic or lipolytic action, was not ordinary pancreatic juice. This case, investigated by Murray and Gies, is important in showing that it must not be assumed that a fistula left after operations on the pancreas is necessarily due to the escape of pancreatic fluid.—Ed.]

The most important constituents of the pancreatic juice are three enzymes—namely, an amylolytic, a proteolytic, and a lipolytic enzyme. [Halliburton and Brodie have shown that pancreatic juice precipitates the caseinogen of milk, and Vernon has proved that, as suggested by Edkins, there is a rennet ferment in the pancreatic juice.—Ed.]

The *amylolytic enzyme* of pancreatic diastase acts in a similar manner to ptyalin, and possesses the power of converting boiled starch into maltose very rapidly at body-temperature. In addition small quantities of dextrin and traces of dextrose are formed. The efficiency of the fermentative action depends largely upon whether the reaction is acid or alkaline. According to V. Hofmeister, the maximum diastasic action is attained in the presence of 0.05 per cent. of acetic acid and 0.03 per cent. of lactic acid. According to Kröger, 1 gram of a dog's pancreatic juice converts 4.6 grams of starch into sugar (and dextrin) in thirty minutes. Cane-sugar and inulin are unaltered by pancreatic juice. According to Zweifel and Korrwin, this ferment is absent from the pancreas of new-born children. Ewald, on the other hand, succeeded in demonstrating its presence in the pancreatic juice of a puppy three days old. [H. M. Vernon finds that ptyalin and the amylolytic ferment of the pancreas are different and not identical bodies, and that the pancreatic amylolytic ferments or diastases of various animals are different bodies. It appears that pancreatic diastase is a single definite chemic substance, and thus differs from "trypsin" and pancreatic rennin. He confirms Chittenden and Griswold's observations that the activity of pancreatic diastase is increased by very small quantities of acids.—Ed.]

The *lipolytic ferment* (steapsin) has so far not been isolated. It possesses the power of splitting neutral fats into fatty acids and glycerin (Bernard and Berthelot). $C_3H_5O_3R_3$ (neutral fat) $+ 3H_2O = C_3H_5O_3H_3$ (glycerin) $+ 3$ (HOR) (fatty acid). Nencki has shown that other esters can also be saponified by this enzyme. The cleavage of the fat molecule occurs very slowly. Berthelot, for instance, allowed about 15 grams of pancreatic juice from a dog to act upon a decigram of monobutyrin, and found that it required twenty-four hours before the monobutyrin molecule was completely transformed into butyric acid and glycerin. Blank allowed 50 grams of finely chopped ox's pancreas to act on 5 grams of mutton fat for twenty-four hours, and found that in one experiment 20.7 per cent., and in another experiment 20.4 per cent., were split up into glycerin and fatty acids. The fatty acids thus freed combine with the alkali present in the intestine to form soaps. This subsequently greatly aids in emulsifying the fats (Brücke, Gad), and in this way is said to promote their absorption.

The *proteolytic ferment* was first clearly described by Kühne, who called it trypsin. The first observations on the proteolytic ferment of the pancreas were made by Claude Bernard and Corvisart. Subsequently numerous other workers have attempted to isolate the ferment in a pure state and have investigated its action on proteids (Danilewski, Hüfner, Kühne, Löw, and others). [The zymogen of trypsin was discovered by Heidenhain in 1875. H. M. Vernon found that trypsin was destroyed by sodium carbonate and showed grounds for the supposition that trypsin is not a single substance, but that there is a series of trypsins of varying degrees of stability, the more unstable being destroyed first by sodium carbonate and the more stable slowly. He also believes

that there is an insoluble prozymogen in the pancreatic tissue which yields definite amounts of soluble trypsin-zymogen and of soluble rennetic zymogen ; and that there is also an insoluble diastatic pro-zymogen which yields soluble diastatic zymogen.—Ed.]

This enzyme acts on proteids in alkaline or neutral medium. A markedly acid reaction inhibits, while slight degrees of acidity seem to favor, tryptic digestion ; hence proteolysis occurs more rapidly in the presence of small quantities of acetic or lactic acid than in a neutral medium (Lindenberger). Albumin becomes dissolved without swelling ; all the proteids—with the exception of serum-albumin (Neumeister) —are first converted into a globulin which is insoluble in water, and are later converted into peptones soluble in water. According to Kühne, tryptic digestion occurs as follows : The proteid molecule, after passing through numerous intermediate stages, is converted into two different peptones—called hemipeptone and antipeptone. Anti-peptone resists any further catabolic change during digestion, while hemipeptone is converted into leucin, tyrosin, and asparaginic acid. This scheme of tryptic digestion is not universally accepted ; we do know, however, that large quantities of tyrosin and leucin are always formed (Kühne). To take an example, 382 grams of desiccated fibrin were digested artificially with pancreatic juice, and after six hours 343.7 grams were found to be dissolved ; the products formed consisted of 212.2 grams of peptone, 13.3 grams of tyrosin, and 31.6 grams of leucin. Schmidt-Mühlheim and Hofmeister found only small quan-tities of leucin and traces of tyrosin in the intestinal tract of dogs and pigs. Sheridan Lea found larger quantities in dogs ; thus the intestinal contents of a dog six hours after a meal of 500 grams of meat contained 1 gram of leucin and 0.3 gram of tyrosin.

Indol is not a product of pancreatic digestion, as was formerly be-lieved, but owes its origin to the action of micro-organisms on proteids. In addition to small quantities of ammonia (Hirschler, Stadelmann) another unknown substance is found in the intestine, when putre-factive processes are inhibited. It gives a violet color when treated with chlorin or bromin water, and may be considered as a chromogen. This substance was also described by Tiedemann and Gmelin, and was later designated as proteinochrome or tryptophane. Since it is con-stantly formed during advanced proteolysis,—that is, when the proteid molecule undergoes very thorough decomposition,—this chromogen is particularly interesting.

Of the organic bases (hexabases), there are formed lysin (Drechsel and Hedin), some histidin, and a large amount of arginin (Kutscher). In tryptic digestion of globin leucinimid has been demonstrated (Sal-askin), and cystin in the tryptic digestion of blood-albumin (Mörner).

The action of trypsin on other bodies is as follows : The proteids undergo cleavage, and the albuminoids thus split off undergo further digestion. Casein is converted into proto- and deuterocaseoses and casein-peptone (Chittenden). Gelatin is first converted into proto- and deuterogelatose, then into a peptone, and finally into amido-acids. The

gelatinous substances of connective tissue are never digested until they have been subjected to the action of dilute acids, which causes swelling, or to the action of hot water, which shrivels them up. Elastin is dissolved and forms elastoses that are not convertible into peptones.

The quantity of ferments in the pancreatic juice presents considerable variations, which chiefly depend on the rate of secretion and on the character of the diet. If more pancreatic juice is secreted in a given time unit, the percentage of ferments is usually correspondingly reduced, and vice versâ. The effect of the diet is the following: If the diet contains much proteid material, large quantities of tryptic ferments are excreted; when consisting chiefly of milk and bread, correspondingly large quantities of the amylolytic ferments are poured into the intestine (W. Wassilieff).

M. Abelmann has reported a number of interesting experiments on the assimilation and the metabolism of different articles of food after extirpation of the pancreas in dogs. From 7 to 24 grams of nitrogen (45 to 155 grams of albumin) in the form of meat, bread, and milk were given daily, and it was found that 44 per cent. was absorbed; 151 to 176 grams of starchy food were given daily, and 57 to 71 per cent. was assimilated. Fat, when given in the form of butter, lipanin, or as olive oil, in quantities of from 36 to 78 grams daily, was not absorbed, but was split up and passed in the feces as free fatty acids or as soaps. From 28 to 53 per cent. of the fat of milk, however, was absorbed. [Halliburton and Brodie find that the pancreatic juice obtained from temporary pancreatic fistula in dogs precipitates casein from milk in a form they provisionally term pancreatic casein. This action differs from that of gastric rennet, but pancreatic casein is converted into true casein by rennet. As regards its solubility, pancreatic casein is intermediate between casein and caseinogen. Kühne, Sir W. Roberts, and Edkins previously observed the coagulation of milk by extracts of the pancreas and by Benger's "Liquor Pancreaticus." Vernon has shown that there is a pancreatic rennin, and that, like trypsin, it exists in the gland as a zymogen and is not a single chemic substance, but a series of rennins of varying degrees of stability. Sahli has introduced as a means of diagnosis glutoid capsules containing iodoform. If the capsule comes in contact with pancreatic juice, the iodoform is liberated and can be detected, but when there is disease of the pancreas interfering with its secretion, or when, from any cause, the capsule is retained in the stomach, iodoform is not liberated. In utilizing this test as a means of estimating the existence of pancreatic secretion it is essential that the motor power of the stomach be normal.—Ed.]

The Bile.—The bile, secreted by the liver, is also poured into the duodenum and must be considered. While it is now well known that the bile exercises no chemic effect on the food, it nevertheless plays an important part in the process of digestion: in the first place, it tends to alter the reaction of the intestinal contents; in the second place, it exercises a direct effect upon intestinal function in different ways. The most important function of the bile of course is

excretory, for through this channel many of the useless products of metabolism are eliminated. Here, however, it is only necessary to give a brief description of the secretion of bile and its chemic composition.

Bile obtained from biliary fistulas can be analyzed, and results are more easily and satisfactorily obtained than in the case of the pancreatic juice. The contents of the gall-bladder are unsuitable for analysis, inasmuch as the bile in the gall-bladder is much more concentrated than the bile that flows through the ducts into the intestine. The bile is a clear, tenacious, mucoid fluid with an alkaline reaction, due to the presence of alkaline carbonates and phosphates. The color varies from a golden yellow to an olive brown, and is never green or greenish (Hammarsten). The ingestion of food increases the secretion of bile, water apparently exercising the greatest influence in this respect (C. Voit).

According to Bidder and Schmidt, Wolf, and von Voit, the curve of biliary secretion varies according to the character of the food ingested. The highest point is always reached during the first hour after a meal (Arnold, Voit). Proteids increase the amount of bile excreted; fats reduce it; while carbohydrates are without any appreciable influence (Voit). During fasting only one-half to one-third as much bile is excreted as on a normal diet. [Bruno and Kladnizki, pupils of Pawlow, by means of an ingenious fistula which allowed the bile to be collected from the biliary papillæ, showed that during fasting no bile enters the duodenum. Investigation of the effect of food showed that the administration of water, acids, raw egg-albumen, and boiled starch was not followed by the passage of bile into the bowel, but that fats, extracts of meat, and the products of the digestion of egg-albumen set up a free flow of bile into the duodenum.—Ed.]

The total quantity of bile excreted in twenty-four hours, according to Ranke, Wittich, and others, amounts to about from 500 to 600 c.c. Hammarsten has indorsed these figures as a result of observations on the bile in 7 cases of biliary fistula in human subjects. In his cases the percentage of solids in the bile was found to be as high as 2 or even 3 per cent., and was consequently greater than is usually assumed. The most important and most characteristic constituents of the bile are the bile acid salts (glycocholic and taurocholic acid salts) and the pigments (bilirubin and biliverdin). In addition there is a peculiar, mucin-like substance which has been carefully analyzed by Paijkull, who, working with ox bile, came to the conclusion that it was nucleo-albumin. In Hammarsten's cases a considerable quantity of mucus containing mucin was constantly found; nucleo-albumin alone was found in only 1 case. It is probable that this mucoid substance is an addition to the bile and is poured out by the glands in the walls of bile-ducts and the gall-bladder. In addition, small quantities of cholesterin, lecithin, soaps, fats, and salts are found.

The amount of iron found in fresh bile was 0.003 to 0.0044 per cent. A diastatic ferment has also occasionally been found in the bile. According to Neumeister, this is not a specific constituent of the bile, and has no more significance than the diastatic ferment of the urine,

which is identical with the ptyalin-zymogen of the pancreas. [Rachford found that bile itself had some slight diastatic action, but that its chief effect on the digestion of carbohydrates was in helping the pancreatic juice—viz., in neutralizing free acid and in diminishing the retarding influence which bicarbonate of soda has upon the diastatic power of the pancreas.—ED.]

Hammarsten's quantitative analysis of the bile yielded the following figures:

Solids	1.620– 3.520
Water	96.470–98.370
Mucin and pigments	0.270– 0.910
Alkaline salts of the bile acids	0.260– 1.820
Taurocholates	0.053– 0.303
Glycocholates	0.204– 1.610
Fatty acids (from soaps)	0.024– 0.136
Cholesterin	0.048– 0.160
Lecithin	0.048– 0.065
Fat	0.061– 0.095
Soluble salts	0.676– 0.887
Insoluble salts	0.020– 0.049

As has been pointed out, experiments *in vitro* show that bile exercises no chemic effect on proteids and fats. It also appears that the effect of the bile on carbohydrates is infinitesimal as compared with the effect of an equal quantity of pancreatic juice. Another method of studying the influence of the bile on digestion is by chemic examination of the intestinal contents in the subjects of biliary fistula, in whom no bile passes into the bowel. The problem has been worked out by a number of investigators, with the following results: Röhmann, von Voit, and J. Munk found that albumin, gelatin, and carbohydrates are absorbed from the intestine in a perfectly normal manner in the absence of the bile. The normal absorption of fat, however, is interfered with. A healthy animal was found to absorb nearly 99 per cent. of 150 to 200 grams of fat given by the mouth, and to pass only 1 per cent. of the fat in the feces. With a biliary fistula, more than 60 per cent. of the fat was passed in the stools (Voit). More than 150 grams were not tolerated at all. Munk's experiments showed a rather higher absorption of fat. He administered 247.6 grams of fat (lard) and 56.5 grams of nitrogen (albuminous diet), and found that 66.9 per cent. of the fat and 90 per cent. of the nitrogen were absorbed. The composition of the fat was the following: Neutral fat, 7.85 per cent.; free fatty acids, 61.84 per cent.; fatty acids,—as soaps,—10.93 per cent.; and cholesterin, 1.43 per cent. This shows that only one-tenth of the fats passed in the stools was neutral fat, and that a large proportion of free fatty acids was excreted (Röhmann, Hédon, and Ville arrived at similar results). Voit, on the other hand, found that the fat in the feces was mainly unconverted. In animals with a biliary fistula symptoms of inanition usually appear, and must be considered as the result of deficient absorption of fat. Inanition can be prevented by a carefully arranged course of food.

To sum up our knowledge at the present time with regard to the action of the bile:

The fats are emulsified by the action of the bile and of the pancreatic secretion so as to form a very minute and stable emulsion. The bile, according to Nencki, supplements the lipolytic action of pancreatic juice. Thus in the absence of bile only 61 per cent. of tribenzoïcin was split up into fatty acids and glycerin by pancreatic juice; while on the addition of bile, the whole amount was acted upon. At the same time the alkalis that are poured into the intestine with the bile render the formation of soaps possible. Bile can dissolve fats, although only to a slight degree, and dissolves the insoluble earthy soaps present in the chyme and renders their absorption possible. Von Wistinghausen has shown that an animal membrane impregnated with bile is more permeable to fat emulsions than one that is moistened with water. Heidenhain considered that the bile favors the entrance of fat into the epithelial cells of the intestine, and that it acts directly on the surface of these cells, causing their membrane to become permeable to fat. [In an experimental research on the absorption of fat B. Moore and Rockwood emphatically oppose the view that fat in an emulsified form enters the epithelial cells of the intestinal mucosa. This is supported by the fact that fat-globules are never seen in the free and striated border of the cells, by the gradual growth of fat-globules in the cells observed by Krehl and Altmann, and by Cash and Ludwig's observation that active fat absorption, as shown by the lacteals, occurs from parts of the intestine containing no emulsified fat. Fat is absorbed by the epithelial cells in a soluble form either as free fatty acids or as soaps. In experiments with dog's pancreas and bile they found that minced pancreas and bile combined both decomposed and dissolved fat, that pancreas alone decomposed but did not dissolve fat, and that bile alone did neither. Pawlow, from a systematic series of experiments, concludes that the chief use of bile is to augment the activity of the pancreatic ferments, more especially of the fat-splitting ferment.—Ed.]

Bile also constitutes an irritant to epithelial cells, stimulates their function, and maintains their functional powers (Röhmann).

It is also thought that the bile causes the precipitation of pepsin and of the dissolved albuminoids entering the intestine from the stomach; that in this way a resinous precipitate forms and adheres to the intestinal wall and is less readily propelled onward when bile is present than when it is not, and that in this way the assimilation of proteids is facilitated. [From experiments with pills formed of bile E. P. Joslin found that the digestion of proteids is improved by the use of bile when the stools contain a large quantity of fat. This is probably brought about by the bile clearing off the excess of fat, and so enabling the proteid elements of the food to come into closer contact with the digestive juices.—Ed.]

Some authors consider that the bile has antifermentative and antiputrefactive powers (Maly and Emmich). Lindenberger has shown that

0.05 per cent. of lactic acid alone is without influence on the putrefaction of an infusion of pancreas, whereas the presence of a small amount of bile, together with 0.05 per cent. of lactic acid, effectually prevents putrefaction, even after two weeks' digestion. The contents of the duodenum are always more or less acid, so that it must be conceded that possibly under these conditions the bile may exert an inhibitory influence on fermentation. Lastly, the bile seems to exercise a distinct effect on the peristalsis of the intestine. As the result of his researches, Röhmann believes that the bile increases peristalsis.

The Intestinal Juice.—The third of the digestive fluids that are present in the intestine is the intestinal juice (*succus entericus*). A great number of more or less contradictory statements, as regards the origin and the digestive action of this secretion, have been made. Hoppe-Seyler does not even believe that it has ever been positively shown that the glands of Lieberkühn secrete a fluid that might be called intestinal juice. Many authors, on the other hand, positively regard the succus entericus as the secretion of the glands of Lieberkühn, and have succeeded in obtaining it in an uncontaminated state by means of a Thiry-Vella fistula. Démant examined the succus entericus from a human subject with an intestinal fistula, following herniotomy, and found the fluid to be light wine-yellow in color, distinctly alkaline in reaction, owing chiefly to the presence of nearly 0.5 per cent. of sodium carbonate. Of the 3.5 to 4.5 per cent. of solid residue, 1.5 was organic. The chief constituents of the intestinal juice are albumin and mucin. The quantity of these bodies present seems to fluctuate within wide limits. Ptyalin and an inverting enzyme are the only ferments that have been discovered. Intestinal juice does not seem to act on albumin and fat. It appears, therefore, that the chief rôle of the intestinal juice in digestion is to neutralize the acids formed by the fermentation of the carbohydrates. The presence of mucin seems to show that the succus entericus plays an important part in the onward movement of the intestinal contents. [Pawlow's experiments have shown that the succus entericus augments the activity of the pancreatic ferments, and more especially of trypsin. The activity of the fat-splitting and amylolytic pancreatic ferments is augmented equally by the succus entericus derived from the duodenum and from other parts of the small intestine, but the succus entericus of the duodenum augments the activity of the proteolytic pancreatic ferments in a more marked degree than does the succus entericus from the rest of the small intestine. Pawlow considered that the succus entericus contained a ferment of other ferments, which he called "enterokinase." The succus entericus secreted in response to mechanical irritation, such as that of a tube when inserted into a fistula, is chiefly water and contains little of the ferment. The specific stimulus which calls forth a secretion of the ferment is the presence of pancreatic juice in the intestine. Hamburger and Hekma have found, from experiments in a case of intestinal fistula, that enterokinase exists in the succus entericus of man. Pregl has estimated that the total amount of succus entericus secreted during the twenty-four hours is 3 liters. Wein-

land finds that in the new-born child and in all mammals during the suckling stage the small intestine contains a ferment, lactase, which has the power of splitting up lactose. Hamburger and Hekma have shown the existence, in the succus entericus of man, of a ferment, "erepsin," described by Cohnheim in the cat and dog, which transforms hemi-albumose into other bodies.—ED.]

In the preceding paragraphs the action on food of the different intestinal secretions and of their unorganized ferments or enzymes has been discussed. The effect of the organized ferments—that is, of the *ferment organisms*—on the food must now be considered—in other words, the bacterial fermentation processes that occur in the intestine. Proteids, carbohydrates, and fats are all decomposed by micro-organisms in the intestine. A small proportion of the fat (Nencki, Blank) is decomposed into glycerin and fatty acids, and subsequently into other acids of a smaller molecule of the fatty acid series. This process occurs in the lower portions of the small intestine. The interesting process of carbohydrate fermentation which goes on chiefly in the small intestine (for a detailed description of the organisms concerned see p. 48) leads to the formation of acetic, lactic, and butyric acids, alcohol, etc. (see Nencki below), carbon dioxid, and hydrogen. What proportion of the ingested carbohydrates is decomposed by these fermentative processes is as yet undetermined.

The decomposition of proteids by intestinal bacteria (p. 48), usually spoken of as putrefaction, occurs only when the reaction of the intestinal contents is alkaline. In the early stages the bodies formed are the same as in pancreatic digestion,—namely, albumoses, peptones, amido-acids, and ammonia,—but the process is much slower than in tryptic digestion. The process of disassimilation is carried on further and leads to the formation of aromatic oxy- and hydroparacumaric acids, which are derived from tyrosin. From these, later, paraoxyphenylacetic acid, paracresol, and finally phenol are formed by oxidation. In addition a certain quantity of phenylpropionic and of phenylacetic acids are formed. These two may also be formed directly from albumin. A second series of aromatic substances, which are not related to tyrosin, is represented by indol, skatol, and skatol-carbonic acid. [The production of indol depends largely on the activity of the colon bacillus. When carbohydrates are present as well as proteids, the colon bacilli ferment the carbohydrates first and no indol is manufactured until this is nearly completed. Indol is moderately toxic, and may give rise to symptoms of neurasthenia (Herter). When absorbed from the intestine, it is oxidized to indoxyl and further combines, probably in the liver, to form indoxyl sulphate of potassium, which is less toxic than indol, is readily excreted in the urine, and is commonly called indican (Herter).—ED.]

Other products of the putrefaction of albumin, in addition to leucin, are the ammonia salts of capronic, valerianic, and butyric acid. These are derived from the fat nucleus of the proteid molecule. A number of gases are also formed during this process of putrefactive fer-

mentation—namely, carbon dioxid, hydrogen, sulphureted hydrogen, hydrocarbons, and methylmercaptan (Nencki).

Laufer has studied the influence of intestinal bacteria on the digestion of nitrogenous food under both normal and pathologic conditions by making quantitative estimations of the nitrogen value of the excreta. The results do not yet justify a positive conclusion as to the physiologic and pathologic limits of bacterial activity in the intestines.

While we are unable to estimate the amount of carbohydrate fermentation, we can determine with a fair degree of accuracy the intensity of proteid putrefaction in the intestine. This depends on the fact that the aromatic disassimilation products of albumin described above are excreted in the urine either alone, as ethereal sulphates, or in combination with glycuronic acid (for the details, see below).

We have so far discussed the action of the organized ferments of the intestine on the different groups of food material as they occur *in vitro*. In the living organism the conditions are much more complicated than in the test-tube, for the different digestive fluids exert their effects simultaneously, so that all the processes that we have described occur together, and thus, no doubt, exert a certain amount of interaction on each other. In addition, we must always remember that the intestinal juices act chiefly upon food material that is already partially disassimilated or digested by the saliva and the gastric juice, and acts only in small part on food material in its original form.

We shall now attempt to study the changes that the chyme, after leaving the stomach, undergoes in the intestine, and shall endeavor to imitate the natural conditions as nearly as possible. For this purpose we shall study the effect of several secretions at once when mixed with food material in a beaker. In addition, we shall analyze these processes in animals or human subjects with intestinal fistulas, and finally discuss the analysis of the contents of different portions of the intestine in animals just killed.

The composition of the chyme may vary greatly at the time when it passes from the stomach into the intestine. Very much will depend on the character and the quantity of the food. The proteids are either unchanged or appear as syntonin, albumose, or peptone. The carbohydrates may also be unchanged or appear as dextrin and maltose. The fats remain unchanged. Occasionally a minute portion of the fat has undergone cleavage. The reaction of the chyme at this period is usually more or less acid, owing to the presence of free hydrochloric acid. Much will depend upon the duration of digestion. As soon as bile acts on this acid chyme a resinous, flocculent precipitate and a finely granular clouding appear. The former, according to Hammarsten, consists of syntonin and some bile acids; the latter, of bile acids and small quantities of peptone. If more bile is allowed to flow into the mixture, the precipitate may become redissolved, even though the reaction is not rendered alkaline. This is especially the case when syntonin is not present in large quantities. The majority of investigators

(Ewald, Schmidt-Mühlheim) have never found a precipitate of this character in the small intestine of an animal killed during digestion. The hydrochloric acid is partially or completely neutralized as soon as the chyme enters the intestine. In this way all peptic digestion is inhibited. As a matter of fact, if we are justified in concluding that the precipitate is formed, pepsin will be precipitated with the sediment, for finely divided sediments seem to have a tendency to precipitate the ferments. Boas denies that pepsin is precipitated in this way. The reaction of the intestinal contents furnishes a sure guide as to the mode of action of the pancreatic and intestinal juices. Simon and Zerner found that of the contents of the small intestine from recently dead corpses that from the duodenum and upper jejunum always showed an alkaline reaction to litmus, and that from the lower jejunum and ileum an acid reaction. The acid intestinal juice exerted a digestive action on fibrin only after it had been rendered alkaline by sodium carbonate. On the other hand, the intestinal contents which had been thus alkalinized lost their previously strong diastatic property. In the normal contents of the upper portion of the intestine, which are usually alkaline in reaction, the action was reversed. Fibrin was digested in a few hours, but starch was saccharified only after complete neutralization of the intestinal contents. The same condition of action was observed in the intestinal fluid obtained by a jejunal fistula. These observations as to the action of the pancreatic secretion of animals apply to man as well. Bile does not interfere with the proteolytic power of pancreatic juice, as was shown many years ago by Claude Bernard. As a matter of fact, albumin is rapidly digested by the intestinal contents of an animal killed during digestion, even though a considerable quantity of bile is present. Boas found that the same applies to the secretion of the small intestine in man. The fluid he obtained was a mixture of bile, pancreatic juice, and succus entericus, and had an alkalinity corresponding to 0.8 pro mille of sodium bicarbonate. Digestion experiments showed that at a temperature of 40° C. it dissolved 81 per cent. of the serum-albumin mixed with it within three hours. In another experiment in which he determined the amylolytic powers of this fluid the alkalinity was equal to 0.2 pro mille of sodium bicarbonate, and 25 per cent. of the starch was converted into maltose within three hours. Lastly, 12.1 per cent. of fatty acids was formed from a given quantity of neutral olive oil in three hours.

The lipolytic power of the pancreatic juice, as we have already seen, is augmented by the bile; this, as Martin and Williams have shown, also applies to its amylolytic power. The effect of trypsin on rennet has also been studied, and it has been shown that a neutral solution of the former destroys rennet within a short time, even at room-temperature (A. Baginsky). Trypsin has no effect on pepsin; on the other hand, trypsin is destroyed by pepsin in acid solution (Kühne, Ewald, Mays, Langlay, Baginsky).

The chyme, in addition to being changed by the digestive action of the pancreatic juice, supplemented and augmented, as we have seen, by

the action of the bile, undergoes fermentation changes induced by the action of micro-organisms. Under normal conditions it appears that only the carbohydrates are affected in this way in the small intestine. Ewald and, of late years, Nencki have shown that no putrefactive decomposition of proteid occurs in the small intestine, but that certain products of carbohydrate fermentation—viz., considerable quantities of lactic acid, alcohols, particularly ethyl alcohol, and carbonic acid—are always present, so that carbohydrate fermentation certainly occurs there. They performed their experiments with the contents of the small intestine obtained from a fistula opening into the lowest portion of the ileum. They believe that the reason why putrefactive decomposition of albumin does not occur in this portion of the intestine is that the acid reaction prevents this putrefaction, and that, moreover, the duodenal contents are so rapidly propelled into the large intestine as to render putrefaction impossible.

The investigations of Macfadyen and Nencki and Sieber just mentioned contain so many important details on the chemic processes going on in the human intestine that it may be worth while to refer to the chief results of their work. The quantity of chyme that passed from the ileum into the cecum was found to depend on its consistence. If it was thin and pultaceous, 550 grams, with 4.9 per cent. of solid residue, could pass; when the evacuations were thick, only 282 grams, with 11.23 per cent. of solid residue, passed in the twenty-four hours. They found that the food never reached the ileocecal valve sooner than two hours, and never later than five hours and a quarter, after ingestion. When the diet consisted chiefly of proteids, the intestinal contents flowing from a fistula were usually yellowish or yellowish-brown in color, owing to the presence of bilirubin. As a rule, it was almost odorless, and only occasionally had a slightly burnt odor or an odor of volatile fatty acids. Occasionally the odor was slightly fecal and similar to indol. In consistence the intestinal contents were usually thin and liquid, occasionally somewhat thickened, or even as thick as cold cream or vaselin. The reaction of the chyme under normal conditions was acid, and the average acidity, calculated for acetic acid, was 1 pro mille. This acidity is undoubtedly due to the presence of organic acids, particularly acetic acid, since the hydrochloric acid of the stomach and the lactic acid formed by the fermentation of sugar are neutralized by the succus entericus. After removal of the insoluble portions of the chyme the following bodies could be determined in the liquid: As much as 1 per cent. of albumin coagulable by heat; in addition, peptone, mucin, and disassimilation-products of starch, such as dextrin and sugar. The quantity of sugar present varied from 0.3 to 4.75 per cent. In addition to inactive lactic acid, sarcolactic acid, small quantities of volatile acids of the fatty acid series, especially acetic acid, succinic acid, bile acids, and bilirubin were found. Leucin, tyrosin, urobilin, and ammonia were shown to be absent. No odor or chemic evidence of the presence of the characteristic putrefaction-products of albumin, as indol, skatol, phenol, methyl-

mercaptan, could be detected even when a kilogram of the chyme was analyzed. Phenylpropionic acid, paraoxyphenylpropionic acid, and skatolacetic acid were also absent. An analysis of the ash of the contents of the small intestine showed that from 20 to 40 per cent. of the bases were combined with mineral acids; 60 to 80 per cent., with organic acids.

[In a series of experiments on herbivora and carnivora Moore and Rockwood found that in herbivora the contents of the small intestine are more alkaline than in carnivora. Carbohydrate food increases the alkalinity of the small intestine, which it certainly would not do if attacked by bacteria and decomposed into organic acids. When the chyme reaches the cecum, it becomes acid from bacterial fermentation. From analogy the authors conclude that the human small intestine, which in reaction probably is intermediate between that of the herbivora and the carnivora, cannot, under normal conditions, have an acid reaction in any considerable part of its extent. They consider the three observations on human fistulas (Ewald's, Macfadyen, Nencki and Sieber's, Jakowski's) too small to form a decision on, and suggest that there may have been excessive bacterial activity in the small intestine.—ED.]

As soon as the intestinal contents passed from the small intestine into the colon a completely different chemic picture was presented : the reaction became alkaline, the action of the enzymes stopped, and the ordinary fecal odor appeared, showing that putrid fermentation of proteid material had begun. The exact composition of the contents of the colon was determined by M. Jakowski, who studied the intestinal contents obtained from a fistula situated in the ascending colon. From 150 to 200 grams of fecal intestinal contents of a doughy consistence and of a neutral or alkaline reaction were passed daily. There was 6.3 per cent. of dry residue, one-seventh of which consisted of mineral material. Neither unconverted bile-pigment nor bile acids could be discovered. Urobilin, skatol, phenol, traces of oxy-acids, ammonia, leucin, cadaverin, capronic acid, valerianic acid, succinic acid, lactic acid, ethyl alcohol, butyl alcohol, sulphureted hydrogen, and methylmercaptan were all present.

Nencki, by his investigations, quoted above, was the first to estimate the amount of albumin which normally enters the large intestine. He calculated the nitrogen of the food, on the one hand, and of the chyme obtained from the fistula, on the other. The food contained 70.74 grams of proteid and 10.602 grams of nitrogen ; while the fluid passed in twenty-four hours from the fistula contained 26.5 grams of solid material, containing 1.61 grams of nitrogen in 10.06 grams of proteid. These figures show that only one-seventh of the food proteid, or, to be more accurate, 14.25 per cent., remained to be digested and assimilated in the large intestine, since 85.75 per cent. had been absorbed in the stomach and small intestine.

The activity of the putrefactive processes depends on a variety of factors. In the first place, the amount of material available for putrefaction, represented chiefly by the proteids, exercises an influence on the

intensity of the process. The quantity of proteid material present is, of course, primarily dependent on the amount ingested, and secondarily on the amount that is assimilated. Other factors that influence putrefaction are the length of time the material remains in the intestine, which again is dependent on the activity of the peristaltic movements of the intestine. Lastly, the reaction of the intestinal contents is of some importance. If markedly acid, especially if due to the presence of free acids, putrefaction is inhibited. Reference should be made to the remarks on the antiseptic powers of the bile (*vide* p. 24). Hirschler has recently shown that the carbohydrates of the food seem to inhibit putrefaction to a certain extent. Laas studied the same problem and made out that the fats do not exercise the same inhibitory influence on putrefaction as the carbohydrates. Nencki and Blank have shown that putrefactive organisms have very little power to decompose fat.

J. Wortmann's work on the carbohydrates shows that starch is acted upon by putrefying infusions of beans and potatoes in the same manner as by diastase. It appears, however, that this occurs only in the absence of other carbon compounds and in the presence of plenty of air. If putrefaction is allowed to continue for some time, the carbohydrates are completely converted into lactic, butyric, acetic, and carbonic acids and hydrogen.

[B. Moore and Rockwood's experiments, already referred to, indicated that very little bacterial fermentation of carbohydrates took place in the small intestine of animals.—ED.]

The Excreta.—The excreta leaving the rectum consist partly of undigested or indigestible constituents of the food, and partly of more or less changed digestive fluids and bacteria. According to Rieder and Rubner, a diet limited to eggs and meat gives rise to feces consisting of intestinal secretions, as is the case during fasting.

The consistence of the feces is determined by the proportion of the solid to the liquid constituents. The liquid portions are almost exclusively water. Under normal conditions, therefore, the consistence of the feces is doughy or pultaceous. The quantity of solid matter varies with the character of the diet (Pettenkofer and Voit). On a mixed diet, 120 to 150 grams, containing from 30 to 37 grams of solid residue, are excreted in twenty-four hours. Rubner found that 2438 grams of milk were represented by 96 grams of fresh feces, leaving a dry residue of 24.8 grams. With 1435 grams of meat, 64 grams of fresh feces, containing 17.2 grams of dry residue, were passed. If the diet consists largely of vegetable material, the quantity of feces increases considerably, as will be seen by the following table:

1360 grams of black bread	yield	815	grams of fresh feces,	115.8 grams of dried feces.
3078 "	potatoes	"	635	"	"	"	93.8	"	"	"
3831 "	cabbage	"	1670	"	"	"	73.8	"	"	"

With a mixed diet the color of the feces is a dark brown (urobilin); if much meat is eaten, it is brownish black (hematin, sulphid of iron);

on a milk diet it turns light yellow (owing to the large quantity of fat in the feces). The characteristic fecal odor is produced chiefly by skatol and volatile fatty acids. The reaction of the feces is usually slightly alkaline or neutral ; less frequently slightly acid (Nothnagel).

Qualitative analyses of the feces show the following constituents : Water, traces of albumin, mucin, various fats, occasionally cholalic acid, dyslysin, cholesterin, koprosterin, lecithin, skatol, indol, salts of the volatile fatty acids (especially butyric and acetic acids) and of lactic acid ; also calcium and magnesium soaps. Von Jaksch succeeded in demonstrating the presence of two ferments in human feces—one invertin, the other diastatic.

[Hemmeter, in the sterile extract of human feces, the action of bacteria being thus excluded, found a proteolytic ferment acting best in an alkaline medium.—ED.]

The following inorganic salts are found in the feces : chlorids and carbonates of alkaline bases, earthy phosphates, and small quantities of iron salts and silicates.

The quantitative composition of the feces depends largely on the diet and the digestive powers of the individual, and consequently varies greatly. The amount of nitrogen present in the stools of fasting persons is about 0.2 gram (this was found to be the case by Müller in the stools of Cetti). If the diet contains no nitrogen, the amount of nitrogen excreted in the feces is somewhat larger, owing to the fact that a more abundant secretion of digestive fluids occurs in a diet of this character (Rieder). The amount of nitrogen fluctuates between 0.5 and 0.8 gram, or 4.1 to 5.8 per cent., of the dried feces.

[Ury has investigated the point as to how much of the nitrogen normally found in the feces is derived from food and how much from intestinal secretions and excretions. The amount of nitrogen derived from the secretions and excretions of the intestines constitutes from 18 per cent. to 32 per cent. of the total nitrogen of the feces, which corresponds fairly well with Rieder's estimate of the amount of nitrogen found in the feces when food free of nitrogen is taken.—ED.]

When 884 gm. of meat per day were taken, 1.2 gm. N were excreted = 6.9 per cent. of the dried feces (Rubner). With a milk diet, 1.1 gm. N = 4 per cent. were excreted. A diet containing little feces-producing matter showed a daily excretion of 1.14 gm. N (Praussnitz). A vegetable diet (rich in feces-producing matter) raises the N-contents to $3\frac{1}{2}$–4 per cent. (Voit, Rumpf, and Schumm).

Under normal conditions the quantity of fat contained in the feces is independent of diet and excretions. The quantity of fat elaborated by the latter is determined by examination of the feces of fasting persons. These showed 1 gm. of fat a day. Both the quality and the quantity of the diet are to be considered. However, persons on the same diet have shown individual differences in fat-excretion.

Carbohydrates give rise to but little starch. Sugar cannot be demonstrated at all. Schmidt and Strassburger demonstrated its presence under normal conditions after a test-meal to the amount of 2.3 and 4.8

per cent. of the dried feces. The quantity and solubility of food are factors in the formation of starch.

Very little is known about the inorganic constituents of human feces. Under mixed diet the percentage of fecal ash varies between 11 and 15 per cent. (Ranke, Praussnitz, Grundzach). With milk diet this rises between 27 and 35 per cent. of the dried feces (Rubner, Fr. Müller), almost three times as high as under mixed diet. On a meat diet the feces contain from 13 to 16 per cent. of ash. There are on record two analyses of feces dating back to 1840–50 made by Fleitmann and by Porter; also one of more recent date by Grundzach. The results obtained were the following. One hundred parts of ash contained:

Constituents.	Fleitmann.	Porter.	Grundzach.
Sodium chlorid	0.58	4.33	0.344
Potassium chlorid	0.07	. . .	
Potassium oxid	18.49	6.10	12.000
Sodium oxid	0.75	5.07	3.821
Calcium oxid	21.36	26.46	29.250
Magnesium oxid	10.67	10.50	7.570
Ferric oxid	2.09	2.50	2.445
Phosphoric acid	30.98	36.03	13.760 [P_2O_5]
Sulphuric acid	1.13	3.13	0.653 [SO_3]
Silicic acid	1.44	. . .	0.052 [SiO]
Sand	7.39	30.00	4.460

It will be noticed that the quantities of phosphoric and sulphuric acids found by Fleitmann and Porter differ greatly from those found by Grundzach. This is due to the fact that their methods of analysis were different. The first-named investigators incinerated the undigested particles of food, like albumin, lecithin, nuclein, etc., and in this way obtained higher figures for phosphoric and sulphuric acid. Grundzach, on the other hand, determined the sulphuric acid, the phosphoric acid, and the chlorin in the powdered feces and not in the ash, and consequently obtained smaller values.

From these analyses it will be seen that 22.13 per cent. of all the alkalis contained in the feces are combined with mineral acids, and that 77.87 per cent. are combined with organic acids and with carbonic acid. This is due to the fact that the secretion of the large intestine neutralizes the acid contents of the small intestine (Nencki and Sieber).

According to the investigations of Brauneck, ammonia is always present in small quantities in the feces of healthy subjects. On a milk diet and on a mixed diet 0.0408 gram of ammonia is excreted daily, and dried feces contain 0.151 per cent. of NH_3.

[Ury has shown that the calcium in the feces is derived from the food and not from the secretions and excretions of the intestines. It therefore follows that calcium derived from metabolism of the fixed tissues leaves the body by the urine.—Ed.]

LITERATURE.

Abelmann, Diss., Dorpat, 1890.
Baginsky, A., Zeitschr. f. phys. Chem., 1882, vol. vii.
Balke, ibid., vol. xxii., p. 248.
Bernard, C., Leçons des physiolog. expérim., etc., Paris, 1856.

Bernard, C., and Berthelot, Leçons d. phys., 1855, p. 263.
Bernstein, Sitzungsber. d. Akad. d. Wiss., Leipsic, 1869, p. 96.
Bidder and Schmidt, Die Verdauungssäfte und der Stoffwechsel, Leipsic, 1852.
Blank, Arch. f. exp. Path., 1886, vol. xx.
Boas, Zeitschr. f. klin. Med., 1890, vol. xvii., p. 158.
Boas, Deutsch. med. Wochenschr., 1891, No. 28.
Brauneck, Mittheil. a. d. Med. Klinik z. Würzburg, vol. ii., p. 886.
Brücke, Wien. akad. Berichte, 1870, 61. Jahrg., p. 362.
Chittenden, ref. in Maly's Jahresber., 1890.
Cohnheim, O., Zeitschr. f. phys. Chem., vol. xxxiii., p. 451.
Corvisart, Sur une fonction peu connue du pancréas, Paris, 1857.
Demant, Virchow's Arch., 1879, vol. lxxv., p. 419.
Drechsel and Hedin, Du Bois' Arch., 1891, p. 273.
Ewald, Klinik der Verdauungskrankheiten, 1886, vol. i., p. 136.
Ewald, Virchow's Arch., vol. lxxv., p. 409.
Fleitmann, Poggendorf. Ann., 1849, vol. lxxxvi., p. 371.
Gad, Arch. f. Anat. u. Phys., 1878, p. 187.
Grundzach, Zeitschr. f. klin. Med., vol. xxiii., p. 70.
Hammarsten, ref. in Maly's Jahresber., 1893.
Heidenhain, Arch. f. d. ges. Physiol., vol. x., p. 557.
Heidenhain, Pflüger's Arch., 1888, vol. xliii., p. 91.
Hirschler, Zeitschr. f. phys. Chem., 1886, vol. x., p. 302.
Hirschler, ibid., vol. x., p. 306.
Hofmeister, Deutsch. Zeitschr. f. Thiermed., 1890, vol. xvi., p. 226.
Hofmeister, V., Berichte über die Veterinärwesen im Königreich Sachsen, 1889, p. 156.
Hoppe-Seyler, Handb. d. phys. Chemie, 1878, p. 275.
Hüfner, Jour. f. prak. Chem., N. F., vol. v., p. 372.
Jakowski, Arch. d. sci. biol., St. Petersburg, vol. i., p. 539 (ref. Centralbl. f. d. med. Wiss., 1893).
Jaksch, v., Zeitschr. f. phys. Chem., vol. xii., p. 116.
Jawadsky, ref. Centralbl. f. Phys., 1891, vol. v., p. 179.
Korowin, Centralbl. f. d. med. Wiss., 1873, No. 20, p. 46.
Kröger, De succo pancreatico, Diss., Dorpat, 1854.
Kühne, Arch. f. path. Anat., vol. xxxix., p. 130.
Kühne, Verhandlung. d. Heidelberger naturhist. med. Vereins, N. F., 1876, vol. i., pt. iii.
Kühne, Verhandlung. d. naturhist. Ver. in Heidelberg, N. F., vol. i., p. 196.
Kühne, W., Arch. f. path. Anat., vol. xxxix., p. 130.
Kutscher, F., " Die Endproducte der Trypsinverdauung," Habilitationsschrift, Marburg, 1899.
Kutscher, F., Zeitschr. f. phys. Chem., vol. xxv., p. 195, and vol. xxvi., p. 110.
Laas, ibid., 1894, vol. xx., p. 233.
Laufer, L., Zeitschr. f. diätet. u. physikal. Therapie, vol. v., part vi., 1901–1902.
Lea, Sheridan, Jour. Phys., 1890, vol. xi., p. 226.
Lindenberger, ref. in Maly's Jahresber. über d. Fortschr. d. Thierchem., 1884, vol. xiii., p. 280.
Lindenberger, ref. in Maly's Jahresber., 1884, vol. xiv., p. 334.
Macfadyen, Nencki and Sieber, Arch. f. exp. Path., 1891, vol. xxviii., p. 311.
Maly and Emich, Monatsh. f. Chem., vol. iv., p. 89.
Mörner, Zeitschr. f. phys. Chem., vol. xxxiv., p. 238.
Munk, J., Virchow's Arch., vol. cxxii., p. 302.
Nencki, Arch. f. exp. Path., 1886, vol. xx.
Nencki-Blank, ibid., vol. xx.
Neumeister, Zeitschr. f. Biol., N. F., 1887, vol. v., p. 399.
Neumeister, Physiolog. Chem., 1893, p. 158.
Paijkull, Zeitschr. f. phys. Chem., 1887, vol. xii., p. 196.
Porter, Ann. Chem. Pharm., vol. lxxi., p. 109.
Rieder, Zeitschr. f. Biol., vol. xx., p. 384.
Röhmann, Pflüger's Arch., vol. xxix., p. 509.
Rubner, Zeitschr. f. Biol., vol. xv., p. 115.
Salaskin, S., Zeitschr. f. phys. Chem., vol. xxxii., p. 592.
Salaskin, S., ibid., vol. xxxv., p. 419.
Schmidt-Mühlheim, Du Bois' Arch., 1879, p. 39.
Siegfried, Zeitschr. f. phys. Chem., vol. xxi., p. 360.
Simon, O., and Zerner, Th., " Untersuchungen über die digestiven Fähigkeiten des Dünn-darmsaftes," Arch. f. Verdauungskrankheiten, 1901.
Stadelmann, Zeitschr. f. Biol., N. F., 1880, vol. vi., p. 261.

Tiedemann and Gmelin, Die Verdauung nach Versuchen, 1831.
Voit, C., "Beiträge z. Biol.," Jubiläumsschrift f. Geheimr. v. Bischoff, 1882.
Wassilieff, W., Arch. d. Sci. Biol., vol. ii., p. 219.
Wistinghausen, v., Diss., Dorpat, 1851.
Wortmann, Zeitschr. f. phys. Chem., 1882, vol. vi., p. 318.
Zawadsky, ref. Centralbl. f. Phys., 1891, vol. v., p. 179.
Zweifel, Untersuchungen über d. Verdauungsapparat d. Neugeborenen, 1874.

An exhaustive dissertation on the chemic processes in the intestine will be found in Hofmeister's *Text-Book of Physiologic Chemistry* (1893), which I can strongly recommend.

The *Chemical Composition of Feces under Normal and Pathologic Conditions* is the subject of a monograph by Schmidt and Strassburger (Berlin, 1902).

SUPPLEMENTARY LITERATURE.

Edkins, Jour. Physiol., Cambridge and London, 1891, vol. xii., p. 217.
Hamburger and Hekma, Jour. de Physiol. et de Patholog. General, September, 1902.
Halliburton and Brodie, Jour. Physiol., Cambridge and London, 1896, vol. xx., p. 97.
Hemmeter, Pflüger's Arch. f. d. ges. Physiol., vol. lxxxi., p. 151; Diseases of Intestines, 1901, vol. i.
Herter, Lectures on Chemical Pathology, 1902.
Joslin, E. P., Jour. Exper. Med., vol. v., p. 513.
Moore, B., and Rockwood, Jour. Physiol., Cambridge and London, vol. xxi., p. 58 ("Absorption of Fat"); ibid., p. 373 ("Intestinal Reaction").
Murray, F. W., and Gies, Amer. Med., July 26, 1902, p. 133.
Pawlow, The Work of the Digestive Glands, translated by W. H. Thompson, 1902.
Pregl, Arch. f. d. ges. Physiol., Bonn, 1896, vol. lxi.
Rachford, Amer. Jour. Physiol., vol. ii., p. 483.
Roberts, W., Proc. Royal Soc., 1879, 1881.
Sahli, Deutsch. Arch. f. klin. Med., vol. lxi.
Starling and Baylis, Proc. Royal Soc., 1902, vol. lxix., p. 352.
Ury, Deutsch. med. Wochenschr., October 10, 1901.
Vernon, H. M., Jour. Physiol., Cambridge and London, 1901, 1902, vols. xxvii., xxviii.
Weinland, Zeitschr. f. Biol., vol. xxxviii., p. 16.

THE BACTERIA OF THE INTESTINE.

By Prof. Dr. Julius Mannaberg.

A GREAT variety of micro-organisms are found in the intestinal tract both in health and in disease. There are more bacteria in the intestine of man than in any of the other organs which normally contain bacteria, such as the respiratory tract, the genital tract, the conjunctival sacs, and the cutaneous surfaces. Not only are bacteria present in greater numbers in the gastro-intestinal canal than in any of the other parts of the body, but the physiologic and pathogenetic functions of the bacterial flora of the intestine are much more important than those of the bacteria in other organs. The bacteria of the air-passages, for example, never, so far as is known, play any physiologic rôle, and it is only occasionally that they develop pathogenetic powers. The bacteria of the gastro-intestinal tract, on the other hand, are constantly engaged in the fermentative and putrefactive processes in the intestine, and therefore exercise a very important influence on the organism as a whole. At present their pathogenicity is not completely understood,

research having only recently been directed to this subject. Our knowl-edge, however, of the bacteriology of the alimentary canal and its bear-ing on the body as a whole is continually advancing. In this place we shall confine our remarks to the bacteria of the intestine and shall enter into a description of the bacteria present in other portions of the digestive tract, as the buccal cavity and the stomach, only in so far as this is necessary in order to understand cause and effect. Our descrip-tion will be further limited by the omission of any detailed account of the specific pathogenic micro-organisms, such as those of tubercle, anthrax, leprosy, glanders, cholera, etc., that are occasionally present in the intestine and may exert their deleterious influence there. These micro-organisms will be dealt with subsequently. In this work we shall, therefore, deal only with the obligate intestinal bacteria, or those usually present in the intestine or in the feces. The bacterial flora of the in-testine, as a rule, presents a uniform typical picture, but occasionally— and special stress must be laid on this point—certain bacteria which are not among those normally found in the bowel make their appearance as the result of some accidental occurrence. The number of germs present may also vary greatly. Hence it may be definitely stated that the bac-teria normally occurring in the feces fluctuate within wide limits, both quantitatively and qualitatively.

Leeuwenhoek was the first to discover the presence of micro-organ-isms in the buccal cavity and in the stools. He described the peculiar rounded and elongated structures he observed as "animalculæ," because they possessed such great motility. Leeuwenhoek's description was apparently forgotten, for no further mention of these microbes was made for one hundred and thirty years. At the end of this time Frerichs described the same organisms and distinguished yeast-cells, hyphomy-cetes, frustulariæ, and sarcinæ ; he also mentioned that these organisms are frequently found in the intestinal canal. At the same time Frerichs did not attach any particular importance to these bodies. Billroth, while investigating the coccobacteria, also examined the contents of the intestinal canal. As the result of microscopic examination, he found that meconium is usually sterile, but that the first yellowish stools passed by the new-born infant usually contain an abundant "coccobacterial growth." Billroth also made some observations on the distribution of bacteria in the different parts of the alimentary canal. He stated that the number of micro-organisms always increases from the stomach downward, and that in the colon there are large numbers of medium-sized forms, some motile, some non-motile. Woodward also attached some importance to the large number of bacteria normally found in the stools.

None of these authors attached any physiologic or pathologic signi-ficance to the micro-organisms of the intestinal tract. Nothnagel, in his dissertation on intestinal bacteria, was the first to suggest that the different species of intestinal microbes might be concerned in producing fermentation and putrefaction of the carbohydrates and the proteids in the intestine. He also gave a careful morphologic description of numer-ous intestinal bacteria ; and must, therefore, be credited with having

formulated the theory of the physiologic significance of intestinal bac-
teria and with having identified numerous intestinal microbes, such as
Bacillus subtilis, Clostridium butyricum, saccharomyces, streptococci,
etc.

Some time after this, Koch's discovery of the vibrio of cholera gave
a stimulus to the study of the bacteriology of the stools. It became
necessary to gain a clear understanding and a thorough knowledge of
the normal bacterial flora of the intestine, for without a knowledge of
the species normally present it was impossible to appreciate the signifi-
cance of abnormal bacteria. At the same time the question had to be
decided whether or not the normal bacteria of the intestine possessed
any physiologic or pathologic significance. These problems were under-
taken by a number of authors and thoroughly elaborated. Escherich was
first to utilize in this research the whole of the bacteriologic methods de-
vised by Koch. He carefully examined the feces bacteriologically, and
at the same time carried out a large number of investigations into the life-
history of the various species of bacteria by means of cultures. In this
way he elucidated the physiologic significance of many of the bacteria
concerned in digestion. As his observations will frequently be referred
to in the following paragraphs, it is unnecessary to go into further detail
at present.

As already pointed out, Billroth's microscopic examinations tended
to show that meconium is sterile. This was duly confirmed by Esche-
rich's cultivation experiments. The intestine of a new-born infant be-
comes infected by the swallowing of air, even before any milk is taken.
Breslau, who carefully investigated this process of air-borne infection,
found that twenty-four hours must elapse, on an average, before the
descending colon of the new-born infant becomes filled with air taken in
by the mouth, and that the air is undoubtedly the means by which micro-
organisms first gain a foothold in the meconium and alimentary canal.
Escherich confirmed these observations and found that the meconium con-
tains numerous microbes, cocci, bacilli, and blastomycetes at the expiration
of twenty-four hours after birth. Escherich, it should be stated, believes
that germs can enter the intestinal tract through the anus, as he found
bacteria in the meconium within from three to seven hours after birth.
This fact has recently been corroborated by Schild's experiments.

Bacteria are found in meconium in great variety, several species of
cocci, bacilli, and saccharomyces being seen. With the beginning of
nourishment of the new-born, this picture changes. Escherich was the
first to point out the fact that the feces of breast-fed children exhibits
a very simple, typical bacterial picture. Exclusive of a few cocci, there
are found only slender, rod-shaped bacteria of uniform appearance.
The later researches of Tissier and Moro have shown that there is
found, not a pure culture of Bacterium coli, but apparently a mass of
Gram-positive bacteria, such as Bacillus bifidus, Bacillus acidophilus,
and Bacterium coli, with a marked preponderance of the former. The
stools of bottle-fed children, whether sterilized or unsterilized milk is
used, present a less simple appearance than those of the breast-fed ones,

resembling somewhat the picture seen in meconium and in feces under mixed diet. The most marked changes in the bacteria of the intestine are caused by a mixed diet. Even here, however, a few varieties greatly preponderate and determine the bacterial picture. From the stools of adults cultures of a great variety of other bacilli have been made. In the former case the bacteria may be regarded as obligate intestinal micro-organisms ; in the latter, as accidental invaders.

The greatest success thus far obtained in the isolation of fecal bac-teria has been achieved by Matzuschita. He has succeeded in cultivat-ing 44 varieties from 48 specimens of feces. Bacillus coli commune was always found, Bacillus mesentericus vulgatus (potato-bacillus) almost always, Bacillus subtilis very frequently, Bacillus aërophilus, Bacillus aquatilis sulcatus, Micrococcus luteus, aurantiacus, Staphylo-coccus aureus, albus, and citreus in about one-half of the specimens, and frequently Streptococcus (pyogenes (?)) and Penicillium glaucum. Nothing is known as yet of the proportionate numbers of the different varieties found in the stools. Certain it is that Bacterium coli commune vastly preponderates, as a rule, and with ordinary culture methods is frequently the only variety that develops. Nevertheless, it is well to bear in mind that with improving culture methods (notable advances have been made in recent years in acid and anaërobic media) other re-sults may be obtained.

A comparison of the multitude of bacteria in feces seen under the microscope with the relatively small number grown on culture plates may well lead one to the conclusion that the greatest part of the bac-teria are either incapable of development in culture or have died natur-ally. Klein estimates the latter at 98.9 per cent., while Matzuschita found the proportion of culturable species to vary widely in different specimens of feces.

Strasburger has recently added considerably to our knowledge of the number and relative proportion of fecal bacteria. The older investi-gators took as a basis for their estimates the number of bacteria devel-oped from a small portion of feces—usually 1 mg. Strasburger makes his estimate from the dry weight of the organisms contained in 2 c.c. of feces treated by a method seemingly unobjectionable and dependent upon centrifugation. The advantages of Strasburger's method are evi-dent. While with the older methods only an approximate idea could be obtained of the bacteria passed with the feces, with Strasburger's method we obtain the entire amount of all the bacteria contained in the excretion. It is, of course, self-evident that all estimates must be based upon the total quantity, as those bacteria found dead in the feces have been alive at some point of the intestine, have had an influence on the food taken, or have been otherwise active. Naturally, Strasburger's figures are considerably higher than those of his predecessors. They show that the feces of adult and nursling alike contain bacteria to the extent of one-third of the dried weight of the excretion. Adults pass an average of 8 gm. (dried) of bacteria daily. In dyspeptic conditions the average is 14 gm., though in exceptional cases 20 gm. is reached.

Where constipation is habitual, the average is 5.5 gm., and exceptional cases show 2.6 gm. Thus it will be seen that Strasburger's method is certain to play a prominent part in examinations of fecal bacteria in health and disease. Strasburger places the average daily passage of bacteria in adults at 128 billions. Sucksdorff and Klein, who considered only the culturable bacteria, are, of course, far below this figure. The former placed the number at 53.124 millions, the latter at 8800 milliards. Klein gives as the average nitrogen-content of the intestinal bacteria 4.39 mg. for the twenty-four hours ; Strasburger advances direct evidence for placing it at 0.827 gm. Thus, at least one-half the nitrogen in feces is attributable to the fecal bacteria.

When we consider the question of the source of the intestinal bacteria, it is indubitable that the organisms taken with food, combined with those contained in the saliva, form the seed from which the intestinal flora is developed. To these must be added those that reach the mouth with the air. Of the germs that are swallowed, some are destroyed by the gastric juices. In the case of others, the conditions existing in the intestines—notably a lack of acid media—are unfavorable to great multiplication. There are still others, however, that are immune to these conditions and retain the power to multiply enormously in the intestines, particularly in the large intestine. These last constitute the intestinal flora. No evidence has been adduced to prove the correctness of the foregoing process in detail, but numerous examinations of the bacterial contents of food, of the oral cavity, and of feces, together with the results of experiments, all tend to establish its truth. The following will serve as examples of the species found in food and drink : Moro succeeded without difficulty in demonstrating his Bacillus acidophilus in both mother's and cow's milk—it is the organism most numerous in infants' stools. In drinking-water, both good and bad, we find Bacterium coli largely present—it is also found as an obligate in feces under mixed diet. Out of 56 specimens of water from different sources, Weissenfeld found it present in all. Refik found it in nearly all cisterns and springs in Constantinople. Papasotiriu and Wolffin, as well, demonstrated its presence in bread, cereals, legumins, and flour. Lehman and Conrad found it in great numbers in sauer-kraut. Under certain conditions pathogenic bacteria are carried along with the ingesta, and these invaders may contest the field with the obligates. Let us take, for example, the cholera vibrio, Bacillus typhosus, the pyogenic cocci (streptococci and staphylococci), and Proteus vulgaris, all or any of which at times cause pathogenic states in the intestines. In numbers of cases these organisms have been demonstrated in food and drink. Experiments on animals have, in the majority of cases, proved that bacteria taken with the ingesta can be recovered in the feces. Lembke has furnished an interesting example. He mixed with the food of a dog a culture of Bacterium coli anindolicum, and observed that for a time it completely eliminated the Bacterium coli commune from the dog's feces. Success attended experiments with another variety of Bacterium coli. Macfadyen fed Staphylococcus aureus to dogs, and

five hours later found hordes of the bacteria in all parts of the intestinal tract.

The bacteria found in the mouth have to be considered in the second place. Miller, in particular, has shown that numerous bacteria are normally present in the buccal cavity, and that enormous numbers may accumulate there if the cleansing of the mouth is not attended to. Miller determined the number of bacteria present in a foul and neglected mouth, and found 1,140,000,000 bacteria. Organisms must, therefore, be continuously swallowed with the saliva, and Miller has demonstrated that many of them enter the intestine notwithstanding the acidity of the gastric juice, for of 25 different kinds of bacteria in the mouth, he succeeded in finding 12 in the feces and 8 in the contents of the stomach. The number of bacteria present in the feces is, however, probably not dependent on the number of the bacteria in the mouth to any great extent when the mouth is kept clean. According to Van Puteren, the number of bacteria present in the stomach-contents in breast-fed infants is directly dependent on the number of bacteria present in the buccal cavity. By washing out their mouths with sterilized water before and after they took the breast, he succeeded in greatly reducing the number of bacteria present in the stomach of healthy breast-fed children, and in 18 per cent. of the cases he got rid of the bacteria entirely.

It has already been mentioned that the stomach does not exert an active sterilizing influence, as is indeed evident from the fact that the bacteria originally present in the food and the saliva reappear in the feces. Miller, Baumgarten, Wesener, Macfadyen, and others proved this experimentally several years ago, and their investigations make it probable that even sporeless bacteria may pass unchanged through the stomach. Miller believes that only the less resistant bacteria are destroyed in the stomach, and that the stomach possesses germicidal powers only toward these weaker organisms when digestion is at its height—that is, when the hydrochloric acid of the gastric juice is at its maximum. The stomach-contents appear never to be quite sterile, although Minkowski has shown experimentally that gastric juice removed at the height of digestion may stand for weeks without the occurrence of any fungous growth; apparently, therefore, the germs that are present cannot multiply in an acid medium, and eventually perish. Within the body the circumstances are not precisely the same, for the bacteria that enter the stomach remain in it only for a few hours and then pass into the intestine, where conditions are more favorable for their growth.

The investigations of Capitan and Moran show conclusively that organisms are present in the stomach-contents even at the height of digestion. They examined 30 individuals two hours after eating, some of them being in good health and some being dyspeptic, and they always succeeded in finding at least three kinds of bacteria in the stomach-contents. Abelous found a still more luxuriant bacterial flora in the washings from his own stomach after a period of fasting. Coyon's findings agreed with this.

Bacteria may be introduced into the intestine from another source, namely, the blood. This, of course, can occur only in pathologic conditions. Buchner discovered that when subcutaneous injections of pure cultures of the vibrio of cholera were made in guinea-pigs, the vibrio appeared in large quantities in the intestine. Lichtheim had previously determined the same thing for the spores of mucor. Bacterial infection of the intestine from the blood never occurs in health.

The intestinal canal, which, as before explained, is first infected with bacteria from the air, then with those from the food and saliva, remains to the end of life a most fertile field for bacterial growth. The peculiar conditions under which the vegetation of bacteria proceeds in the intestines necessitate a certain degree of uniformity in their qualitative relations. As before stated, this applies chiefly to the intestine of the healthy nursling, in consequence of the absolute sameness of food as regards both nutrient and bacterial contents. In harmony with this is Escherich's opinion that the "normal intestinal flora of the suckling babe is the expression and one of the conditions of a normal function of the intestine."

Food, irrespective of its bacterial contents, has an influence on the intestinal vegetation by virtue of the quality of its nutrient matter. This was demonstrated by Lembke in experiments on dogs which he fed on bread, meat, and fat successively. Results showed that the vegetations remained practically unchanged under the bread and the fat diets, but underwent marked alteration under meat diet. However, under all three diets certain species of bacteria remained constantly present, or nearly so. These were Bacterium coli, a yellow sarcina, and a species of coccus. Kuisl found that the spirillum of Finkler-Prior, which had been plentifully in evidence in two healthy subjects under meat diet, disappeared when the subjects were fed exclusively amylaceous food. Purgatives and enemas alter the bacterial picture, at least for a time (Tissier).

We will refer here to the attempts that have been made, by means of agglutination methods, to demonstrate that in spite of their peculiarities the bacteria of the intestines belong to one single type. H. L. Smith found that the Bacillus coli of some nurslings is agglutinized by the serum of the immunized guinea-pig, yet the reaction failed in some cases. Kreisel had the same result with adults. If this result were positive under all circumstances, the conclusion would be justified that in the intestines of every individual a wholly peculiar species of Bacterium coli is formed, under biochemic conditions, specially adapted to its own host. On the other hand, we must not forget that these bacteria are daily increased in number by others that have been swallowed, hence the primary species can scarcely have an opportunity to develop undisturbed into an individual type. According to the experiments of van der Velde and von Radziewsky, the results of Smith and of Kreisel do not seem to merit general acceptance. Van der Velde found that the serum of a horse immunized against Bacillus coli was effective against 21 strains of different origins, and von Radziewsky found that

the various colon bacilli from one individual behaved differently in presence of an immune serum obtained from a single strain.

The various portions of the intestine differ in their bacterial contents both as to quantity and quality. Billroth has called attention to the fact that far more bacteria are found in the large than in the small intestine. Nencki, in examinations on dogs, observed only micrococci in the small intestine, and but few of these; the lower he searched, the more organisms (*i. e.*, rod-shaped bacteria) he was able to demonstrate. In the upper sections of the intestines of dead sucklings Escherich found only a very few germs of cocci and bacilli (Bacterium lactis aërogenes); in the lower portions—*i. e.*, below the cecum—he discovered numerous slender bacilli. Gessner also demonstrated on the cadaver that Bacterium lactis aërogenes and Streptococcus pyogenes are most numerous in the duodenum, and Bacterium coli in the large intestine. Kohlbrugge found the empty parts of the jejunum and ileum in animals sterile in all cases, but in the cecum there were regularly great numbers of Bacterium coli. He speaks of an autosterilization of the small intestine by its own fluids, but fails to adduce any proofs. As a result of his experiments Macfadyen is inclined to doubt the sterilizing powers of the intestinal and pancreatic secretions. He has found that biliary acids and their salts, as well as pepsin, are wholly ineffective as sterilizers, and hence in his opinion we must attribute effective germicidal influence to the organic acids in the chyme. According to v. Mieczkowski's examination of the fluid obtained by means of a Thiry's fistula of the small intestine, the pure intestinal fluid has no bactericidal properties whatever, nor does it exercise even an inhibitory influence on bacterial growth. The observations of Cushing and Livingood are worthy of note. These investigators had many opportunities in a number of operations to study the bacterial contents of the human intestine during life. They observed that when about six hours have elapsed after the last meal the duodenum and jejunum are nearly or entirely germ-free. The few germs found at times are chiefly cocci. By giving only sterile food and paying particular attention to the cleansing of the mouth for a few days before operation, they found they could count with certainty on the sterility of duodenum and jejunum. Under such conditions, of course, operation on these parts offers the best chances for success. In the upper part of the small intestine they found the bacteria of varying types, and decide that the character of vegetations in this part depends upon the bacteria ingested with the food.

The difference in the types of bacteria found in the large and in the small intestine is due, in the main, no doubt, to the different conditions for microbial life existing in the two portions, particularly as regards the characters of the nutrient media. The rapid progress of the chyme through the small intestine, and its long stay in the large, account for the quantitative differences. In their normal condition the intestinal walls are free from bacterial life. Only in the rabbit Bizzozero found numerous intracellular bacteria in the lymph-follicles of the processus vermiformis and of the ileum.

[According to Cushing and Livingood, the number of colon bacilli in the alimentary canal steadily increases from the duodenum to the ileocecal valve, where the maximum is reached. In the colon there is a very definite fall in their number. The duodenum is often sterile when not occupied by any food. The observations of Lorrain Smith and Tennant make it probable that the secretion of the intestinal mucosa exerts an inhibitory effect on the multiplication of micro-organisms, and that this is a more important factor than the reaction of the intestinal contents. The presence of intestinal worms leads to a marked increase in the number of bacteria in the immediate neighborhood of the parasite.—ED.]

The rôle of the pathogenic bacteria in the intestinal tract has not yet been fully determined. Pasteur first offered the suggestion that animals would not thrive on sterile food. Recent experiments in this direction have not yielded uniform results. Nuttall and Thierfelder delivered guinea-pigs aseptically—by Cesarean section—and saw them thrive and gain weight considerably under absolutely sterile food (milk). Schottelius conducted a series of experiments on chicks brought into the world germ-free and fed on germ-free food (steeped millet-seeds, finely divided hard-boiled white of eggs, and egg-shells). His results showed that the chicks steadily lost weight despite plentiful consumption of food, until at the end of a period varying from fourteen to thirty days, and after a loss in weight of about 32 per cent., all of them died. Meanwhile it still remains doubtful whether the solution of these conflicting results is to be sought in the difference between animal and vegetable diet or in other factors. It is further to be noted that Mrs. O. Metschnikoff observed that the larvæ of frogs developed but poorly on a diet of sterile bread. Levin's statement that the intestinal contents of animals of the arctic regions are either poor in bacteria or else entirely germ-free, has recently been disputed by Chauveau. Even though the results of recent experiments and researches do not justify any positive conclusions, particularly in regard to man, we still may conclude that in future a greater importance must be ascribed to intestinal micro-organisms in any consideration of the internal economy of the body than has heretofore been the case. No final conclusions can be formed of the mode of action of bacteria until more is known of their biology. However, we do know that in the small intestine the exclusive rôle of micro-organisms is to produce fermentation of carbohydrates, and in the large intestine the decomposition of albumin. The latter process occurs in the small intestine only under pathologic conditions—as, for example, in stenosis of the small intestine or of the upper half of the large. It may be assumed as probable that the liberation of cellulose, which may be of importance to the assimilation of vegetable matter, is brought about by intestinal bacteria. We instance in this connection the properties of Bacillus butyricus. A further function of intestinal micro-organisms is to produce peristalsis, inasmuch as their fermentation-products or acids incite the motorial action of the intestines. Well known, too, is the excessive peristaltic

activity of the small intestine when the hydrocarbon fermentation in it exceeds normal requirements. It is worthy of remark that Strasburger was successful in demonstrating a marked decrease of bacteria in feces in cases of chronic constipation.

Nothing is known at present of the influence of specific bacterial products on nutrition. As regards their amylolytic and proteolytic properties, the following may be said : Escherich concludes from his researches that bacterial activity in the intestines is of but secondary importance in the nutrition of nursing infants. The schizomycetes present do not change casein at all, and their action on the fats is slight and physiologically insignificant. Lactose alone is appreciably changed. It is split into lactic and carbonic acids and hydrogen by the Bacterium lactis aërogenes. According to Escherich again, the bacteria in the colon do not subsist on the ingested food, for under normal conditions none of it reaches them, but they live upon the secretions of the intestinal wall. Experiments on adults have led Macfadyen, Nencki and Sieber to the same conclusion. The sum-total of results from the experiments of these investigators is that the schizomycetes are entirely unnecessary to digestion. On the one hand, their patient, a woman with a deep-seated fistula of the small intestine, remained well notwithstanding complete occlusion of the large intestine, in which alone the decomposition of albumin takes place; on the other hand, the fermentation of carbohydrates occasioned in the small intestine by bacterial activity is to be regarded as a positive detriment, since the bacteria subsist on the ingested carbohydrates and naturally deprive the system of a portion of its nutriment.

We may mention, also, the view that the normal intestinal bacteria serve as a protection against pathogenic invaders. According to Metschnikoff, for example, the cholera vibrio is hindered in its development by other bacteria, such as torulæ and sarcinæ.

A question of importance is, Under what conditions may the intestinal micro-organisms assume pathologic significance?

Under the following conditions the intestinal bacteria may play a pathogenic rôle : (1) Increasing virulence ; (2) excessive multiplication ; (3) migration into the lymph- or blood-channels. As evidence of the influence of increasing virulence we may point to the findings in appendicitis, colitis, and epidemic dysentery, in which Bacterium coli, or organisms very similar to it, has been found in enormous numbers, and practically alone ; or, again, to the enteritides, in which we find virulent streptococci, the Bacillus proteus. The more recent investigations of Shiga and of Kruse into epidemic dysentery have shown that, despite their similarity, Bacillus dysenteriæ and Bacterium coli are to be regarded as distinct types. Their agglutinating properties, as found in experiments in immunity, indicate that they are separate entities. It would seem, therefore, that in dysentery we are confronted not with Bacterium coli of increased virulence, but with a similar, though wholly specific, pathogenic bacterium. Whether or not the autochthonous Bacterium coli commune can, under certain conditions,—*e. g.,* changes

in the " disposition " of the organism,—assume pathogenic properties
in the intestines must, for the present, remain undecided. Nor can we
accept as proof the fact that Bacterium coli commune causes diseased
conditions when it invades parts not normally its habitat, such as the
gall-bladder, urinary bladder, peritoneum, thyroid gland, skin, etc. It
is equally uncertain whether we are to consider such streptococci as
Bacillus proteus, etc., found in many of the enteritides and regarded as
pathogenic, as autochthonous intestinal parasites of increased virulence
or as invaders. Experiments in Escherich's clinic seem to indicate the
presence of a true infection. Pigeaud, on the contrary, was uniformly
successful in cultivating streptococci from the feces of nurslings with-
out intestinal disease. He goes so far as to discredit the existence of
streptococcic enteritis, though for this there appears to be insufficient
reason, as his streptococci were not pathogenic for any animals, while
those of Escherich were, at any rate, effective against white mice.
There are other reasons, based on experiments, for accepting as prob-
able the theory of increased virulence of the intestinal bacteria. Thus,
Valagussa found that the colon bacilli of the cat were more virulent
with vegetable than with meat diet. He found increased virulence of
the bacilli also in decomposing feces. According to Coco, the Bacillus
coli increases in virulence in presence of staphylococci and streptococci.
Mixed cultures introduced into the intestines of animals caused fever ;
pure cultures did not.

It is a well-established fact that obligate intestinal bacteria may
multiply beyond normal limits and thereby assume pathogenic proper-
ties. A frequent forerunner of such an occurrence is intestinal dys-
pepsia (caused most frequently by improper food), which affords the
zymogenes of the small intestine an unusually favorable medium for
development. The bacteria of the small intestine, usually small in
number, multiply greatly, and carbohydrate fermentation is correspond-
ingly increased. This leads to extreme acidification of the chyme, and,
by extension, to irritation of the intestinal wall, diarrhea, flatulence, etc.
It seems to be perfectly safe to state that intestinal parasites cannot,
physiologically, leave their normal abode by way of the blood- or lymph-
channels. The experiments of Neisser, Opitz, Buchbinder, and Mar-
cus, conducted with greatest care, leave no room for doubt on this
point.

In organic disease of the intestinal walls, such as erosions or ulcera-
tions, the possibilities of infection of the peritoneum or the blood-
channels are, of course, greatly increased. Our daily experience shows
this, and Klecky and Buchbinder have demonstrated it experiment-
ally. Buchbinder brought to his experiments an unobjectionable
technic ; his results show that even in extreme disturbances of circula-
tion in the intestines the bacteria do not migrate to the serosa, as that
would require more extensive injury of the tissues, with subsequent
gangrene. Klecky demonstrated in intestinal necrosis a simple pro-
liferation of the bacteria as far as the serosa. In marked venous
stasis he found bacteria in the vessels of the mucosa, submucosa, and

subserosa. In proof of the transmigration of obligate and facultative intestinal bacteria we instance the following points : The frequent development of miliary tubercles on parts of the serosa affected with tuberculous tumors ; the occurrence of ascending inflammation of the mesenteric veins in non-tuberculous tumors, generally attended by diapedesis of the bacteria into the blood-channels ; peritonitis resulting from dysentery and from strangulation of the intestine.   .

Disinfection of the intestines is a problem that has awakened interest everywhere, yet up to the present time it has found no solution. Investigators turned their efforts in this direction in the hope that the extermination of intestinal bacteria, as far as such a thing is possible, would remove their decomposition-products as well and cure, or at least alleviate, many of the concomitant ills. A brief period of optimism (Bouchard) consequent upon the rapid advances in chemistry and the discovery of numberless antiseptics was followed by a return to more sober thought, and to-day we have arrived at the conclusion that actual disinfection of the intestines is impossible.

Experimentation with some of the so-called intestinal antiseptics, like the use of sterile food, has given no reliable diminution in the number of intestinal bacteria (Stern). Fr. Müller was, therefore, right when he said : " None of the so-called intestinal antiseptics really deserves that name." There is only one means of securing anything like intestinal antisepsis, and that is a thorough emptying of the bowels. To this end calomel ranks first in effectiveness. Not only is it a powerful purgative, but, according to R. Stern, it has the additional advantage that all, or nearly all, bacteria removed with the calomel stools are completely destroyed after twenty-four hours. From this observation of R. Stern we may conclude that when calomel is exhibited and purgation does not follow sufficiently soon, it is quite possible that the calomel is exerting within the intestines a restraining influence on the development of the bacteria. Von Mieczkowski observed that the chyme from the small intestine after free exhibition of menthol is not a suitable culture-medium for intestinal bacteria. The last two observations explain why the use of antiseptics is not wholly valueless, despite their inability to produce any great reduction in the intestinal bacteria. It is well known that the administration of such remedies as resorcin and menthol is often effective in cases of intestinal dyspepsia accompanied with skin eruptions, such as furuncle or urticaria. The persistent use of saline waters (Marienbad, Carlsbad) has frequently been of value in such cases, evidently by producing regular movements of the bowels. [Herter concludes that there is no safe intestinal antiseptic which can be relied upon to exert a distinct effect in reducing intestinal putrefaction. In connection with estimation of the ethereal sulphates in patients taking intestinal antiseptics of the aromatic class, such as creasote, it should be borne in mind that these drugs produce ethereal sulphates which are indistinguishable from those due to putrefaction.—ED.]

The failure of intestinal antisepsis, pure and simple, led to the discovery of other means of influencing intestinal micro-organisms. Fr.

Müller recommended the use of substances that exert a stimulating and astringent action on the intestinal cells, such as bismuth, silver, and tannin. It occurred to some that the intestinal bacteriology could be changed by the introduction of cultures of bacteria (bacteriotherapy), and this has been attended with success—witness the cures of furunculosis and diarrhea with brewers' yeast. Brudzinski administered Bacterium lactis aërogenes to nurslings with foul-smelling clayey stools showing great masses of Proteus vulgaris. This was followed by improvement in the constitution of the stools.

Change of diet is another valuable means of modifying the intestinal flora, and consequently the chemic processes in the bowels as well. We have already referred to the influence of foods on intestinal microorganisms. It follows from this that the diet of a patient should be radically modified, the nature of the case and the end in view being the guides. Albumin decomposition seems to be best combated with an exclusively milk diet, one in which milk greatly preponderates, or one with diminished albumin-contents, relatively small fat-contents, and rich in hydrocarbons to supply the needed quantity of calorics (Backmann). In intestinal dyspepsia from excessive carbohydrate fermentation the proper course would be to reduce the carbohydrates and to replace them with albumin and fats.

In examining the feces for micro-organisms, all the methods of bacteriology must be employed. The sample should be previously examined with the naked eye for the presence of blood, mucus, pus, etc., and microscopically with a view to the detection of undigested food, mucus, epithelium, etc. The reaction (acid or alkaline) must always be tested. The amount of food eaten during the twenty-four hours preceding the evacuation of the sample and the number of defecations should be noted. The results of the investigation will be useless for purposes of comparison unless all these precautions are observed. After these points have been determined, the investigation of the bacteria may be proceeded with.

In order to gain a general idea of the bacterial flora a small quantity of recently voided feces may be diluted with a little water (when the stools are liquid, dilution is superfluous), and immediately examined without staining or other manipulation. In inspecting fresh specimens of stool the relative number of germs present, the morphology of the different germs, and their motility should be studied. Then stain some of the dried feces with dilute fuchsin or methylene-blue. Treat other specimens by the method of Gram or Weigert, and stain with fuchsin, as recommended by Escherich. By these means we get a good idea of the Gram-negative and Gram-positive bacilli, streptococci, and staphylococci present. It is advisable also to treat a specimen in Lugol's solution, to determine the presence of bacteria giving the amyloid reaction. This reaction is no longer regarded as specific for Clostridium butyricum, for Grassberger and Passini were able to obtain it with three varieties

of aërobes cultivated from meconium, and under certain conditions with Bacterium coli as well.

The cultivation of intestinal bacteria requires the best possible bacteriologic apparatus, and we confidently expect that there will be many improvements. For the most detailed description of the various culture-media and their productiveness we refer to the works of Matzuschita-Teisi.

The bacteria in cultures are counted by the plate method or by the new Strasburger scale. From the former we gain an approximate idea of the number of the cultivable fecal micro-organisms, but as a considerable number of fecal bacteria do not develop on plates, the plate-method must gives results below the actual value. Strasburger's method ascertains the total quantity of both the living and dead organisms.

We will now proceed to enumerate the best-known species of microbes found in the feces and their most important characteristics.

I. BACILLI.

1. Bacterium Coli Commune (Escherich).—This bacterium is to all appearances constantly present in the feces. It is always found in the meconium and in the feces after a milk or a meat diet. With mixed diet and under normal conditions it forms by far the greatest part of the fecal bacteria. It is found in particularly large numbers in the large intestine.

Bacterium coli commune is a pleomorphous organism, showing great differences in size, form, and motility, as well as in biologic and pathogenetic properties. Escherich was the first to call attention to these manifold differences, but he did not feel justified in subdividing the "group" of colon bacteria into several species, not considering the differences sufficiently marked nor sufficiently constant to warrant such a subdivision. Since that time numerous authors have attempted to arrange a number of subspecies of colon bacteria, but none of these attempts has been successful; consequently we shall not pursue the subject, for in this place we are chiefly concerned with the practical application of our knowledge of intestinal bacteria and not so much with bacteriologic detail. We shall limit ourselves, therefore, to a brief description of the more important variations that the different colon bacteria show.

[In connection with this paragraph it may be mentioned that Durham, in 1898, formulated a division of three groups which has generally been adopted; these groups are: (1) The Eberth group, Bacillus typhosus and its allies; (2) the Gaertner or intermediate group, Bacillus enteritidis, bacillus of hog cholera, Bacillus psittacosis, paracolon, paratyphoid; (3) the Escherich group, the typical Bacillus coli communis. A fourth group, that of the aërogenes, may also be made.

A great deal of work has been carried out with regard to the varieties of the colon bacillus. Gordon, in 1897, distinguished 22 distinct varieties, and Pakes, in 1901, examined 52 strains which he considered

as typical colon bacilli and well differentiated from paracolon and other groups.

The term paracolon was introduced by Gilbert to describe members of the colon group which differ in a few reactions from the typical colon bacillus. Widal and Nobecourt described a paracolon bacillus from an abscess in the thyroid gland in 1897. In 1898 Gwyn reported a case of paracolon infection which presented all the clinical aspects of typhoid fever; this form of infection has also been spoken of as paratyphoid fever, and Johnston has analyzed 26 such cases. Cushing regarded the paracolon bacillus as allied to the bacillus of hog cholera and Gaertner's Bacillus enteritidis, and belonging to the intermediate division of the colon group between the typical colon bacillus and the Bacillus typhi. Cushing and Durham have worked extensively at the mutual serum reactions of the various members of this intermediate group.—Ed.]

The size and form of the colon bacteria vary greatly. Some specimens can hardly be recognized as bacilli, for they are nearly as broad as they are long; these forms are usually very small, their diameter rarely exceeding $0.5\,\mu$. [Adami and his pupils have called attention to the diplococcoid form of the colon bacillus.—Ed.]

Other specimens are oval in outline or show slight lateral indentations, and some of them form slender rods. The length of these elongated forms is usually from 2 to 3μ, their width from 0.4 to $0.6\,\mu$. Double rods are also frequently seen.

The motility of the colon bacteria is very inconstant. Occasionally they move with the greatest rapidity; at other times they seem to be perfectly quiescent. Stoecklin, therefore, differentiates motile and non-motile species, estimating that the former constitute two-fifths, and the latter three-fifths, of the whole group. According to Matzuschita, the non-motile bacteria regain their motility when transferred repeatedly to different culture-media.

In fresh feces motile forms are common. The double rods spoken of above show this property with particular distinctness. Occasionally one rod may be in active to-and-fro movement, while the second one is motionless, so that the first rod appears to be dragging the second one along with it. The colon bacteria possess flagella, which are their means of locomotion. According to Nicolle and Morax, these bacteria rarely possess more than 6 flagella, and it is very exceptional for them to have as many as 8 or 10. Typhoid bacilli, on the other hand, according to the same authors, always possess more. According to Bunge, however, Bacterium coli commune often has very numerous flagella.

Bacterium coli commune can be readily stained with the ordinary anilin dyes. Dilute carbolfuchsin stains it excellently within five minutes. Bacterium coli commune stains only with Gram's method if it is cultivated or has been growing on a medium containing much fat. This fact was discovered by Schmidt. The formation of spores has never been observed in this bacterium. It is easily cultivated on the ordinary media. On meat-peptone-agar slants it forms a white, thin, juicy-look-

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ing growth that can be readily scraped off, and is slightly iridescent when viewed by transmitted light. Cultivated on peptone bouillon, it gives the indol reaction. The growth on gelatin plates is not characteristic. The deeper colonies are light in color and occasionally show a radiating or a concentric structure. The superficial colonies usually grow larger than the deep ones, and may be either perfectly smooth or finely granular, or may show delicate furrows and ridges. Frequently a concentric arrangement and umbilication may be seen. The margin of these colonies is sharp and lobate. On potato the colonies grow luxuriantly and form a brownish-yellow, juicy, shiny layer that extends over the greater portion of the surface of the potato. In this culture ammonia is developed (Escherich, Pfaundler). Bacterium coli commune acidifies milk and causes curdling in the course of a few days. The curd is firm and lumpy.

Bacterium coli commune grows under anaërobic conditions only on media containing dextrose; the latter undergoes fermentation, and Escherich has determined the presence of CO_2 and H in approximately equal parts in the gas that is evolved. Macfadyen, Nencki and Sieber, on analyzing the solutions containing the products of this anaerobic dextrose fermentation, found ethyl alcohol, acetic acid, and inactive lactic acid. They performed their experiments with Bacillus bischleri, an organism probably identical with Bacterium coli commune. They also found that the Bacillus bischleri does not decompose albumin.

Escherich grew the Bacterium coli commune on milk, which he afterward analyzed, and ascertained that neither the milk-sugar nor the casein were acted on to any great extent. Pfaundler observed that Bacterium coli exerts no proteolytic action on the serum from cow's blood, but that they do so on peptones and albumoses, with a plentiful splitting-off of indol. He found also that free starch is not affected by the colon-bacillus. The most characteristic property of this microbe is its power of causing dextrose to ferment; occasionally, however, the reaction fails to take place. Ury cultivated two varieties of the Bacterium coli commune—one from a case of cystitis, another from a case of cholecystitis—and found that neither of them could ferment dextrose or curdle milk.

Experiments performed on animals with Bacterium coli commune show that its pathogenic power is not constant. Escherich found that subcutaneous inoculation of mice with it did not produce any effect; on the other hand, intravenous injection of small quantities caused the death of guinea-pigs within twenty-four hours. On autopsy an intense intestinal catarrh with swelling of the plaques, and occasionally some serous exudation in the peritoneal cavity, were found. The lesions were found chiefly in the duodenum and throughout the whole small intestine, and became less frequent, less intense toward the colon. If small quantities of colon bacilli are injected subcutaneously into guinea-pigs, an abscess may be formed, or, on the other hand, no reaction whatever may occur; if large quantities are injected, the same phenomena appear as after intravenous injection. Rabbits react similarly to guinea-pigs, but

death occurs somewhat later in them than in guinea-pigs, and peritonitis does not supervene. It has been remarked before that Bacillus coli develops greater virulence in the presence of streptococci and staphylococci. According to Dreyfus, the virulence of colon bacilli cultivated from normal feces is much less than that of the same organism cultivated from the excreta of dysentery, typhoid fever, gastro-enteritis, and cholera nostras.

Bacterium coli plays a very prominent rôle in human pathology. A number of diseases are recognized in which this micro-organism figures quite regularly as the causative factor. We may cite cholecystitis, cholangitis, scolecoiditis, pericarditis, meningitis, cystitis, pyelitis, pyelonephritis, strumitis, tonsillitis, panaritium, and gas-phlegmons. Further instances are cutaneous ulcers, with a strong-smelling, dirty-gray surface, and evincing little tendency to repair. They are caused by infection of wounds with Bacterium coli commune. Finally may be mentioned some infrequent cases of septicemia.

Bacterium coli commune seems to be widely disseminated throughout nature. Henke, for instance, succeeded in finding it in bandages. It has been found in drinking-water, flour, dough, cereals, and sauerkraut.

Bacterium coli commune is so universally present that it has been, so to say, repeatedly rediscovered by sundry observers, and consequently a variety of names have been given to it. The identity of some of these organisms was ultimately recognized, but not until the characteristics of the Bacterium coli commune were generally known to bacteriologists. The following organisms must be considered identical with Bacterium coli commune : Bacillus neapolitanus (Emmerich) ; Bacillus pyogenes fœtidus (Passet) ; Bacillus pyogenes of the bladder. Very closely related to Bacterium coli are : Bacillus endocarditidis (Gilbert and Léon) ; Bacillus enteritidis (Gaertner) ; the bacillus of epidemic dysentery of Chantemesse and Widal ; Bacillus endocarditidis griseus (Weichselbaum), and the bacterium of typhoid fever.

Attention has already been called to the fact that the differentiation between Bacterium coli commune and the bacillus of typhoid fever may, under certain circumstances, be very difficult. Up to the present time the two have been distinguished by the circumstance that the Bacterium coli commune brings about the curdling of milk, the fermentation of dextrose, and the acidification of alkaline culture-media, whereas the bacillus of typhoid does not possess these properties. Attention has already been called to the fact that occasionally these typical reactions do not occur, so that these methods of differentiation are not always satisfactory.

Cesaris Demel and Orlandi have recently succeeded in immunizing animals with the bacillus of typhoid fever against the Bacterium coli, and conversely they have used the Bacterium coli commune for the immunization of animals against the bacillus of typhoid fever. Great expectations were entertained that the agglutination-test would prove a conclusive means of differentiation, but these have not been

realized. Frequently the serum from typhoid patients agglutinizes the colon bacilli even better than the typhoid bacilli themselves (R. Stern). Again, the serum of animals immunized to colon bacilli acts upon only a limited number of strains, and further typhoid immune-serum agglutinizes many of the strains equally as well as typhoid cultures (Sternberg). Thus it will be seen that a differentiation of the two types must take into consideration all their cultural, morphologic, and biochemic characteristics.

[For a summary of the means of distinguishing the Bacterium coli commune from the Bacillus typhosus the reader is referred to Horton Smith's Goulstonian lectures for 1900. The indol test, which depends on the fact that certain members of the colon group produce indol which the typhoid bacilli do not, is open to considerable objection. The agglutination test in sufficient dilution is the most valuable, and should be employed not alone, but in association with other tests for the differentiation of the Bacterium coli and Bacillus typhosus. When incubated in tubes containing media tinged with neutral red, Bacterium coli reduces the red to a fluorescent yellow color, while Bacillus typhosus does not (Rothberger, W. Hunter). This reaction is very useful in distinguishing between these two bacilli, but the reduction of neutral red is not specific to the colon group, since it is brought about by the Proteus vulgaris, Bacillus prodigiosus (Pakes).—Ed.]

2. Bacterium Lactis Aërogenes (Escherich).—Escherich found this bacterium in large numbers in the intestinal canal of animals and of human subjects who were fed on milk, but it occurred sparingly in the feces of the same individuals. Tissier found it but seldom in the stools of breast-fed children, and almost constantly in that of bottle-fed ones. This organism is on an average 1 to 2 μ long, and 0.5 to 1.6 μ broad. There are, therefore, short forms which, by the free rounding off of their angles, eventually appear as round or oval objects of some 0.5 μ in diameter. Most of the elongated forms are contracted in the middle, so that they may even simulate a diplococcus. Escherich failed to observe the formation of spores, of degenerative forms, or of outgrowths. This organism stains readily with anilin dyes, but not by Gram's method. It is non-motile. Cultivated on gelatin plates, it forms white dots if kept at 22° C. for twenty-four hours. The superficial cultures are round, convex, juicy, and shiny. The surface of the colonies is homogeneous. In gelatin stab culture the growth is beaded. The gelatin is not liquefied, and the surface growth capitulate. On agar the growth is very abundant and juicy. On potato the growth is creamy, deliquescent, and whitish or light yellow in color. The colonies contain gas-bubbles. The bacterium causes curdling of milk within sixty hours. The coagulum is lumpy, and lactic acid is formed. According to Escherich's investigations, about 58 per cent. of the sugar of milk originally present undergoes fermentation. The Bacterium lactis aërogenes possesses slightly proteolytic powers of about the same strength as those of the Bacterium coli.

This bacillus is capable of fermenting cane-sugar and dextrose as well

as lactose. It can grow under anaërobic conditions on culture-media containing lactose, cane-sugar, or dextrose, and can ferment these different sugars even in the absence of oxygen.

The most typical and pronounced property of Bacterium lactis aërogenes, according to Escherich, is its power of very actively fermenting lactose. No other bacterium present in the intestine possesses this property. The fermentation of lactose occurs even in the absence of air, but proceeds more rapidly if oxygen is admitted. The gases developed in this process of fermentation are, according to Escherich, CO_2 and H. In one of his anaërobic fermentation tests performed with sterile milk, the proportion of the two gases was the following : $CO_2 : H = 100 : 61.5$. At the same time there was a loss of sugar of from 3.51 to 2.23 per cent. The acid that is formed is, according to Escherich, lactic acid. Macfadyen, Nencki and Sieber also studied the fermentation of dextrose by a bacterium similar to the Bacterium lactis aërogenes, and probably identical with it ; they found that alcohol, acetic acid, and paralactic acid were formed.

The pathogenic effect exercised on animals by this organism is very similar to that exercised by the Bacterium coli. Denys and Brion discovered that when large numbers of this bacillus from potato cultures were killed with alcohol or ether, the bodies were very poisonous and capable of killing rabbits when injected intraperitoneally.

The pneumobacillus of Friedländer is closely related to the Bacterium lactis aërogenes. Microscopically, they cannot be distinguished, and they present identical appearances in gelatin stab cultures. According to Escherich, the bacillus of Friedländer, when cultivated in milk under anaërobic conditions, produces very much less gas than the Bacterium lactis aërogenes under the same conditions. According to Denys and Brion, the pneumobacillus of Friedländer grows less rapidly on various culture-media than the Bacterium lactis aërogenes. These authors did not succeed in finding any differences between the two organisms in regard to their pathogenicity for animals. At present, therefore, most authorities agree that the two bacteria are varieties of the same species. The bacillus of rhinoscleroma belongs to the same species.

The Bacterium lactis aërogenes does not seem to play an important rôle in human pathology. Physiologically, however, it is exceedingly important, owing chiefly to its power of fermenting lactose in the absence of oxygen. This process, which occurs principally in the intestine of breast-fed infants, exerts a great influence on the processes of digestion that go on in the small intestine.

[B. Goldberg describes postgonorrheal vesical bacilluria produced by the Bacillus lactis aërogenes.—ED.]

3. Bacillus Bifidus Communis (Tissier).—This bacillus was formerly mistaken for the Gram-positive variety of colon bacillus found in the stools of nurslings. Tissier succeeded in cultivating it under strict anaërobic methods, and proved its individuality. He states that it is found in almost pure culture in the stools of breast-fed children,

and in lesser numbers and mixed with other varieties in stools of bottle-fed children. It is recovered with great difficulty from diarrheal stools.

It occurs as a bacillus with somewhat tapering ends, 2 to 4 μ long. Frequently two are united as a diplobacterium, but in that case the two always lie parallel to each other. Their bifurcation and vacuolization are striking. This is noticeable in the stools, and better in older cultures. They result from degenerative processes, and are not further transmissible.

They are fixed with the basic stains, but in the case of the degeneration forms the staining is imperfect.

The young bacilli stain well by Gram's method, while the older forms stain either poorly or not at all. Hence, when fuchsin is employed as a contrast stain, we find specimens colored red, blue, and also both shades. It is non-motile, dies at 60° C., and does not sporulate.

It is an obligate anaërobe, growing best at 37° C. On sugar-agar it develops well in deep-seated growths in the form of white, lenticular bodies. It does not grow in ordinary agar, nor in sugar gelatin, but flourishes in sugar bouillon and oxygen-free milk without coagulating the latter. The sugar media develop acid reaction with the growth of the cultures. The bacillus is non-pathogenic.

4. Bacillus Acidophilus (Moro).—Moro claims this as the Gram-positive micro-organism occurring in almost pure culture in the normal stools of breast-fed children. Tissier denies that it is identical with his Bacillus bifidus, and asserts that Bacillus acidophilus is not found in the stools of healthy breast-fed children, but only in those of infants fed on ordinary and sterilized cow's milk.

Moro isolated it from the stools in weakly acid media, such as wort agar. It is 1.5 to 2 μ in length, 0.6 to 0.9 μ in width, and tapers toward each end. In culture, true ramifications are formed. It is finely Gram-positive both in stools and cultures. Is a non-motile, facultative anaërobe, grows well on acid media, but not on alkaline (Rodella). Isolation from feces by means of acid media is preferable, as this obviates contamination of cultures by Bacillus coli. Develop only at temperature necessary for incubation. On slant-cultures they form a dry film ; in acid bouillon they develop freely, as also in milk, which is coagulated in three days. There is acid-formation, no gas-formation, and no growth on potato medium. They are non-pathogenic.

5. Bacillus Exilis (Tissier).—This species is found frequently in stools of infants fed on ordinary or sterilized cow's milk and on mixed diet.

It occurs as an almost straight, stiff, short, rod-shaped micro-organism, is mainly single, but occasionally is found in short chain formation. It is a Gram-positive, non-motile, facultative anaërobe, does not sporulate, and is of slight vitality in culture. It forms small, transparent or whitish dots on sugar-agar, which soon diappear. It develops poorly in bouillon and coagulates milk in eight to ten days. It does not grow in gelatin, potato, or acid media.

6. Bacillus Subtilis (Ehrenberg).—The distribution of this

micro-organism is very wide-spread. Its presence in the feces was first determined microscopically by Nothnagel. Escherich subsequently cultivated it from meconium. I have repeatedly succeeded in cultivating this organism from the feces of adults. According to Boas, the bacillus is extremely abundant in the contents of the stomach.

Bacillus subtilis forms cylindric rods of considerable length and breadth. Fully developed specimens may be 6 μ long and 0.6 μ broad, or even larger. Frequently a number of them unite to form long chains. At a certain stage of its development this organism is in active serpentine motion. Its motility is due to the presence of long flagella that are attached to its poles. It multiplies by division and in this way forms long rows of bacilli. After a short time spores develop. The latter are 1.2 μ long and 1.6 μ broad, and usually appear near the poles of the bacilli. Very soon after the formation of these spores the rods become disintegrated, liberating the spores, which have a gelatinous capsule. Both the bacilli and the spores can be stained without difficulty and can be easily grown on a variety of culture-media.

On gelatin plates this bacillus forms white colonies with an irregular margin in twenty-four hours. From the periphery of each colony a large number of filaments radiate, presenting the appearance of a corona around the growth. In course of time the opaque central colony becomes surrounded by a narrow transparent zone, and around this again is seen a gray ring that represents the corona of radiating filaments already described. The gelatin is rapidly liquefied ; at the same time that portion of the colony which consists chiefly of the above-mentioned filaments remains as a tough film that can be easily raised from the liquefied gelatin. The colonies develop rapidly even at the ordinary temperature, and may grow to be several centimeters in diameter. On agar (streak culture) a moist white layer forms that occupies the whole surface of the agar and soon becomes rugose. In gelatin stab cultures the liquefaction of the gelatin rapidly produces tubular cavities with simultaneous formation of a tough, uneven film on the surface. On bouillon the same characteristic film makes its appearance and at the same time a slight sediment is deposited. The greater portion of the bouillon, however, remains clear. Occasionally the cultures in bouillon emit a musty odor. Blood-serum is liquefied. On potato there is a luxuriant growth of yellowish colonies that afterward turn brown. They rapidly cover the whole surface of the potato and form a wrinkled layer in a few days ; at the same time the potato shrivels up. Milk is curdled in the course of a few days, and its reaction becomes slightly acid or neutral. The Bacillus subtilis is incapable of fermenting carbohydrates, but possesses very great proteolytic powers. This organism requires oxygen for its growth, and is an obligate aërobic microbe. Under physiologic conditions the Bacillus subtilis is, therefore, unable to multiply in the intestine, where no oxygen is present. For this reason it is of no importance in the physiology of digestion. It also appears to be pathologically inert.

It should be mentioned that bacteria are frequently found in the feces that are very similar to the Bacillus subtilis, but are not absolutely

identical with it. Bienstock has described two of them as Bacillus subtilis simulans I. and II.

7. Proteus Vulgaris (Hauser).—This bacillus has been found in meconium by Escherich, and is constantly present in the feces of dogs fed on meat. Maggiora succeeded in demonstrating the presence of this organism to a small extent in 11 cases of dysentery, in which it was associated with Bacterium coli. Baginsky found it in the evacuations of breast-fed infants suffering from diarrhea. It forms rods of varying length and an average thickness of 0.6 μ. It may present the appearance either of spheroids or of slender bacilli from 1.25 to 3.75 μ in length or of long threads. The latter are frequently twisted or coiled. This organism is stained without difficulty. The color is removed by alcohol or by Gram's method.

The growth of proteus on nutrient gelatin containing much water (preferably about 6 per cent.) is characteristic. Within from six to eight hours, at the ordinary temperature, liquefied depressions appear which are surrounded by a narrow ring of bacteria. From the periphery of this ring a broad, thin, superficial layer extends in all directions; it sends out processes and can be recognized by the fact that those portions of the gelatin that are covered by it are not shiny, but dull. Gradually islets become separated from the main mass of this layer and extend slowly over the surface of the gelatin, which soon becomes covered with them and may be completely liquefied at the expiration of one or two days. A corona of radiating filaments starts from the deeper colonies, and the gelatin is liquefied over the whole area covered by these structures. At the periphery of the liquefied portions the above-mentioned spirilla or spirulina are to be seen, often in association with spiral or club-shaped masses of zoöglea. The chains of bacilli which constitute the corona of radiating filaments are in active to-and-fro movement. Blood-serum is rapidly liquefied. On agar a white, thin membrane develops and extends in all directions; the agar itself is not liquefied. On potato, light yellow, flat, shiny colonies make their appearance. In bouillon there is a luxuriant growth, and the fluid gives a positive indol reaction. All these cultures are characterized by a pungent, ammoniacal odor. The growth of the organism is much slower when oxygen is excluded.

Among the most important biologic properties of proteus must be reckoned its power of causing putrefaction of proteids and ammoniacal decomposition of urea, both under aërobic and anaërobic conditions (Schnitzler). Among the decomposition-products of proteid in long-kept meat cultures of proteus Carbone found cholin, ethylendiamin, gadinin, and trimethylamin.

Experiments on rabbits gave the following results : Subcutaneous injection of Proteus vulgaris produced gangrene and abscesses. Injection into serous cavities produced either fibrinopurulent or sanious inflammation. Intravenous injection gave rise sometimes to uncomplicated toxic symptoms and sometimes to pyemia. Introduced into the bladder, it set up cystitis with simultaneous formation of ammonia

(Schnitzler). The pathogenicity of this organism seems to fluctuate within wide limits. Proteus vulgaris is a frequent cause of cystitis associated with ammoniacal decomposition of the urine (Krogius, Schnitzler). According to the investigations of Jaeger, it is also the main etiologic factor in Weil's disease. [In a recent paper Satterlee has given a table showing the characters of the bacilli found by Weil, Jaeger, Brooks, Libman, and himself.—ED.] Hajek and Schnitzler report that it is frequently present in ozena. Proteus has also been found in isolated cases of inflammation of the middle ear and of phlegmon. It is regularly present in suppurating carcinoma of the uterus.

Several organisms known under other names are identical with Proteus vulgaris, including Urobacillus liquefaciens septicus (Krogius) and Bacillus ozænæ fœtidus (Hajek).

8. Bacillus Mesentericus Vulgatus (*Potato Bacillus*) (Flügge). —This species was found by Matzuschita almost regularly in forty-eight specimens of feces. It is a shorter and narrower form than Bacillus subtilis, and is motile. It forms large spores, highly resistant to boiling. It liquefies gelatin, and forms on agar and potato media a thick, white, corrugated layer. It coagulates milk, the coagulum being, for the most part, redissolved.

Matzuschita has found simultaneously, but less frequently, Bacillus mesentericus fuscus et ruber.

9. Bacillus Aërophilus (Flügge).—This has been frequently demonstrated in feces by Matzuschita. It is more slender than Bacillus subtilis, is non-motile, forms oval spores, and rapidly liquefies gelatin. On potato it forms a yellowish, faintly bright, smooth layer. It is an obligate aërobe.

10. Bacillus Aquatilis Sulcatus IV. (Weichselbaum).—This species has been frequently demonstrated in feces by Matzuschita. Morphologically it is similar to Bacillus typhosus. It is very motile, Gram-negative, does not sporulate. On gelatin plates it appears like Bacillus typhosus, but it is obligate aërobic, therefore it does not show growth in depths of puncture. It forms no indol, does not ferment sugar, nor coagulate milk. It is non-pathogenic.

11. Bacillus Putrificus Coli (Bienstock).—According to Bienstock, this bacillus is constantly present in the feces, except when the diet consists only of milk. Escherich observed organisms morphologically similar to it in meconium, but did not succeed, like Bienstock, in making cultures of them. According to Boas, this bacillus is frequently found in the contents of the stomach.

It forms slender bacilli which are about $3\,\mu$ in length, but frequently develop into very long threads. The bacilli are very motile. The method of spore formation is characteristic, for the spores are invariably terminal, and form small round bodies at either one or both ends of the organism. Examined at this stage, the bacillus presents the form of a drum-stick or of a so-called "Köpfchenbacterium" (knobbed bacterium). The knob—that is, the spore—is liberated, owing to the disintegration of the body of the bacillus.

It is stated that the growth in agar stab cultures is at first shiny, like mother-of-pearl, afterward turning yellowish. Gelatin is not liquefied by Bacillus putrificus coli. According to Bienstock, the bacillus is capable of decomposing proteid very energetically, with the formation of ammonia, amin bases, amido-aliphatic acids, tyrosin, phenol, indol, skatol, etc. The decomposition of proteid occurs even when air is excluded, but the process is slower.

12. Bacillus Liquefaciens Ilei (Macfadyen, Nencki and Sieber). —This organism was cultivated by the three authors above mentioned from the intestinal contents of a female with a fistula of the small intestine. She was on meat diet, and the contents of her intestine always gave an acid reaction. Ciechomski and Jakowski rediscovered this bacillus in another case of fistula of the small intestine.

The bacillus is from 2 to 3 μ long and 0.4 μ broad, and is motile. It liquefies gelatin, does not curdle milk, and has practically no effect on dextrose. Proteids are decomposed. In this process a peculiar odor, as of old cheese, is emitted, but there is no formation of skatol, indol, nor methylmercaptan.

13. Bacterium Ilei (Frey).—This bacterium was found by Macfadyen, Nencki and Sieber in the same case as Bacillus liquefaciens ilei (No. 12). It forms bacilli from 2 to 3 μ long and 1 μ broad. The two species are frequently seen together, or there may be an association in clusters. The motility of the organism is very slight. It forms spores.

Bacterium ilei is capable of fermenting dextrose to methyl-alcohol, succinic acid, and active paralactic acid. It does not possess proteolytic powers.

14. Bacterium Ovale Ilei (Macfadyen, Nencki and Sieber).— This organism was cultivated by these three authors from the same case as the bacillus described under No. 12. The Bacterium ovale ilei forms such short bacilli that they might be taken for cocci. It does not liquefy gelatin, curdle milk, or produce any putrefactive change in proteids. It ferments dextrose to ethyl-alcohol, acetic acid, and paralactic acid.

15. The Slender Bacillus of the Ileum (Macfadyen, Nencki and Sieber).—This bacillus was cultivated from the same case as the bacillus described under No. 12. It is motile and does not form spores.

It ferments dextrose and forms alcohol, acetic acid (?), and paralactic acid. It does not decompose meat.

16. The Veil Bacillus (Escherich).—This bacillus is morphologically similar to the Bacterium coli and the Bacterium lactis aërogenes. The only way, in fact, in which these organisms can be differentiated is by their cultural peculiarities.

The " veil bacillus " of Escherich grows less abundantly on gelatin than either of the two organisms just mentioned. In stab cultures growth occurs exclusively at the surface. The appearance of this surface growth is that of a white, leaf-shaped colony with serrate margins and a dry surface. The growth along the stab forms a thin canal that

runs out to a point. It is transparent, and has the appearance of being covered with a veil-like membrane ; hence the name of the bacillus.

The bacillus does not grow to any great extent on potato, and does not curdle milk. [The editor has not been able to find any further information as to this bacillus.]

17. Bacillus Butyricus (Prazmowski).—Synonyms, Clostridium butyricum ; Bacillus amylobacter. This organism was first recognized by Nothnagel in the feces. It is found abundantly in acid, fermenting stools. According to Nothnagel, it exists only to a small extent in feces that contain neither amylaceous material nor vegetable residue, but it occurs in very large numbers in stools that contain these ingredients. Boas succeeded repeatedly in finding the Bacillus butyricus in the gastric secretion after a period of fasting.

It forms bacilli that are from 3 to 10 μ long and 1 μ broad, and are often united to make up chains or long filaments. The organisms may be either motile or non-motile. At a certain stage in their development they increase in breadth without increasing in length. The short ones then become fusiform ; they surround themselves with a thick membrane ; and at the same time their plasma refracts light strongly. Under the microscope these fusiform bodies can be readily seen in the feces and identified as Bacillus butyricus, but when the bacillus is in the form of rods and filaments, it resembles the Bacillus subtilis so much that the differentiation can be made only by cultures. Spores are formed within the fusiform bodies and are liberated by the membrane of the parent cells becoming dissolved, after which they germinate. A characteristic reaction for Bacillus butyricus is the so-called granulose reaction. If a slide containing Bacillus butyricus is treated with iodin, certain granules and bands within the bacillus, or even the whole bacillus, are stained blue or violet. This reaction is particularly well marked when the organism was cultivated on a medium containing starch. The Bacillus butyricus is strictly anaërobic.[1]

The Bacillus butyricus can ferment starch, dextrin, sugar, and lactates, the products being butyric acid, carbonic acid, and hydrogen. The fermentation produced by it does not convert lactose directly into butyric acid ; this conversion takes place only indirectly, and when bacteria of the lactic acid fermentation are also present. Bacillus butyricus can also ferment cellulose. It is probable that from its power of setting up special fermentations the Bacillus butyricus plays an important rôle in certain physiologic processes, and possibly in the etiology of certain morbid conditions. So far, however, nothing definite is known in regard to its action on the animal body.

18. The Bacillus of Utpadel.—This bacillus was found by Gessner in the duodenal contents obtained from several dead bodies.

It forms thick rods that are from 1.25 to 1.5 μ long and from 0.75

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[1] According to Gruber, there are three species of Bacillus butyricus. Of these, two are strictly anaërobic, while the third is a facultative anaërobe. Gruber states that the last-named species fails to give the granulose reaction in any stage of its development ; also that it liquefies gelatin, whereas the other two do not possess this power.

to 1 μ broad. These rods exhibit sluggish motion. The bacillus grows very rapidly in gelatin without liquefying it. The superficial colonies on gelatin plates are milk white, and rise cone shaped above the level of the plate. The protuberances formed in this way may be 2 mm. high.

The bacillus of Utpadel is pathogenic for guinea-pigs, mice, and cats. This organism was first discovered by Utpadel in the padding of quilts.

In conclusion a few species of bacteria must be enumerated which are to all appearances often concerned in the pathogenesis of certain intestinal disorders.

19. Bacillus Enteritidis (Gärtner).—This bacillus was discovered by Gärtner in the flesh of a cow which had presented symptoms of enteritis. He also found it in the spleen of a man who died twenty-four hours after eating some of this meat. Lubarsch found the same bacillus in a case of septic pneumonia occurring in a new-born infant.

The Bacillus enteritidis forms short motile rods that frequently occur in pairs. It is often surrounded by an areola. One pole seems to stain with particular intensity ; the rest of the body stains very slightly. The bacillus grows on gelatin without liquefying the culture-medium. In stab culture the growth is very slight along the course of the stab. On the surface of the gelatin, however, a thick, grayish-white coating forms. The Bacillus enteritidis grows rapidly on agar-agar, potato, and blood-serum. It does not form spores.

Both when injected subcutaneously and when mixed with food, this bacillus is pathogenic for a number of animals, particularly for mice. It also generates toxic substances in media in which it is cultivated. Animals inoculated with this bacillus develop symptoms which are mainly those of enteritis and are similar to the symptoms exhibited by the sufferers from the epidemic at Frankenhausen. For this reason the organism has received the name of Bacillus enteritidis.

[The position of the Bacillus enteritidis in the intermediate class of the colon group has been referred to on page 48. Durham has investigated epidemics of poisoning due to the presence of this micro-organism in unsound meat.[1] Klein, from a study of their mutual serum reactions, came to the conclusion that Danysz's rat bacillus and Gärtner's Bacillus enteritidis were identical.—Ed.]

20. The Bacillus of Green Diarrhea (Lesage).—This bacillus was repeatedly observed by Lesage in the intestinal canal and in the evacuations of children suffering from green diarrhea. It forms rods that are from 2 to 4 μ long and 0.75 μ wide. Occasionally they may attain a length of 15 μ. They are motile and form spores when cultivated on gelatin. The bacillus grows on gelatin without liquefying the medium. It also grows on potato, bouillon, etc. On all culture-media it forms a green pigment, provided sufficient oxygen is admitted.

[1] *Trans. Path. Soc.*, vol. l., p. 262.

In stab cultures the development at the lower end of the stab is very scanty. It appears, therefore, that the organism is essentially aërobic. If the culture-medium is rendered unsuitable for its purpose (as by the addition of antiseptics, etc.), no formation of pigment occurs. Lactic acid, even in a low degree of concentration, possesses the power of destroying the bacillus.

Lesage succeeded in producing transient diarrhea in rabbits both by intravenous injection of the bacillus and by introduction of it into the stomach.

[Lesage's observations have not met with confirmation. Booker failed to find any chromogenic bacteria in 39 cases of green stools in infantile diarrhea, while Garrod, Kanthack, and Drysdale confirmed Booker's negative results and regarded biliverdin as the cause of the green color. According to Baumgarten, the bacillus described by Lesage is the Bacillus fluorescens non-liquefaciens, a common saprophyte.—ED.]

21. Bacillus Enteritidis Sporogenes (Klein).—Demonstrated by Klein in epidemic diarrhea, cholera nostras, summer diarrhea of children, and in milk and ichor. Hewlett found it frequently in the stools of healthy persons, in street-dust, milk, and water. It is very similar morphologically and biologically to Bacillus anthracis and Bacillus butyricus of Botkin. It is motile by means of flagella, is Gram-positive, and strictly anaërobic. It produces considerable gas-formation and precipitation of casein in milk. It liquefies blood-serum and is pathogenic for rabbits and guinea-pigs. In the latter, wide-spread gangrene is produced.

22. Spirillum of Finkler-Prior.—This was found by Kuisl in the feces of two healthy individuals and in the alkaline contents of the cecum and ascending colon of three suicides. With exclusively meat diet they increase rapidly ; with amylaceous diet they disappear.

[**Bacillus Dysentericus** (Shiga).—This micro-organism was first described by Shiga as the cause of dysentery in Japan ; subsequently it was found to be the cause of acute dysentery by Flexner and by Strong in the Philippines and Porto Rico, by Vedder and Duval in America, and by Kruse in Germany.

The bacillus is 1 to 3μ and has rounded ends, is flagellated, and sometimes has a capsule. Shiga, Flexner, and Strong described some motility, while Kruse, Vedder, and Duval were unable to detect it. The bacillus readily stains with ordinary anilin dyes, but not by Gram's method. Culturally, it forms colonies very similar to those of Bacterium coli and Bacillus typhi. It does not liquefy gelatin, gives an alkaline reaction to culture-media, does not ferment sugars ; it renders bouillon turbid and gives rise to a sediment, but not to a pellicle. It does not coagulate milk. The bacilli are agglutinated by the serum of patients suffering from the disease ; this has been shown by Flexner and Vedder and Duval to hold true between strains obtained from the various sources mentioned above. There is, therefore, reason to believe that the micro-organisms described in these different parts of the world are identical. The dysentery of asylums for which a bacillus was described

by Gemmel and Goodliffe in 1898 and Kruse in 1900 appears to be of the same nature as that described by Shiga.

In a recent critical review, Miss Sheldon Amos concludes that no organism has been finally shown to cause any one form of dysentery.—Ed.]

II. COCCI.

That micrococci occur in the feces has been clearly proved by Nothnagel's investigations. Bienstock's opinion that the feces contain only bacilli and never cocci has been refuted by all subsequent investigators. The number of cocci present in the feces is always very considerable, although, as a rule, it is less than the number of bacilli. In certain acute contagious enteritides of infants so great a preponderance of streptococci has been demonstrated in the stools that the possibility of their causative influence cannot be ignored off-hand (Tavel, Escherich).

The following are some of the best-known cocci:

1. Streptococcus Coli Gracilis (Escherich).—This coccus, according to Escherich, is constantly found in the intestinal canal when animal food has been taken, but it never occurs in infants that are breast-fed or live on milk exclusively.

The streptococcus forms chains consisting of from 6 to 12 or 20 individuals, each of which is from 0.2 to $0.4\,\mu$ in diameter. It liquefies gelatin rapidly; in gelatin stab cultures it causes a canalicular liquefaction of the medium. It coagulates milk after some time, with formation of acid. It is not pathogenic for animals.

2. Streptococcus Coli Brevis (Escherich).—This streptococcus, according to Escherich, is very frequently found in the stools of subjects living on a milk diet, less frequently in the meconium. Occasionally it is present in raw milk.

It forms chains consisting of from 3 to 8 individuals, each of which is from 0.2 to $0.4\,\mu$ in diameter. Distended forms are quite frequently seen. The streptococcus liquefies gelatin rapidly. When grown on plate cultures of gelatin, the colonies assume an olive-green color in direct light. On agar the streptococcus forms a thin, greenish-yellowish coating. On blood-serum it forms a luxuriantly growing lemon-yellow ridge. On young potatoes it forms extensive lemon-yellow colonies with a shiny surface.

3. Streptococcus Liquefaciens Ilei sive Acidi Lactici (Macfadyen, Nencki and Sieber).—This coccus was cultivated by the above-named observers from the case of fistula of the small intestine that has been repeatedly mentioned in the preceding pages.

It forms long chains and liquefies gelatin. It ferments dextrose and forms inactive lactic acid. It is pathogenic for guinea-pigs.

4. Streptococcus Pyogenes Duodenalis (Gessner).—This organism was found by Gessner six times in 18 dead bodies. It is a long streptococcus. It is pathogenic for mice, and if injected into these animals, gives rise to abscesses or general sepsis.

5. The White Liquefying Staphylococcus (Escherich).—This organism is from 0.8 to $1.2\,\mu$ in diameter, and appears as round,

sometimes oval, cocci. It liquefies gelatin slowly, and with the production of a ropy fluid. The growth of this organism is slight and in no way characteristic.

6. The Yellow Liquefying Staphylococcus (Escherich).—This is a small coccus, $0.2\,\mu$ in diameter. It forms sulphur-yellow colonies and liquefies gelatin very slowly, converting it into a ropy fluid.

7. Micrococcus Ovalis (Escherich).—This organism is frequently found in meconium and in the stools of milk-fed subjects. It appears as a diplococcus of from 0.2 to $0.3\,\mu$ in diameter. There is nothing characteristic about its growth.

8. The Porcelain Coccus (Escherich).—This is a staphylococcus, $0.3\,\mu$ in diameter. In gelatin stab cultures it shows scanty development along the tract of the stab canal, but moist, shiny colonies having the appearance of porcelain develop on the surface of the gelatin. On potato it forms moist white colonies.

9. Micrococcus Luteus.—Frequently found by Matzuschita. Is $1\,\mu$ in size, elliptic, and refracts light markedly. It forms on potato yellow droplets of 1 to 3 mm.; on liquid media, a thick yellow surface.

10. Micrococcus Aurantiacus.—Frequently found by Matzuschita. Oval cocci $1.5\,\mu$ in diameter, often arranged as diplococci and tetracocci. Forms an orange-yellow deposit on boiled egg-albumen.

11. Sarcina Aurantiaca et Lutea.—According to Boas, these occur frequently in the stools in dilatation of the stomach. Matzuschita cultivated them from a few specimens of feces. By triple fission and subsequent grouping together of the parts they form wool-sack bacteria, or sarcinæ.

Growth on gelatin is in yellowish to orange-yellow colonies; the latter liquefy gelatin. On agar and potato they form a thick, yellow to reddish-yellow covering.

III. THE BLASTOMYCETES.

Nothnagel was the first to call attention to the fact that yeast fungi are present in nearly every stool, although usually in very small numbers. He also discovered that these organisms are frequently present in astonishing numbers in the diarrhea of children. According to Boas, the stomach also contains small numbers of yeast fungi in health. Escherich, Macfadyen, Nencki, Sieber, and others have cultivated these organisms from the meconium, the contents of the intestine, and the feces. The following species have been obtained by cultivation :

1. The Torula of Pasteur.—This torula has been repeatedly found in meconium by Escherich. It is, on an average, from 2 to $2.5\,\mu$ in diameter. The outline of the torula is circular, occasionally elliptic. Sometimes germination forms are seen. Many individuals are frequently congregated in little heaps. The organism forms no mycelium. It grows on gelatin, but not on potato.

The torula, of which Pasteur and Hauser have described several species, is closely related to the yeast fungus. It is differentiated, how-

ever, from the latter by the fact that it forms neither ascospores nor mycelium.

2. Red Yeast.—This organism was first found by Escherich in meconium, and is often present in the air. When cultivated on artificial media, it forms colonies of a scarlet color. Red yeast does not grow on potato.

3. Encapsulated Yeast (Escherich).—This organism forms elliptic cells from 5 to 10 μ broad. Old specimens do not stain well ; they are encircled by a broad, light-colored areola which is a gelatinous capsule. The organism liquefies gelatin, but does not grow on potato.

4. Monilia Candida (Hansen).—This organism is probably identical with Saccharomyces albicans (thrush). It was cultivated by Escherich from a diarrheal stool and from raw milk.

Monilia candida is an oval or a round organism, from 5 to 10 μ long and from 4 to 7 μ wide. It forms mycelium with gonidia. It grows in gelatin stab cultures and forms white, rugose colonies on the surface of the gelatin. Laterally, from the tract of the stab, bushy masses of mycelium radiate into the medium. It grows also on potato. It possesses the power of fermenting sugar with the production of alcohol.

IV. MOULDS.

Of the filamentous fungi, Matzuschita has isolated Oïdium lactis, Mucor mucedo, and Penicillium glaucum, the last with greatest frequency.

LITERATURE.

Abel, Rudolf, " Ueber das Vorkommen feiner Spirillen in Dejectionen Cholerakranker," Centralbl. f. Bakt. u. Parasitenk., 1894, vol. xv., No. 7.

Adrian, " Ueber die Abhängigkeit der Ausscheidung aromatischer Körper im Harne, etc.," Arch. f. Verdauungskr., 1896, vol. i.

Albu, Bericht des 16. Cong. f. innere Med., 1898, p. 192.

Arnaud, O., " Recherches sur l'étiologie de la dysenterie aiguë des pays chauds," Annales de l'institut Pasteur, 1894, vol. viii., No. 7.

Ascher, " Studien zur Aetiologie der Ruhr und zur Darmflora," Deutsch. med. Wochenschr., 1899, No. 4.

Aufrecht, " Ueber den Befund feiner Spirillen, etc.," Centralbl. f. Bakt. u. Parasitenk., 1894, vol. xv., No. 12.

Austerlitz and Landsteiner, " Ueber die Bakteriendichtigkeit der Darmwand," Centralbl. f. Bakt., 1898, vol. xxiii.

Babes, V., " Ueber die durch Streptokokken bedingte gelbe Leberatrophie," Virchow's Arch., 1894, vol. cxxxvi.

Backmann, W., " Ein Beitrag zur Kenntniss der Darmfäulniss, etc.," Zeitschr. f. klin. Med., 1902, vol. xliv., parts v. and vi.

Baginsky, " Ueber Cholera infantum," Arch. f. Kinderheilk., 1890, vol. xii., 1, 2.

Bail, M., " Die Schleimhaut des Magen-Darmtractus als Eingangsporte pyogener Infection," Arbeiten aus der kgl. chir. Klinik, Berlin, 1901, vol. xv.

Bary, W. de, " Beitrag zur Kenntniss der niederen Organismen im Mageninhalte," Arch. f. exp. Path., 1886, vol. xx., p. 243.

Baumgarten, " Ueber die Uebertragbarkeit der Tuberculose durch die Nahrung," Centralbl. f. klin. Med., 1884, No. 2.

Beco, L., " Etude sur la pénétration des microbes intestinaux dans la circulation générale pendant la vie," Annales de l'institut Pasteur, 1895, vol. ix.

Beco, L., " Note sur la valeur de l'agglutination par le sérum antityphique, etc.," Centralbl. f. Bakt., 1899, vol. xxvi.

Bernheim, A., " Ueber den Befund des Bact. coli in einem Panaritium bei Typhus abd.," Centralbl. f. klin. Med., 1893, vol. xiv., p. 13.

Bienstock, B., " Ueber die Bakterien d. Fæces," Zeitschr. f. klin. Med., 1884, vol. viii.

Bienstock, B., "Untersuchungen über die Aetiologie der Eiweissfäulniss," Arch. f. Hygiene, 1899 and 1900, vols. xxxvi., xxxvii.

Billroth, T., Coccobacteria septica, p. 94.

Birch-Hirschfeld, A., "Ueber das Eindringen von Darmbakterien, etc.," Ziegler's Beiträge z. path. Anat., vol. xxiv., pt. ii.

Bizzozzero, G., "Sulla presenza costante di batteri nei folliculi linfatici, etc.," Archivio per le scienze med., vol. ix., No. 18.

Blachstein, A. G., "Intravenous Inoculation of Rabbits with Bacterium coli, etc.," Bulletin Johns Hopkins Hosp., Baltimore, 1891, vol. v., 2, No. 14; ref. in Centralbl. f. Bakt. u. Parasitenk., vol. xii., p. 278.

Boas, J., Diagnostik u. Therapie der Magenkrankh., pt. i., third ed., 1898.

Booker, William; "A Study of Some of the Bacteria Found in the Dejecta of Infants, etc.," Trans. Ninth Internat. Med. Cong., vol. iii.; ref. in Centralbl. f. Bakt. u. Parasitenk., 1889, vol. v., p. 316.

Breslau, "Ueber Entstehung und Bedeutung der Darmgase bei neugeborenen Kindern," Monatsschr. f. Geburtsk. u. Frauenkrankh., 1866, vol. xxviii.

Brieger, L., "Ueber Spaltungsproducte der Bakterien," Zeitschr. f. physiol. Chem., 1883–84, vol. viii.

Brieger, L., ibid., 1885, vol. ix.

Brudzinski, J., "Ueber das Auftreten von Proteus vulgaris in Säuglingsstühlen, etc.," Jahrb. f. Kinderheilk., 1900, vol. lii.

Brunner, Conrad, "Eine Beobachtung von Wundinfection durch das Bacterium coli commune," Centralbl. f. Bakt. u. Parasitenk., 1894, vol. xvi., No. 24.

Buchbinder, "Experimentelle Untersuchungen am lebenden Thier- und Menschendarm," Deutsch. Zeitschr. f. Chir., 1900, vol. lv.

Buchner, Hans, "Beiträge zur Kenntniss der Neapeler Cholerabacillen," Arch. f. Hyg., 1885, vol. iii.

Bunge, R., "Zur Kenntniss der geisseltragenden Bakterien," Fortschr. d. Med., 1894, vol. xii., No. 17.

Cadéac, C., and Bournay, J., "Rôle microbicide des sucs digestifs, etc.," La Prov. médicale, 1893, vol. viii., No. 28; ref. in Centralbl. f. Bakt. u. Parasitenk., 1894, 15, 16.

Cahn, "Ueber die nach Gram färbbaren Bacillen des Säuglingsstuhles," Centralbl. f. Bakt., 1901, vol. xxx., p. 721.

Capitan and Morau, "Recherches sur les microorganismes de l'estomac," Compt. rend. de la soc. de biol., 1889, p. 25.

Carbone, Tito, "Sui veleni prodotti del Proteo volgare," Rif. med., September, 1890; ref. in Baumgarten's Jahresber., 1890, vol. vi.

Casagrandi, O., "Ueber das Vorkommen von Blastomyceten in dem Darmcanale, etc.," Annali d'igiene sperimentale, vol. viii.

Ciechomski, A., and Jakowski, M., "Ungewöhnlich lange dauernder künstl. After, etc.," Arch. f. klin. Chir., 1894, vol. xxviii., pt. i.

Coco, A. M., "Il coli bacillo ed i cocchi piogeni nell' etiologia delle febbri intestinali," Gazzetta degli ospedali, 1898, No. 10; ref. in Centralbl. f. Bakt., 1899, vol. xxv.

Cotton, F. J., "Ein Beitrag zur Frage der Ausscheidung von Bakterien durch den Thierkörper," Sitzungsbericht der k. Akad. der Wiss. in Wien, vol. cv., pt. iii., Mathem.-naturw. Classe.

Coyon, A., Flore microbienne de l'estomac, Paris, 1900, Carré and Nauel.

Cushing, H., and Livingood, L. E., "Experimental and Surgical Notes upon the Bacteriology of the Upper Portion of the Alimentary Canal, etc.," Johns Hopkins Hospital Reports, vol. ix.

Demel, C., and Orlandi, "Die Serumtherapie und das Bacterium coli commune," Centralbl. f. Bakt. u. Parasitenk., 1894, No. 6.

Denys, J., and Brion, E., "Etude sur le principe toxique du Bacterium lactis aërogenes," La Cellule, 1892, vol. viii.; ref. in Centralbl. f. Bakt. u. Parasitenk., vol. xvi., No. 3.

Denys, J., and Brion, E., "Sur les rapports du Pneumobacille de Friedländer, etc.," ibid., 1893, vol. ix., ref. ibid.

Dmochowsky and Janowski, "Zwei Fälle von eiteriger Entzündung der Gallengänge, etc.," Centralbl. f. pathol. Anat. u. exp. Path., 1894, No. 4.

Dreyfus, R., "Ueber die Schwankungen in der Virulenz des Bacterium coli," Inaug. Diss., Strassburg, by Gebweiler, 1894, cited in Centralbl. f. Bakt. u. Parasitenk., 1894, No. 14.

Eisenhart, H., "Puerperale Infection, etc.," Arch. f. Gynäkol., 1894, vol. xlvii.

Eisenstadt, H. L., "Ueber die Möglichkeit die Darmfäulniss zu beeinflussen," Arch. f. Verdauungskrankh., 1898, vol. iii.

Emmerich, Rudolf, "Untersuchungen über die Pilze der Cholera asiatica," Arch. f. Hyg., 1885, vol. iii.

Escherich, T., Die Darmbakterien des Säuglings, Stuttgart, by Enke, 1886.

Escherich, T., " Die Darmbakterien des Neugeborenen und Säuglings," Fortschr. d. Med., 1885, vol. iii., Nos. 16, 17.

Escherich, T., " Beiträge zur Kenntniss der Darmbakterien," Münch. med. Wochenschr., 1886, No. 1.

Escherich, T., ibid., No. 43 (" Ueber Vibrio felinus").

Escherich, T., " Ueber das Vorkommen von Vibrionen im Darmkanal, etc.," ibid., No. 45.

Escherich, T., Aerztl. Intelligenzbl., 1884, No. 54.

Escherich, " Ueber specifische Krankheitserreger der Säuglingsdiarrhöen (Streptokok- kenenteritis)," Wien. klin. Wochenschr., 1897.

Escherich, " Die Bedeutung der Bakterien in der Aetiologie der Magen-Darmerkrank- ungen der Säuglinge," Deutsch. med. Wochenschr., 1898.

Escherich, " Pyocyaneusinfectionen bei Säuglingen," Centralbl. f. Bakt. u. Parasitenk., 1899, vol. xxv.

Escherich, " Epidemisch auftretende Brechdurchfälle in Säuglingsspitälern," Jahrb. f. Kinderheilk., 1900, vol. lii.

Escherich, " Die Aetiologie der primären Magen-Darmerkrankungen der Säuglinge bakte- riellen Ursprungs," Wien. klin. Wochenschr., 1900, No. 38.

Escherich, " Zur Kenntniss der Darmcolibacillen unter physiologischen und patholo- gischen Verhältnissen," Verhandlungen des XVII. Cong. f. innere Med.

Escherich, " Ueber Streptokokkenenteritis im Säuglingsalter," Jahrb. f. Kinderheilk., N. F., vol. xlix.

Fischer, A., " Zur Biologie des Bacillus fæcalis alkaligenes," Centralbl. f. Bakt. u. Para- sitenk., 1899, vol. xxv.

Fischer and Lewy, " Ueber die path. Anat. u. d. Bakteriologie der Lymphangitis d. Ex- tremitäten," Deutsch. Zeitschr. f. Chir., vol. xxxvi.

Frerichs, Wagner's Handwörterbuch der Physiologie, 1846, vol. iii., p. 869.

Gärtner, " Ueber die Fleischvergiftung in Frankenhausen, etc.," Correspondenzbl. d. allg. ärztl. Vereins von Thüringen, 1888, No. 9 ; ref. in Baumgarten's Jahresb., 1888, vol. iv.

Gessner, C., " Ueber die Bakterien im Duodenum des Menschen," Arch. f. Hyg., vol. ix.

Glücksmann, S., " Fleischvergiftung durch Bacterium proteus vulgaris," Centralbl. f. Bakt. u. Parasitenk., 1899, vol. xxv.

Grassberger, R., and Passini, F., " Ueber die Bedeutung der Jodreaction für die bakteri- ologische Diagnose," Wien. klin. Wochenschr., 1902, No. 1.

Gruber, " Eine Methode der Cultur anaërobischer Bakterien, etc.," Centralbl. f. Bakt. u. Parasitenk., 1887, vol. ii., No. 12.

Hellström, " Untersuchungen über Veränderungen in der Bakterienzahl der Fäces bei Neugeborenen," Arch. f. Gynäkologie, 1901, vol. lxiii.

Henke, F., " Beitrag zur Verbreitung des Bacterium coli communis in der Aussenwelt," Centralbl. f. Bakt. u. Parasitenk., 1894, vol. xvi., No. 12.

Hewlett, R. T., " Preliminary Observations on the Occurrence of the Bacillus enteritidis sporogenes, etc.," Transactions of the Jenner Institute, London, 1899 ; ref. in Centralbl. f. Bakt. u. Parasitenk., vol. xxx., p. 348.

Hodenpyl, A., " On the Etiology of Appendicitis," New York Med. Jour., 1893, vol. lviii., p. 777; ref. in Centralbl. f. Bakt. u. Parasitenk., 1894, No. 21.

Jaeger, H., " Die Aetiologie des infectiösen fieberhaften Icterus," Zeitschr. f. Hyg., 1892, vol. xii.

Jeanselme, " Contribution à l'étude des thyroidites infectieuses," Arch. gén. de méd., 1893; ref. in Centralbl. f. Bakt. u. Parasitenk., 1894, vol. xvi., No. 21.

Jeffries, John A., " Ein Beitrag zu dem Studium der Sommerdiarrhoen bei Kindern," Arch. Pediatrics, 1889–90; ref. in Arch. f. Kinderheilk., 1891, vol. xiii.

Kempner, W., " Ueber den vermeintlichen Antagonismus, etc." Centralbl. f. Bakt. u. Parasitenk., 1895, vol. xvii., No. 1.

Klecky, Ch. de, " Recherches sur la pathogénie de la péritonite," Annales de l'institut Pasteur, 1895.

Klein, A., "Bacteriologische onderzoekingen van menschelijke faeces," Kon. Akad. v. Wetensch., Amsterdam, 1901 ; ref. in Centralbl. f. Bakt. u. Parasitenk., 1901, vol. xxx., p. 308.

Klein, A., " Morphologie und Biologie des Bacterium enteritidis sporogenes, etc.," Twenty- seventh Annual Report of the Local Government Board, 1897–98, London; ref. in Centralbl. f. Bakt. u. Parasitenk., 1899, vol. xxv.

Klein, E., " Ueber die Verbreitung des anaëroben virulenten Bacillus enteritidis sporogenes," Centralbl. f. Bakt. u. Parasitenk., 1898, vol. xxiii.

Kohlbrugge, J. H. F., " Der Darm und seine Bakterien," ibid., 1901, vol. xxx.

Kohlbrugge, J. H. F., " Die Autosterilisation des Dünndarmes und die Bedeutung des Cöcum," ibid., 1901, vol. xxix.

Kreisel, A., "Studien über Colibacillen," ibid., 1901, vol. xxix.

Krogius, A., "Recherches bactériologiques sur l'infection urinaire," Helsingfors, 1892; ref. in Baumgarten's Jahresber., 1892, vol. viii.

Kuhn, F., "Morphologische Beiträge zur Leichenfäulniss," Arch. f. Hyg., vol. xiii.

Kuisl, " Beiträge zur Kenntniss der Bakterien im normalen Darmtractus," Inaug. Diss., Munich, 1885.

Lannelongue and Achard, "Sur les infections provoquées par les bacilles du groupe Proteus et sur les propriétés agglutinantes du sérum dans ces infections," C. r., 1896.

Lembke, W., " Beitrag zur Bakterienflora des Darms," Arch. f. Hygiene, 1896, vol. xxvi.

Lembke, W., " Weiterer Beitrag zur Bakterienflora des Darms," ibid., 1897, vol. xxix.

Lermoyez, " Un cas d'amygdalite coli-bacillaire," La semaine méd., 1894, No. 37.

Lesage, A., " Du bacille de la diarrhée verte des enfants du premier âge," Arch. de physiol., 1888, No. 1.

Lesage and Macaigne, " Choléra en 1892," Annales de l'institut Pasteur, 1893, vol. vii.

Levin, " Les microbes dans les régions arctiques," ibid., 1899, vol. xiii.

Lewkowicz, X., " Ueber den Enterococcus als Ruhrerreger," Centralbl. f. Bakt. u. Parasitenk., 1901, vol. xxix., p. 635.

Libman, E., "Streptococcus enteritidis," Med. Rec., 1898; ref. in Centralbl. f. Bakt. u. Parasitenk., 1899, vol. xxv.

Lommel, " Eine aus Darminhalt gezüchtete Hefeart," ibid., 1901, vol. xxix.

Lubarsch, O., " Ein Fall von sept. Pneumonie, etc.," Virchow's Arch., vol. cxxiii., p. 70.

Macfadyen, A., " The Behavior of Bacteria in the Digestive Tract," Jour. Anat. and Physiol., vol. xxi.

Macfadyen, Nencki and Sieber, " Untersuchungen über die chemischen Vorgänge im menschlichen Dünndarm," Arch. f. exp. Pathol., 1891, vol. xxviii.

Maggiora, A., "Einige mikrosk. u. bakt. Betrachtungen, etc.," Centralbl. f. Bakt. u. Parasitenk., 1892, vol. xi.

Marcus, H., " Zur Frage der Durchgängigkeit des Darms für Bakterien," Wien. klin. Wochenschr., 1901, No. 1.

Marpmann, " Zur Unterscheidung des Bacterium typhi abd. vom Bacterium coli commune," ibid., 1894, vol. xvi., No. 20.

Matzuschita, T., " Untersuchungen über die Mikroorganismen des menschlichen Kothes," Arch. f. Hygiene, 1902, vol. xli.

Messea, " Contribuzione allo studio delle ciglie, etc.," Rivista d'Igiene e sanitá pubblica anno I, No. 14; ref. in Baumgarten's Jahresb., 1891, vol. vii.

Metschnikoff, " Ueber Immunität gegen Cholera," Wien. med. Presse, 1894, vol. xxxv., No. 39.

Mieczkowski, von, " Desinfectionsversuche am menschlichen Dünndarme," Mittheilungen aus den Grenzgebieten der Med. u. Chir., 1902, vol. ix., p. 405.

Miller, W. D., Die Mikroorganismen der Mundhöhle, second ed., Leipsic, by Thieme, 1892.

Minkowski, D., " Ueber die Gährungen im Magen," Mittheilungen aus der med. Klinik zu Königsberg i. Pr., published by B. Naunyn, Leipsic, by Vogel, 1885.

Mironzescu, Th., " Ueber das Vorkommen von tuberkelbacillenähnlichen Bakterien in menschlichen Fäces," Zeitschr. f. Hygiene, 1901, vol. xxxvii.

Moro, E., " Ueber Staphylokokkenenteritis der Brustkinder," Jahrb. f. Kinderheilk., 1900, vol. lii.

Moro, E., " Ueber Bacillus acidophilus," loc. cit.

Müller, Fr., " Autointoxication intestinalen Ursprungs," Verhandl. des XVI. Cong. f. innere Med., Wiesbaden, 1898, p. 149.

Neisser, M., " Ueber die Durchgängigkeit der Darmwand für Bakterien," Zeitschr. f. Hygiene, 1896, vol. xxii.

Nencki, Ueber die Zersetzung der Gelatine und des Eiweisses, etc., Bern, by Dalp, 1876.

Nicolle and Morax, " Technique de la coloration des cils," Annales de l'institut Pasteur, 1893, vol. vii.

Nocard, " Société de la biologie," Progrès médical, Paris, 1895.

Nothnagel, H., " Die normal in den menschlichen Darmentleerungen vorkommenden niedersten (pflanzlichen) Organismen," Zeitschr. f. klin. Med., 1881, vol. iii.

Nuttall, H. F., and Thierfelder, H., " Thierisches Leben ohne Bakterien im Verdauungscanal," Zeitschr. f. phys. Chem., 1895–96, vol. xxi.

Opitz, E., " Beiträge zur Frage der Durchgängigkeit von Darm und Nieren für Bakterien," Zeitschr. f. Hygiene, 1898, vol. xxix.

Papasotiriu, J., " Untersuchungen über das Vorkommen von Bacterium coli in Teig, Mehl, und Getreide, etc.," Arch. f. Hygiene, 1902, vol. xli.

Passini, Fr., " Ueber granulosebildende Darmbakterien," Wien. klin. Wochenschr., 1902, No. 1.

Pasteur, C. r., 1885, vol. c., p. 68.

Peckham, A. W., " The Influence of Environment upon the Biological Processes of the Various Members of the Colon Group of Bacilli," Jour. Exper. Med., 1897.

Péré, " Bacterium coli commune et bacille typhique," Annales de l'institut Pasteur, 1892, vol. vi.

Pfaundler, M., " Eine neue Form der Serumreaction auf Coli- und Proteusbacillosen," Centralbl. f. Bakt. u. Parasitenk., 1898, vol. xxiii.

Pfaundler, M., " Ueber das Verhalten des Bacterium coli commune zu gewissen Stickstoffsubstanzen und zu Stärke," ibid., 1902, vol. xxxi.

Pfeiffer, R., " Ueber die specifische Immunitätsreaction der Typhusbacillen," Deutsch. med. Wochenschr., 1894, No. 48.

Pigeaud, J. J., " Ueber Bakterienbefunde (bes. Streptokokken) in den Dejectionen magendarmkranker Säuglinge," Jahrb. f. Kinderheilk., 1900, vol. lii.

Pisenti, G., and Bianchi-Mariotti, G. B., " Beziehungen zwischen dem Bacterium coli commune und der Typhusinfektion," Centralbl. f. Bakt. u. Parasitenk., 1894, No. 17.

Posner and Lewin, " Ueber Selbstinfection vom Darm aus," Berlin. klin. Wochenschr., 1895, No. 6.

Radziewsky, A., " Beitrag zur Kenntniss des Bacterium coli," Centralbl. f. Bakt. u. Parasitenk., 1899, vol. xxvi.

Refik, " Sur les divers types de coli-bacille des eaux," Annales de l'institut Pasteur, 1896, vol. x.

Riegner, " Vergleichende Untersuchungen über die Wirksamkeit einiger Magen- und Darmantiseptica," Deutsch. med. Wochenschr., 1898, No. 25.

Rodella, A., " Ueber die sogenannten säureliebenden Bacillen im Säuglingsstuhl," Centralbl. f. Bakt. u. Parasitenk., 1901, vol. xxix., p. 717.

Salus, " Ein Fall von Grünfärbung des Stuhles durch den Bacterium pyocyaneus," Prager med. Wochenschr., 1894, No. 33.

Sanarelli, " Etude sur la fièvre typhoide expérimentale," Annales de l'institut Pasteur, 1894, vol. viii., p. 353.

Scheffer, J. C., " Beiträge zur Frage der Differenzirung des Bacterium aërogenes u. Bacterium coli commune," Arch. f. Hygiene, 1894, vol. xxx., p. 291.

Schild, W., " Das Auftreten von Bakterien im Darminhalte Neugeborener, etc.," Zeitschr. f. Hyg. u. Infectionskrankh., 1895, vol. xix.

Schmidt, Alex., " Zur Kenntniss der Bakterien in den Säuglingsstühlen," Wien. klin. Wochenschr., 1892.

Schnitzler, J., Zur Aetiologie der Cystitis, Vienna, by Braumüller, 1892.

Schott, A., " Berechtigen experimentelle oder klinische Erfahrungen zu der Annahme das pathogene oder nicht pathogene Bakterien die Wand des gesunden Magendarmcanals durchwandern können?" Centralbl. f. Bakt. u. Parasitenk., 1901, vol. xxix., p. 239.

Schottelius, M., " Die Bedeutung der Darmbakterien für die Ernährung," Arch. f. Hygiene, 1899, vol. xxxiv.; Zeitschr. f. diätet. u. physik. Therapie, 1902, vol. vi., pt. iii.; Arch. f. Hygiene, 1902, vol. xlii., p. 48.

Schütz, R., " Bakteriologisch-experimenteller Beitrag zur Frage gastrointestinaler Desinfection," Berlin. klin. Wochenschr., 1900, No. 25.

Shiga, K., " Ueber den Erreger der Dysenterie in Japan," Centralbl. f. Bakt. u. Parasitenk., 1898, vol. xxiii.

Shiga, K., " Ueber den Dysenteriebacillus," ibid., 1898, vol. xxiv.

Singer, H., " Ueber Einfluss des Aspirin auf die Darmfäulniss," Zeitschr. f. klin. Med., 1902, vol. xliv.

Smith, H. L., " Zur Kenntniss der Colibacillen des Säuglingsstuhles," Centralbl. f. Bakt. u. Parasitenk., 1899, vol. xxix.

Sordoillet, Louis, " Peritonite sans perforation Bacterium coli commune," Paris, 1893; ref. in Centralbl. f. Bakt. u. Parasitenk., 1894.

Spiegelberg, H., " Ein weiterer Beitrag zur Staphylokokkenenteritis im Säuglingsalter," ibid., 1898, vol. xxiv.

Stahl, " Mikroorganismen in den Darmentleerungen," Verhandl. des III. Cong. f. innere Med., 1884.

Stern, R., " Ueber Desinfection des Darmcanales," Zeitschr. f. Hyg. u. Infectionskrankh., 1892, vol. xii.

Stern, R., "Typhusserum und Colibacillen," Centralbl. f. Bakt. u. Parasitenk., 1898, vol. xxiii.

Stern, R., "Ueber innere Desinfection," Leyden-Festschrift, vol. i., p. 581.

Stoecklin, "Recherches sur la mobilité et les cils, etc.," Annales suisses de sciences méd. ; ref. in Centralbl. f. Bakt. u. Parasitenk., 1894, No. 3.

Strasburger, J., "Weitere Untersuchungen über Fäcesgährung, etc.," Deutsch. Arch. f. klin. Med., vol. lxvii.

Strasburger, J., "Untersuchungen über die Bakterienmenge in menschlichen Fäces," Zeitschr. f. klin. Med., 1902, vol. xlvi., parts v. and vi.

Sucksdorff, W., "Das quantitative Vorkommen von Spaltpilzen im menschlichen Darmkanal," Arch. f. Hyg., 1886, vol. iv.

Talma, S., "Over de bactericide verking der gal.," Nederl. Tijdschr. o. Geneesk., 1900 ; ref. in Centralbl. f. Bakt. u. Parasitenk., 1901, vol. xxix., p. 366.

Terni and Pellegrini, "Bakteriologische Untersuchungen über die Choleraepidemie in Livorno, etc.," Zeitschr. f. Hyg., vol. xviii., p. 1.

Tissier, H., La flore intestinale, Paris, 1900, Carré et Naud.

Ury, J., "Ueber die Schwankungen des Bacterium coli communis in morphologischer u. cultureller Beziehung," Inaug. Diss., Strassburg i. E., 1894 ; ref. in Centralbl. f. Bakt. u. Parasitenk., 1894, No. 14.

Utpadel, "Ueber einen pathogenen Bacillus aus Zwischendeckenfüllung," Arch. f. Hyg., 1887, vol. vi.

Valagussa, F., "Experimentelle Untersuchungen über die Virulenz des Bacterium coli commune," Centralbl. f. Bakt. u. Parasitenk., 1898, vol. xxiv.

Van de Velde, H., "Valeur d'agglutination dans la sérodiagnose de Widal et dans l'identification des Bacilles éberthiformes," ibid., 1898, vol. xxiii.

Van de Velde, H., "Essai d'agglutination vis-à-vis de 25 variétés de colibacilles," Bull. de l'Acad. Royale de méd. de Belgique, March 27, 1897.

Van Puteren, "Ueber die Mikroorganismen im Magen von Säuglingen," Wratsch, 1888, No. 22 ; ref. in Baumgarten's Jahresb., 1888.

Weissenfeld, J., "Der Befund des Bacterium coli im Wasser, etc.," Zeitschr. f. Hygiene, 1900, vol. xxxv.

Wolf, S., "Beiträge zur Lehre der Agglutination, etc.," Centralbl. f. Bakt. u. Parasitenk., 1899, vol. xxv.

Wesener, F., "Kritische u. exp. Beiträge zur Lehre von der Fütterungstuberculose," Freiburger akad. Habilitationsschrift, 1886 ; cited in Baumgarten's Jahresb., 1885.

Woodward, The Medical and Surgical Report of the War of the Rebellion, 1879, vol. i., pt. ii.

SUPPLEMENTARY LITERATURE.

Adami, Abbott, and Nicholson, Trans. Assoc. Amer. Physicians, 1899.

Amos, Sheldon, Jour. Path. and Bact., vol. viii., p. 346.

Andrewes, F. W., Brit. Med. Jour., 1901, vol. ii., p. 1531.

Booker, Johns Hopkins Hosp. Rep., 1897, vol. vi., p. 159.

Cushing, Johns Hopkins Hosp. Bull., 1900, vol. xi.

Cushing and Livingood, Contributions to the Science of Medicine by the Pupils of W. H. Welch, 1900.

Durham, Lancet, 1898, vol. i., p. 154.

Flexner, Johns Hopkins Hosp. Bull., 1900, vol. xi., pp. 39, 231 ; Univ. Pennsylvania Med. Bull., August, 1901.

Ford, W. W., Montreal Med. Jour., November, 1900.

Garrod, Kanthack, and Drysdale, St. Bartholomew's Hosp. Rep., vol. xxxiii., p. 13.

Gemmel, Idiopathic Ulcerative Colitis, 1898.

Gilbert, La semaine méd., 1895.

Goldberg, Centralbl. f. innere Med., March 29, 1902.

Gordon, Jour. Path. and Bact., 1897, vol. iv., p. 438.

Gwyn, Johns Hopkins Hosp. Bull., 1890, vol. ix., p. 54.

Haslam, Trans. Path. Soc., 1898, vol. xlix., p. 345.

Herter, C. A., Lectures on Chemical Pathology, 1902.

Horton-Smith, Lancet, 1900, vol. i.

Hewlett, A. W., Amer. Jour. Med. Sci., vol. cxxiv., p. 200.

Hunter, W., Lancet, 1901, vol. i., pp. 613, 1079.

Johnston, Amer. Jour. Med. Sci., vol. cxxiv., p. 187.

Klein, Trans. Path. Soc., London, vol. liii., p. 342.

Kruse, Deutsch. med. Wochenschr., 1900, p. 637.

Pakes, Trans. Path. Soc., 1901, vol. lii., p. 167.

Satterlee, Med. News, June 6, 1903, p. 620.
Shiga, Centralbl. f. Bakt., 1898, vols. xxiii., xxiv.
Shiga, Deutsch. med. Wochenschr., 1901, vol. xxxvii.
Smith, J. Lorrain, and Tennant, Brit. Med. Jour., 1902, vol. ii., p. 1942.
Thierfelder and Nuttall, Zeitschr. f. physiol. Chem., vol. xxi., p. 109; vol. xxii., p. 62.
Vedder and Duval, Jour. Exper. Med., 1902, vol. vi., p. 181.
Widal and Nobecourt, La semaine méd., 1897, p. 286.

THE MOVEMENTS OF THE INTESTINE
(Motus Peristalticus).[1]

THE movements of the intestine which may be considered physiologic are represented essentially by two types :

1. Swaying (pendulum) movements ("waving," Raiser). In this form of movement a portion of intestine, usually a few centimeters long, contracts and relaxes alternately in a direction parallel to its longitudinal axis. No appreciable change in the lumen of the intestine occurs during these contractions. "Rhythmic segmentation" is the name applied by Cannon to a form of movement occasioned in the small intestine of cats by the administration of food to which subnitrate of bismuth had been added in quantity. This motion, which was seen by aid of the x-rays, exists independently of peristalsis. At regular intervals of space constrictions appear in the intestine, the segments thus formed are again divided, and so on, in cats to as many as thirty segmentations a minute. The limit of this motion is the ileocecal valve. They persist during sleep, but are suppressed completely by any great excitement.

2. Peristaltic movements proper. The mechanics of this form of movement have been carefully analyzed by S. Exner. The intestine during peristalsis undergoes alternate dilatation and contraction, with the production of a wave-like movement that runs along the intestine a certain distance toward the anus with moderate rapidity. These waves propel the intestinal contents in the direction of their own motion—namely, toward the anus. In the colon the haustra are alternately withdrawn and protruded during peristalsis.

The swaying movements and the functionally identical rhythmic segmentation serve to keep in constant agitation the fluid contents of the small intestine, but always within the same small portion of the gut, so that it is thoroughly mixed with the succus intestinalis and alternately comes in contact with the inner intestinal wall. The motor functions of these forms of intestinal movement chiefly advance the digestive process and the resorption of food. It is very likely too that they contribute in some degree to the onward movement of the intestinal contents. According to Mall, they also favor the evacuation of the venous blood into the vena portæ.

The peristaltic movement itself does not so much serve to advance the actual process of digestion, as to propel the intestinal contents toward the anus, preliminary to its ejection by the rectum. In peris-

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[1] The bibliographic references, excepting those added by editor, will be found in alphabetic order at the end of the volume.

talsis it is very likely that the contraction of the longitudinal muscles always precedes that of the circular muscles (Grützner).

3. Rolling movements. In this form of movement, which is, in fact, a transition-type between the physiologic and pathologic, the contents of a portion of the intestine, from 1 to 20 centimeters in extent, are propelled forward with the energy characterizing what has been called a peristaltic storm. The intestinal contents are actively propelled toward the ileocecal valve ; at the same time the intestine is tensely distended and performs rapid movements resembling the rotation of a wheel. Circular constriction always follows distention of a loop of the intestine. These movements suddenly cease without any appreciable external cause, so that a portion of the intestine that at one moment may be performing very violent movements suddenly becomes quiescent only to resume its energetic rolling movements after a varying interval. I believe that this abrupt cessation of intestinal rolling is due to some sudden nervous inhibition. This form of peristalsis, as has been already said, constitutes a transitional stage, and in some respects is akin to pathologic movements of the intestine. I have frequently seen this rolling movement in experiments on animals when the intestine was considerably distended with semiliquid contents mingled with gas-bubbles. The peculiar peristaltic movements which occur in the human small intestine (*tormina intestinorum*, Kussmaul), and which everybody is personally familiar with, are analogous to this phenomenon. These peristaltic storms are painless, are accompanied by loud gurgling sounds, and often stop suddenly.

These types of movements all occur chiefly in the small intestine. The movements of this portion of the bowel are incomparably more active than those of the colon. The contents of the large intestine require from twenty to twenty-four hours to pass from the ileocecal valve to the rectum, whereas the contents of the small intestine require only from two to six hours to accomplish the longer journey from the pylorus to the ileocecal valve. In order to guard against a very common misconception it must be distinctly stated that under physiologic conditions the same peristaltic wave never runs the whole length of the small intestine from the duodenum to the ileocecal valve. Even the most violent rolling movements stop after they have traversed a portion of the intestine, the length of which may vary, but is always limited.

The most active movements, relatively speaking, are seen under physiologic conditions in the duodenum and the first portion of the jejunum. These portions of the intestine are stimulated to increased movements not only by the presence of food coming from the stomach, but also, as I have proved experimentally, by the influx of bile and pancreatic juice. In all the other portions of the intestine the movements appear slightly different. Here and there pendulum or truly peristaltic contractions are seen which occasionally stop without appreciable external cause, and then, after an interval, reappear. These movements always occur in one portion of the intestine alone. As soon as they stop in one place they begin in another. The cecum, colon,

and rectum, on the other hand, frequently remain entirely quiescent for periods of even several hours.

Cannon describes as the most frequent movements of the large intestine antiperistaltic waves. These waves run along the large intestine as far as the ileocecal valve. He was unable to perceive any antiperistaltic movements in the small intestine. In the large gut these movements recur at intervals of fifteen minutes, last five minutes, and have a frequency of about five a minute. Thus there would be another mixing of intestinal contents in the large intestine, the powerful contractions of the cecum and colon forcing the mass gradually toward the end of the intestine.

Earlier investigators have not been able to observe the physiologic antiperistaltic movements of the large intestine as described by Cannon. Personally, I can but repeat that I have never been able to detect them in the colon of rabbits. In these I have seen the colon perfectly quiet for hours at a time, though meanwhile active peristaltic and swaying movements were going on in the small intestine.

Under physiologic conditions all the different movements of the intestine hitherto enumerated are caused solely by the irritating effects of its contents, whether chyme, feces, or gas. This is the only point on which I disagree with Braam-Houkgeest in regard to these questions. The sudden cessation of contractions in certain areas of the distended intestine is no argument against this view, and this objection is not valid. I have never seen any kind of contraction in any portion of the intestine that was absolutely empty. When empty, the intestine is always quiescent. On the other hand, the phenomenon of rolling movements of the intestine shows how excessive distention of the wall of the intestine by a large collection of gas, for instance, may stimulate the intestine to motor activity. These movements of the intestine, as has been already stated, are to some extent pathologic.

Opinions still differ concerning the causation of physiologic movements of the intestine.

Engelmann believed that peristalsis of the intestine may be due to a stimulus transmitted from muscle-cell to muscle-cell without the intervention of any nervous influence. He failed, however, to adduce any direct or positive experimental proof for this proposition, and, as a matter of fact, many objections can be formulated against this view. I feel justified in drawing an entirely different conclusion from the results of my experiments—namely, that certain nervous influences are always concerned in normal peristalsis. Gad, relying on his own experiments, Lüderitz and others favor the same view. The anatomic starting-point of these nervous stimuli must be sought in the plexuses of Auerbach and of Billroth and Meissner.

Bayliss and Starling believe that the swaying movements are of direct myogenic origin. They explain peristalsis as true coördinated reflexes, incited by irritation of the intestine and transmitted by Auerbach's plexus.

As is well known, the cerebrospinal nervous system exerts an in-

fluence on the movements of the intestine, and this question will be frequently referred to when the clinical aspects of the intestinal movements are under consideration. At present, however, the following brief summary may suffice. Experimental investigations into the influence of the mesenteric nerves on the movements of the intestine have given ambiguous and uncertain results. There is, however, no doubt that stimuli are carried to the intestine through the fibers of the vagus nerve, irritation of which either directly causes movements throughout the whole small intestine or increases peristalsis. The same applies to the movements of the upper half of the large intestine. Pflüger has shown that movements in these portions of the intestine are inhibited by irritation of the splanchnic nerves. A large number of investigations performed after Pflüger show that the inhibitory stimuli that reach the intestine are direct, but that, in addition, certain vasomotor fibers that supply the blood-vessels of the small intestine also run in the splanchnic nerves; for irritation of the splanchnics causes anemia of the intestine, whereas division of the splanchnic nerves causes hyperemia.

The innervation of the intestine, however, is much more complicated, Ludwig and Kupffer, Nasse, S. Mayer, and Basch, in addition to corroborating the presence of the inhibitory and vasomotor fibers above described, found other fibers in the splanchnic nerves that stimulated the movements of the intestine directly. Mayer and Basch discovered that, conversely, irritation of the vagus may inhibit intestinal peristalsis. Basch and Ehrmann, relying on their own experiments, formulated the view that the splanchnic nerve is the motor nerve of the longitudinal muscle-fibers of the intestine, and at the same time the inhibitory nerve of the circular fibers, while conversely the vagus stimulates the circular fibers and inhibits the contractions of the longitudinal fibers. Of the later investigators, Bayliss and Starling dispute any motor functions of the splanchnic nerve. They consider its inhibitory action as specific and independent of its vasoconstrictor action. Pahl, on the other hand, regards the splanchnic as motor and vasomotor; the inhibition of intestinal movements caused by its stimulation is occasioned by a simultaneous contraction of a muscle-layer (either the circular or longitudinal layer) over a portion of the intestine, while the contraction of the vessels of the intestinal wall suppresses the swaying movement and causes relaxation. [Langley and Anderson,[1] working on the innervation of the colon, rectum, and anus, found that this theory was certainly not of universal application, as shown by their observations on the descending colon of rabbits. Starling[2] and Bayliss's[3] observations lent no support at all to Basch's theory of crossed innervation in either of its versions.—Ed.]

The inhibitory and stimulating fibers for the rectum and lower half of the colon are contained in those nerves which originate from the

[1] Langley and Anderson, *Jour. Physiol.*, Cambridge and London, vol. xviii., p. 104.
[2] Starling, *Text-book of Physiology*, edited by Schäfer, 1900, vol. ii., p. 326.
[3] Starling and Bayliss, *Jour. Physiol.*, Cambridge and London, vol. xxiv.

spinal cord, pass through the great gangliated cord, are collected in the inferior mesenteric plexus and hypogastric plexus, to form, respectively, the nervus erigens and the nervus hypogastricus as they approach the intestines. We cannot here detail all the results obtained by various investigators, prominent among whom are Langley and Anderson. We shall, therefore, confine ourselves to brief mention of points bearing on the innervation of the lower rectum and the anus, as they are of the greatest importance in pathology.

The end of the rectum is closed by involuntary tonic contraction, in which both the smooth sphincter internus and the striated externus are engaged. There is innervation for strengthening and for relaxing the part. In the dog, the nervus erigens serves the former purpose, the nervus hypogastricus, the latter (v. Frankl-Hochwart and Fröhlich). The center for constriction is situated in the spinal column (Goltz), but there are other centers in the inferior mesenteric ganglion (Langley, Anderson, *et al.*) and in the rectum itself (v. Frankl-Hochwart and Fröhlich). From the spinal column and inferior mesenteric ganglion reflex dilatation can be excited; from the cortex cerebri, both constriction (Bechterew) and dilatation (Frankl-Fröhlich).

The movements of the intestine are influenced by a large number of factors, a knowledge of which is necessary in order to understand properly the pathologic conditions affecting the intestine. A brief summary of these factors only will be given here; further details will be given subsequently.

It has been mentioned that the contents of the intestine are the primary cause of all intestinal movements. They may either irritate the nervous and muscular structures of the intestine directly by mechanical distention, or they may irritate the sensory nerves of the mucosa and in this way cause intestinal movements reflexly. Substances that are chemically indifferent can act only when their temperature varies greatly from the normal body-temperature (ice water, for instance), or when they are present in large bulk (either solid, fluid, or gaseous), so as to cause considerable distention of the intestine. Rapid distention is a powerful cause of peristalsis; it must be remembered, however, that distention can cause movements of the intestine only up to a certain limit; for as soon as overdistention occurs, intestinal contraction becomes impossible. It would be impossible at present to enumerate all the substances that are chemic irritants to the intestine. Those that are pathologically important will be described in the sections treating on diarrhea and catarrh of the intestine. Of gaseous bodies, hydrogen, oxygen, and nitrogen are chemically indifferent, but carbon dioxid, sulphureted hydrogen, and hydrocarbons stimulate peristalsis energetically. Anemia of the intestine decreases its motility; hyperemia, either venous or arterial, or the accumulation of carbon dioxid in the blood, increases it.

In the normal human subject, under physiologic conditions, the movements of the intestine are very seldom visible through the abdominal walls. Under pathologic conditions, on the other hand, the move-

ments of the intestine may be enormously increased in intensity, so that peristaltic movements are frequently seen through the abdominal walls and constitute an important and a common symptom of certain diseases. When discussing stenosis of the intestine, these visible movements will be described in detail. Normal peristaltic movements are seldom visible through the abdominal wall, because the changes in shape that the intestine undergoes are too slight and because the abdominal walls in healthy subjects are usually too thick. It is only when the abdominal walls are exceptionally thin and the muscles are very much relaxed (particularly when there is divarication of the recti muscles) that these movements can be seen. If under these circumstances observations are made during the period of intestinal digestion or during the rolling movements described above, when the intestine is in vigorous action, normal peristalsis may be perceptible to the eye. Plates Nos. 1, 2, and 3 show the appearance of normal peristaltic movements better than could be done by any description. They may be characterized as slow, successive, rising and falling movements of small elevation. The abdomen at any given moment presents the appearance of a sac filled with potatoes. Visible peristaltic movements are always due to movements of the small intestine, for the physiologic movements of the large intestine can never be seen through the abdominal walls.

There are three forms of pathologic movements of the intestine: first, increased peristalsis; second, tonic contractions; third, antiperistalsis.

Increased peristalsis has already been referred to in the paragraph on the rolling movements of the intestine. I am inclined to regard these rolling movements of the intestine as a transition from normal to abnormal or morbidly increased peristalsis. There is, however, another form of increased peristalsis particularly coinciding with the tetanic contractions of the intestine which will be described below. Whenever the mucosa of the intestine is irritated by some chemic agent and thereby inflamed, very violent contractile movements are seen to occur in the inflamed area, even though this portion of the intestine is perfectly empty. These contractions cause an incessant commotion, and alternately or simultaneously exhibit pendulum movements or circular constrictions. The chief criterion of abnormal peristalsis, however, is the increased rapidity with which the intestinal contents are propelled onward.

Increased peristalsis may occur under the following conditions: (1) When the contents of the intestine are abnormal, as from acidity or copious development of gas; (2) after the administration of purgatives; (3) as an effect of intestinal irritants, either thermic (including the application of cold) or chemic; (4) after violent emotional shocks; (5) in neurasthenia; (6) in acute inflammatory states of the intestine; (7) in stricture of the intestine.

Tetanic contractions are a very important form of pathologic movements of the intestine. Two definite clinical pictures can be distinguished; one is presented when the intestine is empty; the other when

it is not.   The first form is unquestionably very common, but is seen only in experiments on animals after the contents of the abdominal cavity have been exposed.   The second form is comparatively rare, but can occasionally be perceived through the abdominal walls.   If an empty piece of intestine undergoes tonic contraction, it is converted into a solid cord of a pale color.   If these tonic contractions involve a large extent of the intestine, or if they involve the majority or all the intestinal loops, scaphoid retraction of the abdomen occurs.   This condition is occasionally seen in cerebral meningitis and in lead colic.   The other form—that is, tonic contraction of a loop of intestine filled with bowel contents—is very often seen in stenosis of the intestine.   When the contraction occurs, the loop of intestine, which is usually distended with gas or may contain liquid material, is suddenly changed from a soft flaccid into a very resisting, almost rigid, tube.   The spasm, after persisting for a short time, gradually relaxes, and the intestine soon returns to its original elastic state.

One feature of these movements seems to me to be clinically important—namely, the fact that simple progressive peristaltic movements, however active they may be, never cause painful sensations, whereas the tonic contractions are usually accompanied by very violent pain that may even become colicky in character.   This point will be referred to in the section on Intestinal Pain.   This view is not confuted by the argument that violent pain is frequently experienced in simple catarrh of the intestine with violent peristalsis, for this pain is not caused by the peristaltic movements of the intestine, but by the tonic contractions that frequently occur together with these peristaltic movements, and, in addition, by the inflamed condition of the mucous membrane and the abnormal irritability of the intestinal nerves that results therefrom.

The different causes of increased peristalsis and tonic contraction of the intestine will be discussed in detail in the sections devoted to the different diseases of the intestine.   It will, therefore, be unnecessary to describe them here.

Antiperistalsis—that is, true peristaltic movements which proceed in a direction opposite to the normal direction, and propel the contents of the intestine toward the pylorus instead of toward the anus—are occasionally seen even in cases in which the lumen of the intestine is free and patent.   This form of movement, however, occurs only under certain abnormal conditions, and never physiologically, as I have demonstrated by numerous experiments, modified in many ways.   For the details of these experiments I must refer to my special monograph on this question.[1]   The abnormal conditions that I refer to are the introduction of powerful chemic irritants into the intestine—as, for instance, strong solutions of sodium chlorid, sulphate of copper, etc. These chemic irritants alone, however, can never produce antiperistaltic movements in addition to the normal peristaltic movements unless they are introduced into some "unphysiologic" portion of the intestine.   In human subjects the only unphysiologic portion of the intestine is the

[1] *Loc. cit.*, p. 15.

PLATE I.
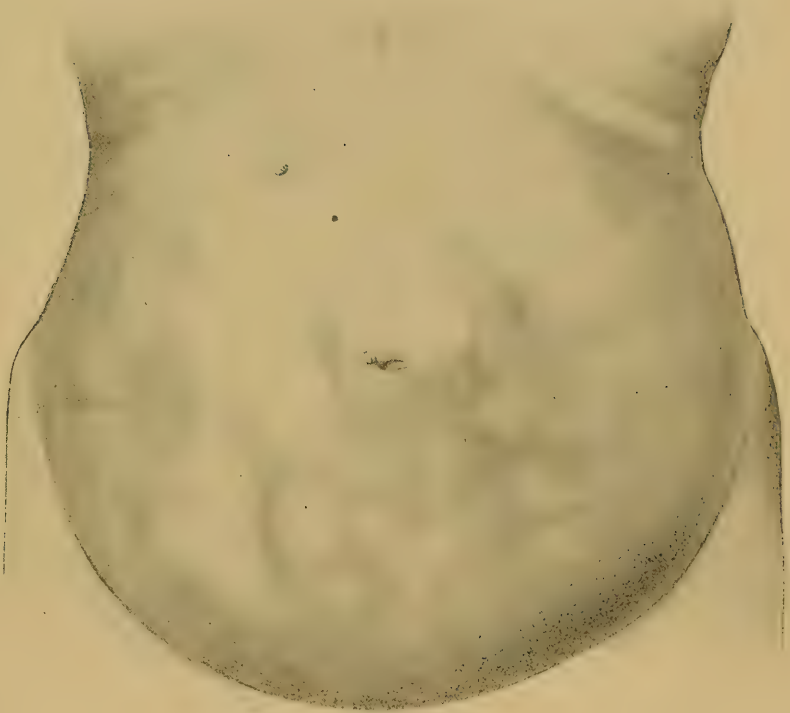
NORMAL PERISTALSIS.

PLATE II.

NORMAL INTESTINAL PERISTALSIS AND SIMULTANEOUS PERISTALSIS IN THE STOMACH, WHICH IS DILATED BY STENOSIS OF THE PYLORUS AND GREATLY DISPLACED.

PLATE III.
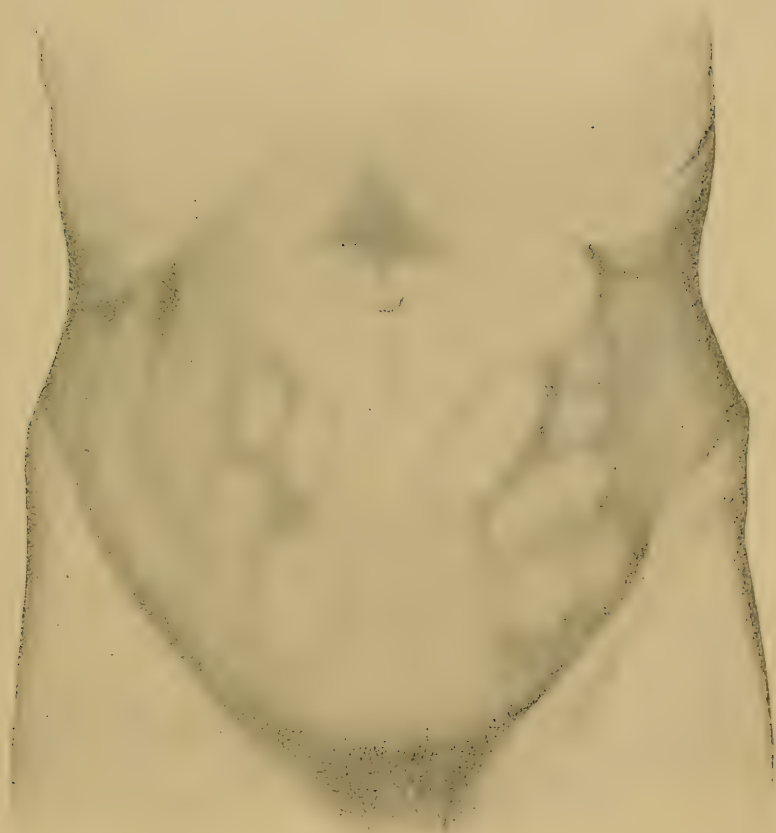
NORMAL PERISTALSIS.

rectum, into which irritant enemas may be injected, but, of course, in experiments on animals any portion of the intestinal tract may be made use of. Even the most powerful chemic irritants, when propelled from the stomach into the intestine, never produce antiperistaltic movements, but only peristaltic movements proceeding in the direction of the anus. (Compare Cannon's latest statement, contradicting the foregoing, concerning a physiologic antiperistaltic movement of the large intestine.) It is impossible to offer an explanation for this very peculiar phenomenon, for at present even the physiologic conditions under which normal peristalsis occurs are not fully understood. A remarkable discovery that I have made in regard to ascending contractions of the intestine following contact of the intestine with sodium salts [1] may perhaps assist in the interpretation.

Grützner has recently conducted a series of experiments on mammals, including man. He proved that fluids containing free particles (starch granules, lycopodium seeds, powdered carbon, etc.) introduced into the rectum, will, under favorable circumstances, find their way high up into the intestine, even at times into the stomach, within a few hours. This upward movement of injecta occurs best, perhaps only, with physiologic sodium chlorid solutions, and not at all with potassium chlorid or hydrochloric acid solutions. The facts concerning this "opposite movement" have been investigated by several observers, and may, therefore, be accepted as final. Comparative quiet of the intestine, as emphasized by Grützner himself, favors the movement. Particularly must there be no motion that has a tendency to force the intestinal contents downward with great force. Grützner originally explained the importance of salt solution by reference to my observations on the action of salt on the intestine. These proved that bringing sodium chlorid in contact with the outer intestinal wall caused contractions extending over several centimeters, invariably in an upward direction—*i. e.*, toward the stomach. At present he believes that it favors the diffusion of the contents over the greatest possible space. The correctness of the latter will not be discussed here.

To my knowledge there has never been demonstrated in any of Grützner's experiments a visible antiperistaltic movement. Hemmeter, in fact, emphasizes the statement that the upward movement is never visible to the unaided eye. By means of the *x*-rays Hemmeter observed that the upward movement of the particles above mentioned goes on simultaneously with the central downward progress of the feces. The former is always eccentric (Grützner's "Randströme"—marginal currents), and Hemmeter believes that the epithelium (and muscularis mucosæ) is instrumental in its progress. At all events, the term antiperistalsis does not fit these movements, and to the antiperistalsis of pathologic processes it cannot be applied at all, particularly as the very existence of the movements presupposes a state of rest of the intestines.

Occasionally antiperistaltic movements are seen in the dying intestine after the death of the animal, but these movements occur under condi-

[1] *Loc. cit.*, p. 51.

tions that are not encountered in clinical pathology. The question of fecal vomiting and its relation to antiperistalsis will be discussed in the section on Stricture of the Intestine, where another form of pathologic movement of the intestine to which I have given the name of recoil contraction (" Rückstosscontraction ") will also be described.

[W. B. Cannon [1] has investigated the intestinal movements by means of the Röntgen rays. The method was practically identical with that employed in 1897 to investigate the movements of the stomach. The animals employed were cats, whose food was mixed with from 10 to 33 per cent. of subnitrate of bismuth, so that the passage of food could be watched on the fluorescent screen. Cannon found that the usual movement of the transverse and ascending colon and cecum is an antiperistalsis. This recurs, as above noted, periodically, about every fifteen minutes, and lasts commonly about five minutes. This antiperistalsis gives new significance to the ileocecal valve ; for the food, now in a closed sac, is thoroughly churned and mixed by contractions running toward the cecum, and is again brought in contact with the absorbing surfaces of the colon without in any way interfering with the processes in the small intestine. As soon as food enters the cecum a vigorous general contraction takes place in the cecum and ascending colon, forcing some of the food onward ; a moment later antiperistaltic waves begin. There is no evidence that antiperistalsis occurs in the small intestine. But the ileocecal valve will allow a considerable amount of nutrient material under pressure to pass into the small intestine, hence the antiperistaltic movements of the colon may force into the small intestine a considerable portion of a large nutrient enema, which the movements of the small intestine deal with in the same way as they do with food which has passed normally from the stomach. Emotion, fear, distress, or rage are accompanied by a total cessation of the movements of both the large and the small intestine.

A remarkable case is recorded by Parkes Weber [2] in which enemas of castor oil and methylene-blue were on several occasions vomited some ten minutes after they had been introduced into the rectum. The patient was a markedly hysteric German woman aged twenty-two years, but fraud seems improbable, as a nurse was in close attendance between the introduction of the enema and its being vomited. A gastrocolic fistula was suspected, and laparotomy was performed on three occasions, but no sign of it was found. Strauss [3] met with a similar case in a man aged twenty-nine years, and Jaccoud, who called the condition " buccal defecation," observed a case in which, after death from typhoid fever, the ileocecal valve was normal. Weber suggests that reversed peristalsis in hysteria may depend on spasmodic stricture of the lower end of the bowel, which acts in the same way as complete obstruction of the bowel.—ED.]

[1] W. B. Cannon, *Amer. Jour. Physiol.*, vol. vi., p. 253.
[2] F. Parkes Weber, *St. Bartholomew's Hosp. Reps.*, vol. xxix., p. 34, and vol. xxxiv., p. 314.
[3] Strauss, *Berlin. klin. Wochenschr.*, 1898, p. 838.

INTESTINAL EVACUATIONS (Feces).

EXAMINATION of the intestinal dejecta is lamentably neglected; this is chiefly due to the peculiarly unattractive character of the work and the disagreeable odor of the material to be investigated. The examination of the feces, however, is much more important in the pathology and the diagnosis of diseases of the intestine than the examination of the sputa in the pathology and diagnosis of diseases of the respiratory tract. In very many cases an examination of the feces is the only basis available for the formation of an opinion as to the nature of intestinal affections, and for this reason it cannot be too strongly urged that very careful examination of the dejecta, both with the naked eye and with the microscope, should be a regular routine procedure in the investigation of cases of intestinal disease. The chemic analysis of the feces is important in physiology and also deserves an assured position in pathology, particularly in solving certain concrete and more or less scientific questions. For the purposes of ordinary medical practice chemic analysis of the feces is of comparatively little consequence, but there are, nevertheless, a few simple tests which must often be employed.

The scope of examinations of feces by the " meat test " has recently been extended by A. Schmidt, and that of the " fermentation test " by A. Schmidt and Strasburger conjointly. It is not yet certain that the complexity of the latter test will admit of its general use. The meat test is equally as simple of execution as it is valuble for testing in various disturbances of the digestive functions. We shall refer to it again later.

In this section no more will be attempted than a general summary and a brief review of this question. Chemic and bacteriologic methods of examination and the presence of animal parasites will not be included, because the chemistry and the bacteriology of the intestine have been discussed elsewhere by Obermayer and Mannaberg (pp. 17, 35). In discussing the different pathologic states that are encountered some of these subjects will, however, be from time to time referred to.

[A glass tube 7 inches long has been recommended by P. Cohnheim[1] as a means of obtaining fecal matter for examination from the rectum of patients. By employing it, infusoria are obtained alive and can be examined at once. When fecal material is brought by patients, infusoria are nearly always dead.—ED.]

Normal human feces essentially consist of the following : (1) Changed or unchanged remnants of the ingesta ; (2) bacteria ; (3) portions of the digestive secretions ; (4) a small amount of material derived from the intestinal wall, partly with morphologic characteristics and partly without ; (5) salts that are the product of physiologic processes of digestion. Schmidt and Strasburger rightly lay stress upon the fact that we may properly speak of typical meat, milk, or fasting feces, but not of *normal*

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[1] P. Cohnheim, *Deutsch. med. Wochenschr.*, May 15, 1902.

feces, because the constitution of feces varies with the kind of nourishment taken. (For an extended discussion of feces I refer to my work on the subject,[1] the work by Lynch, and particularly to the painstaking work of Schmidt and Strasburger.)

The quantity of feces naturally varies with the amount and the character of the food. On an average, from 120 to 250 grams of feces are evacuated daily by a person on an ordinary mixed diet. It is a remarkable fact that even in inanition and in long fasting dejecta are passed that consist chiefly of bacteria, disintegrated intestinal epithelia, mucus, and remains of digestive fluids (compare the section on Constipation). Other things being equal, the child excretes relatively far more feces than the adult or the aged.

The color of the normal dejecta is dark brown, from the presence of altered bile-pigment. Bilirubin is converted into urobilin (hydrobilirubin) by certain processes that occur in the intestine. Normal feces never give Gmelin's reaction. Frerichs tested the intestinal contents of dead bodies from the rectum upward, and found that in subjects who had not suffered from any disease of the intestine, either anatomic or functional, the ordinary Gmelin reaction began to give positive results only when the lowest portion of the ileum was reached, and that it was very rarely positive as low down as the cecum or the ascending colon. From these investigations the important conclusion follows that a positive Gmelin's reaction for bile-pigment in the feces is always pathologic, and indicates that intestinal peristalsis is for some reason increased, and that the contents of at least the lowest portion of the ileum have been propelled into the colon more rapidly than normal. It is possible to estimate approximately how high up increased peristalsis begins and how violent it is from the intensity of the typical green coloration given by Gmelin's test. The deeper the color, the higher up and the more active the abnormal peristalsis. Julius Rosenthal, Frerichs, and I have made these observations. Further details will be given in the section on Localization of Catarrh of the Intestine. The absence of bile-pigment from the feces (acholic stools) will receive more detailed consideration below.

The color of the stools is largely dependent on the diet. With preponderance of meat diet it is darker, from the brown hematin (Fleischer); with vegetable diet it is lighter, on account of numerous formed gas-bubbles, which give it greater translucency (Quincke); with milk and fat diets it is still lighter—nearer to light yellow. Foods of decided colors influence the color of stools, as, for example, green vegetables, whortleberries, and blackberries, also red wine and cocoa. Certain drugs, such as calomel, iron, santonin, rhubarb, etc., or exposure to the air, also change the color of the feces. Some time after evacuation the stools turn darker than they were originally, whereas the interior of the fecal mass remains light colored. Further, the feces turn darker the longer they remain in the large intestine, and in cases of constipation the stools may be dark brown, or even black.

[1] Nothnagel, *Contributions to the Pathology and Physiology of the Intestines.*

[In 3 cases where the stools were red and resembled blood Carter and McMunn [1] found that blood, bilirubin, and biliverdin were absent, and that a pigment allied to stercobilin was present. T. J. Walker [2] has recorded cases to show that the normal color of the feces depends on the entrance of pancreatic juice into the duodenum.—ED.]

The consistence of the stools is usually soft, but formed, as is well known. In general, if the feces pass through the colon rapidly, they are soft or liquid; if they remain there for a long time, they become dry and hard, and occasionally of almost stony hardness, owing to the absorption of water by the intestinal wall. In the latter case the stools rarely retain their typical elongated, sausage-like form, but appear as large or small rounded masses (scybala), the spheric contour of which is probably caused by their prolonged retention in the haustra of the colon. These small masses are about the size of a hazelnut or a walnut, and are occasionally marked with one or two furrows or indentations, which are undoubtedly impressions of the Tænia coli. Still smaller rounded masses, resembling sheep-dung, also with furrows and indentations, are occasionally seen in simple constipation, and the stools may continue to present their peculiar conformation for a long time. This fact should be specially emphasized, as there is a tendency to attribute great importance to this form of dejecta in the diagnosis of stricture of the colon. The same thing applies to the cases in which the feces, when passed, assume a long, thin, pencil-shaped or flattened form. These, too, have been supposed to indicate circumscribed stenosis of the intestine, but this is by no means the case, for they may occur without the presence of such a lesion. Their appearance, however, always indicates abnormal conditions of some kind, and they are particularly frequent in nervous, spastic contractions of the colon and after fasting.

The normal feces, as already mentioned, are more or less semi-solid and formed. If the dejecta are passed unformed, pultaceous, or liquid, some abnormal condition must be present. Occasionally the stools may be partially or entirely formed, and still be softer and more pultaceous than normal. When this is the case, certain pathologic changes have either actually occurred or conditions are present which are just within normal physiologic limits. The following factors, for instance, that are still physiologic, may produce such stools : (*a*) The presence of abundant fat. If too much fat is present, the specimen of feces when crushed between the slide and the cover-glass spreads uniformly over the glass and forms no fissures at the margins when the pressure is relieved. It is tough and sticky in consistence, and remains spread over the surface of the glass. (Exactly the same may be seen when there is an intimate admixture of mucus with fecal matter.) (*b*) The presence of a large percentage of water. When too much water is present, the specimen is not uniformly crushed beneath the cover-glass and is not so tough, but forms little bands and shreds immediately the pressure is relaxed. (*c*) The presence

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[1] Carter and McMunn, *Lancet*, 1899, vol. ii.
[2] T. J. Walker, *Medico-Chirurg. Trans.*, vol. lxxii., p. 257.

of abundant vegetable residue, chiefly young parenchymatous cells, for instance, from certain forms of cabbage, pears, apples, plums, etc. This admixture can usually be recognized with the naked eye, and if not, can certainly be discovered with the microscope. Softness of the stools may also be due to the intimate admixture of mucus—a condition which will be referred to again elsewhere, as it is of special clinical importance.

The chemic reaction of normal dejecta to litmus is usually neutral or alkaline. I have tested this times without number, and am positive on this point. Hoefele very recently stated the results of 620 examinations in individuals with chronic afebrile ailments as follows : In 72.5 per cent. the reaction was alkaline ; in 5 per cent., neutral ; in 22.5 per cent., acid. Occasionally changes in the diet cause a change in the reaction of the stools. If the diet consists largely of vegetable material, the stools are apt to turn faintly acid, owing to acid fermentation of carbohydrates within the intestine. On a diet in which meat and albumin predominate, ammoniacal fermentation is more apt to occur, so that the reaction of the stools becomes alkaline. The various changes in the reaction of the stools that are seen in different diseases of the intestine will be described at the same time as the diseases in question.

The disagreeable odor, even of normal dejecta, is due chiefly to the presence of skatol, and in a less degree to the presence of indol. Certain gases that are usually present in the dejecta, as sulphureted hydrogen and certain hydrocarbons, are responsible for the familiar nauseous odor. Active fermentation frequently gives rise to a penetrating acid odor. In presence of anatomic elements, pus, or blood, the odor of feces is at times foul.

Microscopic examination of normal feces reveals the surprising fact that a large proportion of the total mass consists of vegetable organisms of a very low order—in other words, of bacteria of the most varied species. Woodward, in his excellent work, mentioned this fact long ago, and my experience entirely confirms his statements. Sucksdorff estimates from averages he obtained that a healthy individual passes with his stools about 53,000,000 bacteria. Details will be found on pp. 35–48.

The remnants of the ingesta that are found in the feces deserve special description. It is, of course, impossible to enumerate the almost incredible things that are occasionally found in the dejecta of omnivorous man—things that are swallowed on purpose or that get into the gastro-intestinal tract accidentally with the food—but a brief account will be given of the remnants of the usual diet of civilization, prepared in the ordinary modern way. In normal stools there may be found :

Vegetable Cells.—These are derived from all the varieties of vegetables and fruit that are eaten. The more cellulose the plant contains, the greater is the amount of unchanged vegetable cells. Among the objects that pass through the gastro-intestinal tract and are evacuated with the feces in an almost unchanged form are cranberries, the outer covering of plums, apples, and pears, large pieces of the parenchyma

of these fruits, cabbage, etc. For more extended information see the monographs of van Ledden-Hulsebosch and of Schmidt and Strasburger.

I have looked for *starch* in the feces thousands of times, and with the following result : On an ordinary mixed diet containing a considerable quantity of bread and rolls starch never occurs in the form of isolated granules or balls in the stools of healthy individuals. Whenever, therefore, starch is found in this state, it must be considered a sign of some pathologic condition. Very small particles of material that are colored blue with iodin and may be considered fragments of starch-granules are occasionally present in very small numbers in the stools of adults. As a rule, however, even these are completely absent when a mixed diet is taken. Further than that, the starch-granules almost entirely disappear from vegetable cells in their transit through the gastrointestinal tract in normal and healthy digestion. Occasionally, however, starch is found in this form even in normal feces. In diarrhea of the colon starch is rarely found in the stools. The appearance of considerable quantities of starch in the feces, it may be repeated, is always pathologic. Later investigations of A. Schmidt and Hulsebosch gave the following, practically coinciding, results : Isolated free starch *granules* are absent with mixed diet, but free starch occurs as unformed, pasty clots, even though it may often escape detection microscopically on account of insufficient iodin reaction. In nearly all normal stools are found starch-remnants (from potatoes, pulse, vegetables) in an integument of cellulose. An increase in the quantity of such starch does not necessarily point to defective amyloid digestion. At most it may indicate insufficient dissolution of cellulose. But the frequent occurrence of isolated, more or less pasty, starch-remnants always indicates a disturbance of digestion, though it may be but a slight one. Even the " fermentation dyspepsia in the small intestine of adults " (Schmidt and Strasburger) is sufficient to cause it.

The feces always contain *muscle-fibers* when any meat whatever is eaten ; even when the digestion is perfectly normal, well-preserved bundles of muscle-fiber can be found, but only in very small numbers. Generally single fibers are seen, distinctly showing the normal structure, or as peculiar configurations that are either round or irregularly angular, and apparently completely homogeneous in structure, but which always show a rectilinear boundary and a suggestion of transverse striation under a good microscope. It is a peculiar fact that these fragments of muscle-tissue are very frequently stained yellow by bile-pigment, though Lynch has occasionally observed it uncolored. Both Schmidt and Hulsebosch have recently stated that the particles I described as " mucous granules " are not mucus, but remains of muscle, appearing entirely homogeneous, without the least trace of striation.

In diabetic patients who eat an excessive amount of meat, in fever cases, if a little meat is occasionally eaten, but especially when there is very energetic peristalsis of the small and large intestine, however set up, muscle-tissue is found in large quantities in the stools. These remnants may be visible to the naked eye and may be recognized as muscle-

tissue on simple inspection, or again they may appear as brown spots no larger than a pin's head. It is therapeutically important to remember that in many diseases of the intestine fragments of muscle-tissue and muscle-fibers are evacuated in large quantities, whereas starch is completely absent from the stools or is present only in minimal quantities.

With a meat diet, *connective-tissue* and *elastic fibers* are naturally found in the feces. [According to A. Schmidt,[1] the presence of nuclei in connective-tissue fragments indicates disturbance of normal intestinal digestion, since nucleins are digested in the intestine.—Ed.]

The "meat test" of Schmidt mentioned above is carried out as follows : Feed the subject in the evening 100 grams chopped beef, slightly roasted (Boas and Zweig use raw beef). By means of Boas' feces colatorium examine the excreta of the next two days for meat remains and connective tissue. If the result is positive—*i. e.*, if the microscope reveals appreciable remains of muscle-fiber or connective tissue—the indication is primarily of a disturbance of digestion in the small intestine. The presence of considerable quantities of connective tissue, together with small quantities of meat, indicates gastric indigestion.

On a diet consisting chiefly or exclusively of milk, small masses of *coagulated milk* are passed in the liquid stools, and under the microscope these will be found to inclose numerous droplets of fat. When eggs are eaten, pieces of *coagulated white of egg* are often found in diarrheic stools. It may here be remarked that in certain pathologic conditions casein may appear in the stools in the shape of more or less spheroid masses that may be as small as half a lentil or as large as two peas ; externally, they are of either a pale-yellow or a brownish-yellow color, due to the bile staining, but internally they are always milk-white.

On a mixed diet *fat* is constantly present in the stools. The quantity passed with the feces varies greatly and is directly dependent on the amount of fat ingested. The larger the amount of fat eaten, the larger the amount present in the stools, for the powers of the intestine to digest and to absorb fat are strictly limited. Fat may appear in the feces in different forms : it may either be saponified—that is, the acid radical may be united with bases—or it may be present as neutral fat or finally as fatty acid. It very rarely takes the form of droplets, but may do so if the diet consists mainly of milk. Very frequently it is present in the form of needles or sheaves. The needle-shaped fat-crystals consist either of free fatty acids or of the calcium or magnesium soaps of the higher fatty acids. If a large amount of fat is present in the form of droplets or sheaves, the stools become pultaceous and oily. The so-called fatty stools will be discussed in a special paragraph.

Certain salts, chiefly inorganic in character, are an important constituent of the stools. Some of these salts are constantly present in the feces. The most important inorganic salts are the *triple phosphates* (ammoniomagnesian phosphate). Schönlein-Remak at one time considered the presence of ammoniomagnesian phosphate in the feces as

[1] A. Schmidt, *Deutsch. med. Wochenschr.*, Dec. 7, 1899.

pathognomonic of typhoid fever. Opinions were divided on this subject until Szydlowsky pointed out that triple phosphates might be present in any normal stool and in every variety of morbid process, quite independently of the reaction of the stool. Microscopically, triple phosphates assume different forms : in liquid or pultaceous stools they appear as well-developed coffin-lid crystals of different sizes ; the same applies to the crystals imbedded in mucus. Occasionally, though rarely, they appear as beautiful feathery crystals ; in other cases again as coffin-lid crystals that are cracked, split, or broken in different ways. Crystals are often incomplete, owing to the fact that pieces have been broken off ; in other cases such relatively enormous masses of crystals are formed that they are visible to the naked eye as small white dots on the slide, while under the microscope they appear as closely packed heaps of well-developed crystals. In solid or pultaceous stools they usually appear as fragments of splintered coffin-lid crystals that may, of course, assume a great number of different shapes. They may be triangular, quadrangular, polyangular, or quite irregular. Together with these fragments a number of transition forms leading to normal crystals are seen. It is a characteristic feature of these different forms of ammoniomagnesian phosphate that they are hardly ever bile-stained ; in fact, I have met with this pigmentation on only two or three occasions.

Second in importance to the crystals of triple phosphate are *calcium salts*, probably of the fatty acids. They are usually yellow, and in contradistinction to the triple phosphates are nearly always bile-stained. These calcium salts also appear in the majority of normal stools, and are frequently seen in the feces in various pathologic conditions. Occasionally they are present in such abundance that, to the naked eye, they appear as brown spots on the slide. They have no definite crystalline form, and, as a rule, form lumpy masses of irregular outline, but occasionally they are elliptic, oval, or almost circular in shape.

Neutral phosphate of calcium is another equally common constituent of normal feces. It is not stained by the bile-pigments. It usually forms masses of varying size, composed of short, thick, wedge-shaped bodies arranged with their apices converging toward a common center.

Oxalate of calcium in the well-known typical form appears in the feces only after vegetables have been eaten.

Cholesterin, according to Maly, Hoppe-Seyler, and others, can always be recognized in the normal feces by chemic methods. Microscopically, however, it is not often observed in its typical crystalline form, and even when crystals of cholesterin are found, they possess no diagnostic significance. Particular care should be taken not to mistake fragments of triple phosphate crystals or remnants of vegetable cuticle for plates of cholesterin.

Spindle-shaped pointed octahedra identical with Charcot's crystals have been seen in the dejecta since the year 1880 by several observers, including myself. Leichtenstern discovered the interesting fact that the formation of Charcot's crystals in the intestine, and consequently their appearance in the feces, is, if not always, at least very frequently, due

to the presence of entozoa of any kind in the intestine. This was subsequent to the description of them in the stools in ankylostomiasis by Bizzozzero, Baeumler, and Perroncito. That these intestinal parasites stand in causative relation to the crystals is, as Leichtenstern thinks, very likely, when we consider the great frequency of their occurrence together, even though this is not always the case. Still the crystals have been observed in the feces in other, and entirely different, diseases. For example, in typhoid fever, tuberculosis, dysentery, chronic catarrh (by myself), in enteritis membranacea and in pus (Schmidt), and in enteritis catarrhalis (Lynch).

[It has been suggested that there is a definite relationship between the presence of eosinophile cells in the feces and Charcot-Leyden crystals. Eosinophilia is definitely related to the presence of animal parasites in the body, and eosinophile cells may appear in the feces when the alimentary canal is infected. In 4 cases of amebic dysentery in children Amberg[1] found both eosinophile cells and Charcot-Leyden crystals in the feces.—Ed.]

Under normal conditions *formed elements* derived from the intestinal wall are rarely found in the feces in appreciable numbers. Sometimes the number of these elements is exceedingly small, and it is difficult to find any. In pathologic cases, on the other hand, very large numbers of formed elements can be seen.

Squamous epithelium is very rare. In exceptional cases it may be seen in the mucus present in pultaceous stools, or occasionally in specks of mucus adherent to the outside of a large fecal mass. In the latter instance this epithelium must have been mechanically detached from the surface of the anal orifice. Cells having the characteristics of squamous epithelium are pathologically important only when derived from some new growth of the intestine.

Columnar epithelium is very frequently found in the dejecta. Virchow and Lambl have called attention to this fact. As a rule, this epithelium is colorless; occasionally, however, it may be tinged yellow by bile-pigment. Sometimes the columnar cells are quite unchanged, and in exceptional cases the margin even may be preserved, or they may present the appearance of well-developed goblet-cells. As a rule, however, they are more or less changed, usually to such a degree that an inexperienced observer who sees them for the first time will hardly recognize them as columnar epithelium. All forms and all transitions are seen. In some of the extreme degrees of metamorphosis the epithelial cells consist of small, homogeneous, lusterless, spindle-shaped structures without a nucleus, which, on treatment with carmin, become stained a little more intensely than the other constituents of the stool, but no trace of a nucleus can be discovered within them. From these extreme modifications a great number of transition forms gradually lead to well-preserved, almost normal, columnar epithelium. I have called these changes in the epithelium " Verschollung " (flaking), a term that has found general acceptance. I considered it the result

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[1] Amberg, *Johns Hopkins Hosp. Bull.,* Dec., 1901.

of the withdrawal of water from the epithelial cells, and Kitagawa considered it a form of degeneration. Schmidt more recently has explained it as peculiar imbibition of fat-soap by the cellular protoplasm. The epithelium is almost exclusively found in the mucus of the stools. The manner in which the mucus is mixed with the stools seems to make no difference. The so-called clouding of the mucus, its want of transparency, in the stools of intestinal catarrh, is almost invariably due to the presence of enormous numbers of epithelial cells and not of round-cells. There is, therefore, a great difference in this respect between the catarrhal excretion in diseases of the intestinal mucosa and the catarrhal expectoration in diseases of the respiratory mucosa, for in the latter the turbidity of the mucus is due to the presence of round-cells, and in the former to columnar epithelial cells. The number of cells present fluctuates greatly. The most numerous and best-preserved specimens are found in thin diarrheic stools.

Round-cells—i. e., *pus cells*—hardly ever occur in normal feces, and at the most there are only isolated cells in the scanty flakes of adherent mucus. There is no advantage in attempting to decide whether the cells found in normal dejecta are mucous cells, pus cells, lymphoid cells, or white blood-corpuscles. The significance of the copious production of these cellular elements in a variety of pathologic conditions will be discussed subsequently.

In connection with the preceding paragraphs a few peculiarities of pathologic dejecta must be considered. They will be fully described in the sections dealing with the different forms of intestinal disease, but it will probably be useful to describe them here in a connected form and from different points of view. It will be easier to do this now than to insert these descriptions later on.

MUCUS IN THE FECES.

Mucin, the product of Lieberkühn's crypts, can always be detected in normal feces by chemic examination. Even in a perfectly healthy intestine mucin is continually formed. Not a trace of mucus, however, can be seen macroscopically or microscopically in the normal stools of adult subjects. The amount of mucus present in healthy feces is so small, and it is so intimately mixed with the intestinal contents, that it can be recognized only by chemic tests. It therefore follows that the discovery of mucus in the stools, either with the naked eye or microscopically, always indicates some deviation from normal physiologic conditions. The presence of mucus, however, does not necessarily indicate the existence of an anatomic lesion of the intestine; it may be due to physiologic hypersecretion, as in the thin and clinically insignificant layer of mucus surrounding a solid column of fecal matter, which, after the mucus has become dry, seems to be covered with a layer of shellac or varnish. Tiny flakes of mucus may also adhere to solid stools. These admixtures of mucus are unquestionably due to the irritation exerted by large fecal masses on the mucous

lining of the lower part of the rectum when they are allowed to remain in this portion of the bowel for a considerable time.

Under all other circumstances the detection of mucus is pathologic. It assumes various forms in the excreta.

(*a*) The mucus may form an abundant and thick coating on solid fecal masses. In this instance the mucus is either glassy and clear, or it may be clouded, mainly by epithelial cells, least frequently by round-cells, but is always of a gray or whitish color. Any brownish-yellow tint that may be seen in it is due to the contact of fecal matter.

(*b*) The mucus is intimately mixed with the fecal masses, which may, on this account, vary in consistence. The feces may be a watery liquid with shreds of mucus floating in it; if there is more mucus than fecal matter, the stools are thick, syrupy, or gelatinous in character. Or, again, the fecal masses may be pultaceous; when this is the case, it is often necessary to lift the mucus out of the fecal mass with a rod, as otherwise it is impossible to recognize its character. Finally—and this is an important feature—very small shreds of mucus may be intimately mixed with formed fecal masses. In this instance the mucus can be discovered only microscopically. Under the microscope it appears as small, hyaline, whitish-gray, translucent islets that all look alike. In these cases mucus need not necessarily be present in shreds. Schmidt disputes the mucous composition of the last, which I have termed "hyaline mucous islands," without giving any other explanation of their nature and origin. Their macroscopic appearance reminded him of dead monads, to which I myself called attention years ago.

(*c*) The mucus may be passed in enormous quantities, so that occasionally the dejecta consist exclusively of shreds of glassy or gelatinous mucus, and contain no fecal matter proper at all.

(*d*) Peculiar structures, looking like frogs' spawn or grains of boiled sago, are also occasionally found in the feces; these Virchow many years ago declared to be, in the majority of cases, of vegetable origin. I quite agree with this view, although it has been usual to consider them as pellets of mucus derived from ulcerating follicles in the large intestine (compare the section on Ulceration of the Intestine). Some investigators, of whom Kitagawa is among the most recent, assert that these peculiar structures may either be vegetable in origin or be derived from the follicles. Kitagawa, however, does not consider them pathognomonic of any definite anatomic lesion, and certainly not of follicular ulceration. He argues that their pure mucous character militates against such a conception.

(*e*) Mucus in the stools may also appear in a form that I have called yellow granules of mucus ("Schleimkörner"). These consist of small granules that may be barely visible or may be as large as a pin-head; usually they are about the size of a poppy-seed, and occasionally as big as a pea. Their color is deep yellow to brown, and in consistence they resemble butter. Occasionally there are only one or two of these bodies; in other cases they are present in such enormous numbers that they are disseminated throughout the stool. As a rule, in these cases the

feces are pultaceous or very thin, so that the granules look like brown poppy-seeds scattered throughout the dejecta. (Incidentally, it is well to emphasize the fact that little brownish specks of this kind in the dejecta may be of four-fold origin. They may be—(1) Small pieces of meat ; (2) fragments of some vegetable ; (3) yellow calcium salts ; (4) yellow mucous granules.) Chemic tests prove that these yellow or brown nodules consist either of mucus or at least of a substance that is very closely related to mucin, and that their yellow color is due to bile-pigment. Schmidt, Boas, and Schorlemmer believe that, according to their microscopic and chemic behavior, the yellow granules are not mucus, but albuminous matter : either, as already stated above, structureless remains of muscle substance, or remains of casein or egg-albumen colored with bile-pigment.

In describing the different forms of intestinal disease the diagnostic conclusions to be drawn from the presence of mucus in the stools will be discussed. Special attention will then be paid to the diagnostic significance of the different forms of mucus, and it will be seen that much depends on the manner in which the mucus is mixed with the other constituents of the feces. A clearer picture of these different matters can be gained, however, if a few general preliminary remarks are given here.

It may be taken as an axiom that the appearance of mucus in the dejecta always indicates some pathologic condition of the mucosa of the intestine. This is the case only, however, when the mucus can be recognized by inspection—that is, either with the naked eye or microscopically—and does not apply when the presence of mucus can only be discovered by chemic tests, for the presence of such minute quantities is still physiologic. An exception to this rule is the mucus occasionally seen under physiologic conditions, referred to above—namely, the thin layer of mucus that frequently forms a coating around very large fecal masses. The presence, then, of visible mucus usually indicates catarrh of the intestine—that is, a pathologico-histologic alteration of the mucous membrane that is characterized by the formation of a secretion which runs away and in which mucus occupies an important position. It is quite immaterial what portion of the intestinal canal is affected by catarrh or what the primary causes of the catarrh may be.

There are only two exceptions to the rule that the presence of visible mucus always indicates catarrhal irritation of the intestine. One of these is hypersecretion of mucus, due, most probably, to purely nervous processes. This phenomenon is the primary cause of the symptoms characterizing colica mucosa or enteritis pseudomembranacea (see the section on this disease). The other exception will be discussed in the section on Intestinal Dyspepsia. The special feature of this condition is that it simulates intestinal catarrh and occurs in patients who are free from that disease.

[R. Schutz[1] insists on the importance of an excessive quantity of mucus as evidence of intestinal catarrh. The passage of mucus without feces

[1] Schutz, *Berlin. klin. Wochenschr.*, July 10, 1899.

or mucus surrounding scybala shows that the colon is inflamed. When mucus is intimately mixed with fecal matter, there is catarrh of the upper part of the colon or of the small intestine. Mucus with much food residue points to catarrh of the small intestine.—ED.]

ACHOLOUS AND COLORLESS STOOLS.

The ordinary light- or dark-brown color of the stools is due to the presence of altered bile-pigment. If bile-pigment is absent from the stools, the feces are unpigmented—that is, they are grayish-white, clay colored, or ashy gray. As a rule, the dejecta under these conditions emit a penetrating odor, are of the consistence of butter, and on chemic or microscopic examination are found to contain much fat. The latter is present in the form either of needle-shaped crystals or of sheaves of crystals that appear in enormous numbers, or it may be present, but to a less extent, in the form of fat-droplets. Gerhardt considered these needle-shaped crystals of fatty acid to be tyrosin, but I maintain that this is not the case, and that these crystals are fatty acids and soaps. Von Jaksch, Rosenheim, Oesterlein, Stadelmann, and others bear me out in this view. Experience shows that as soon as bile is absent from the intestine the absorption of fat is greatly interfered with (see the section on Fatty Stools).

In cases in which the common duct or some of the smaller bile-passages are occluded by some pathologic process it is easy to understand why the bile is excluded from the intestine and why a deficiency of color in the intestinal contents results. As a matter of fact, acholic stools are seen as a complication of icterus almost daily.

Bunge has stated that the grayish-white color of feces resulting from occlusion of the bile-duct is not the result so much of the lack of bile-pigment as of the great quantity of fat that results from the scantiness of the bile supply. If the fat is removed by ether-extraction, the fecal mass, though bile-free, appears dark brown—i. e., the color of the hema-tin of the ingested meat, which is no longer obscured by the lighter shade of the fat. Fleischer has corroborated this statement.

On the other hand, however, the stools may be completely devoid of color and of a clayey or whitish-gray color, although there is not the slightest trace of icterus or any symptom of occlusion of the bile-pass-ages. Bamberger and others described this peculiar symptom some time ago. These colorless stools differ from genuine acholic stools—that is, stools which contain no bile—in this respect: the former, it is well known, frequently emit a penetrating putrid odor, whereas the odor of the other class of colorless stools is less putrid and frequently more acid in character. Almost always, however, they also contain enormous quantities of fat, just like the true acholic stools. Berggrün and Katz have demonstrated the presence of urobilin in these apparently acholic stools; von Jaksch and Pel have made a similar observation.

Clay-colored stools without icterus are seen in a great variety of conditions. It is well known that the rice-water stools of cholera, and occasionally the dejecta of dysentery, are quite acholic. This is

due to certain definite conditions that need not be discussed in detail here. Acholic stools are occasionally seen in leukemia, in carcinoma of the stomach and intestine, in simple intestinal catarrh in children, and most frequently in debilitated patients suffering from phthisis. Von Jaksch has also seen acholic stools in a great variety of morbid processes—such as tuberculosis of the intestine, chronic nephritis, chlorosis, and scarlatina. Berggrün and Katz are inclined to attribute an important and special symptomatic significance to clay-colored grayish stools in chronic tuberculous peritonitis occurring in children.

What is the explanation of this clay color of the feces in cases in which no icterus is present and in which the bile-ducts are quite pervious? Formerly it was believed that the formation of bile was reduced or inhibited (acholia). This view, however, is not tenable, for even in the most serious affections of the liver, and especially in many of the above-named diseases, a sufficiency of bile is formed. As a matter of fact, acholia can be excluded by the demonstration of urobilin in the stools of these patients. The results of clinical observation also refute this idea, for in many of the cases that I have watched for a long time the clayey appearance of the dejecta did not persist indefinitely, though the primary disease continued unchanged. Very often the passage of clay-colored stools is followed in a few days by the passage of normal bile-colored feces. I have seen the same patient pass a bile-stained and a colorless stool in the same day; indeed, one portion of the same stool may be clay colored and another portion brown.

Some other explanation must, therefore, be looked for. It is possible that the large amount of fat present in these stools may be responsible for the pale color in whole or in part. (See the preceding statements of Bunge and of Fleischer.) This may perhaps be the explanation of the appearance of acholic stools in those diseases in which a diminished absorption of fat leads to the passing of fatty stools—as, for instance, in tuberculosis of the intestine and the peritoneum.

Another explanation would be that the bile-pigment is so altered in the intestine that it is either not converted into its normal reduction product (urobilin) or that colorless decomposition-products of bilirubin (von Nencki's leuko-urobilin) are formed. The first of these views has been advanced by von Jaksch. This explanation is probably the correct one in most of the cases in which colorless stools give the reactions for bile. As a matter of fact, it is the only conceivable explanation in those cases in which the dejecta are gray or clay colored, but in which, as I have repeatedly seen, no exceptionally large quantities of fat are passed and in which there is no sign of icterus. This explanation, however, merely shifts the inquiry one step backward, for the problem still remains, what causes the formation of leuko-urobilin?

FATTY STOOLS (Steatorrhea).

It has been mentioned that normal stools, when an ordinary mixed diet is taken, always contain a certain proportion of fat, which increases with any rise in the quantity of fat contained in the food (compare

p. 84).   Under pathologic conditions the amount of fat passed with
the dejecta may be so large that it would be impossible to overlook it.
The name steatorrhea is derived from the peculiar appearance that stools
of this character occasionally present—namely, an appearance as of
flowing like oil during the act of defecation.   At the present day this
term is applied to all cases in which isolated masses of fat are present
in the feces and can be recognized with the naked eye.

The form and character of the fat present in increased quantity
under pathologic conditions may vary.   In colorless acholic stools enor-
mous numbers of microscopic fat-crystals are uniformly disseminated
throughout the whole fecal mass (see p. 90).   In diarrhea I have occa-
sionally seen very small, whitish-gray points either lying in the thin
feces or floating on the surface.   These points varied in size from barely
visible specks to masses as large as a pea, and consisted of innumerable
needles of fat crystals mixed with bacteria.   In steatorrhea proper the
fat appears in the feces as whitish or grayish lumps, resembling tallow,
and not often of a softer consistence.   They are rarely larger than nuts.
In other cases the fat solidifies as soon as the stools become cool, so that
it appears as a layer of variable density surrounding the feces or cling-
ing to the sides of the receptacle.   Lastly, some authors state, although
I have personally never seen this phenomenon, that fat may be passed
from the bowels in a fluid form without fecal admixture.   The patients
themselves frequently call attention to these peculiarities of their dejecta.

The chemistry of fatty stools is dealt with on pp. 17–33.   The ques-
tion for consideration here is under what conditions an abnormal quan-
tity of fat can appear in the feces and what the clinical significance of
this symptom may be.

A priori, several possible causes for the evacuation of abnormal
quantities of fat suggest themselves.

In the process of digestion the bile and the pancreatic juice—and,
in addition probably, the succus entericus and certain species of bac-
teria—can apparently dispose of only a limited amount of fat, which
probably bears a definite ratio to the amount of these digestive fluids.
It is, therefore, clear that the ingestion of an excessive amount of fat
may cause an evacuation of the superfluous fat in the feces even when
the bodily functions are normal.   It is universally admitted that the
ingestion of abundant quantities of food containing much fat leads to
the appearance of fat in the feces.

If the mucous lining of the small intestine and the lymphatic system
(the mesenteric glands) lose their powers of absorption, fat must appear
in the stools in increasing quantity, even though the amount of fat
ingested remains constant.   In this manner extensive atrophy of the
mucosa of the small intestine, amyloid disease, tuberculosis of the small
intestine, caseation of the mesenteric lymph glands, chronic tuberculous
peritonitis, and perhaps even simple catarrhal processes may lead to the
passing of stools containing abundance of fat.   Demme and Biedert
have pointed out that in children particularly the absorption of fat is
greatly reduced in catarrh of the small intestine, so that a considerable

quantity of fat is passed in the stools even on a milk diet. Under these conditions the stools look like asbestos, and if examined under the microscope, will be found to contain large numbers of fat-droplets.

It is well known that the presence of bile is essential for the digestion of fat, and consequently it is to be expected that if the entrance of bile into the intestine is insufficient or is entirely arrested, the stools will be markedly changed in regard to the amount of fat they contain. This view is fully borne out by clinical experience.

When the bile is excluded from the intestine, the stools assume the well-known pale-gray color, and at the same time contain a large amount of fat (see above). As a rule, the presence of fat is at first revealed only by microscopic examination and by chemic analysis. Only in rare instances can fat be discovered at once with the naked eye. This explains why in icterus with occlusion of the bile-passages physicians do not speak of fatty stools in the general clinical sense. Bamberger went so far as to say that in a large number of catarrhal inflammations of the duodenum associated with icterus he had never observed the fatty stools which are regarded as a diagnostic symptom of diseases of the duodenum. In reality, however, as soon as bile is excluded from the intestine the dejecta contain an unusually large quantity of fat. This question has been definitely settled by the exhaustive investigations of F. Müller. According to this author, from 55.2 to 78.5 per cent. of all the ingested fat was passed in the feces when the bile was completely excluded from the intestine, whereas under normal conditions only from 6.9 to 10.5 per cent. was evacuated.

The origin of fatty stools in diseases of the pancreas has been the subject of much controversy, and cannot be considered as definitely settled even now. The first discovery in this direction was made by Kuntzmann, who found that in a case of induration of the pancreas with obliteration of the duct of Wirsung there was a causal connection existing between this disease and the presence of large quantities of fat in the stools. The condition of the pancreas was accurately ascertained at the autopsy. [Kuntzmann's observations were made in 1820, but, as Hale White[1] points out, Bright's description of oily stools in malignant disease of the pancreas in 1832 was probably independent. Bright laid stress on the association between ulceration of the pancreatic growth into the duodenum and the fatty condition of the stools.—Ed.] Since Kuntzmann's discovery the subject has been studied with the greatest interest, particularly after Eberle discovered the emulsifying property of pancreatic juice, and Claude Bernard its lipolytic powers. The literature on this important question up to 1878 is given by Friedreich, and since that time by F. Müller and Oser. Some clinicians go so far as to consider the passage of fat in the stools as pathognomonic in the diagnosis of diseases of the pancreas. The coincidence of fatty stools and pancreatic diseases is certainly noteworthy, in particular as in a relatively large proportion of such cases the clinical picture of true steatorrhea is observed. But we must not overlook the fact

[1] Hale White, *Guy's Hosp. Rep.*, vol. liv.

that in the great majority of these cases there existed, besides disease of the pancreas, also icterus and disease of the liver, so that the fatty stools may well have been attributable to these last. Again, in still other cases of pancreatic disease, fatty stools were absent. Such facts as these naturally detract from the value of fatty stools in the diagnosis of pancreatic disease. Müller throws doubt on the view that steatorrhea is a symptom of pancreatic disease *per se*. This author, however, found certain changes in the fat contained in the stools in three cases in which the pancreatic juice was excluded from the intestine (as was definitely ascertained at the autopsy). He did not find any quantitative changes, but there were certain qualitative changes in the fat—namely, that the lipolysis was much slighter, —that is, involved only about 39.8 per cent. of the fat, whereas in healthy subjects or in subjects with icterus in which the pancreatic juice had free access to the intestine, the lipolysis had proceeded much further, accounting for 84.3 per cent. of the fat that appeared in the stool. [Edsall's observations support Müller's conclusions.—Ed.]

It is claimed by some that in diabetes the absorption and the general metabolism of fat are reduced; this view, however, is supported by very little evidence and many authorities deny it entirely. At all events, the appearance of fat in the stools cannot be considered one of the clinical symptoms of diabetes.

Oser sums up our present knowledge of the matter as follows: (1) Steatorrhea *per se* is not diagnostic of disease of the pancreas. (2) In the absence of icterus and of demonstrable intestinal disease it is possible that the disturbed digestion of fat is due to pancreatic disease. (3) Disturbed digestion of fat may be taken as diagnostic of pancreatic disease. It is not yet positively proved, but Müller's observations are confirmatory.

[INTESTINAL SAND (Intestinal Lithiasis; Sabulum Intestinalis).

UNDER this heading two conditions are included which, though essentially different, so closely resemble each other in some particulars that they must be described in connection with each other.

1. True Intestinal Sand or Enteric Lithiasis.—The material is gritty, and besides organic matter, contains inorganic salts, especially calcium phosphate and carbonate, but no cholesterin, which distinguishes it from biliary gravel or sand, for which it is often mistaken. A large number of micro-organisms are naturally found in the sand.

Intestinal lithiasis is nearly always associated with mucous colic (Dieulafoy[1]), but has been found in association with appendicitis (Oddo[2]). It has been regarded as due to a lithogenic catarrh of the bowel, and might be compared with the process which gives rise to the formation of concretions in the vermiform appendix. Dieulafoy regards the con-

[1] Dieulafoy, *Bull. de l'Acad. de Méd.*, Paris, 1897.
[2] Oddo, *Bull. et Mem. de la Soc. Méd. des Hôp.*, June 25, 1896.

dition as a manifestation of the arthritic diathesis. Duckworth and Garrod[1] believe that true intestinal sand is principally formed in the upper part of the colon, as it is rich in urobilin and poor in unaltered bile-pigment, and must, therefore, be formed in part of the alimentary canal where the conversion of bile-pigment into urobilin is already far advanced.

In Duckworth and Garrod's case the chemic composition of true intestinal sand was as follows:

Water	12.40	Calcium oxid	54.98
Organic matter	26.29	Phosphorus pentoxid	42.35
Inorganic matter	61.31	Carbon dioxid	2.20
		Residue, with traces of magnesium and iron	0.47
	100.00		100.00

Very similar results were obtained by Mathieu and Richaud[2] and by Thomson and Ferguson.[3]

Intestinal lithiasis may be prolonged over many years and is usually associated with mucous colic. There may be either constipation or diarrhea. Its most important symptom is severe colic. The onset of colic may be quite sudden and may be accompanied by rapid abdominal distention. The colic may be due to associated mucous colic or possibly to constipation. It is important to distinguish the pain from that due to appendicitis, gall-stones, lead colic, and intestinal perforation. The patients are often nervous or hypochondriacal, and watch their evacuations with conscientious care. The condition is more often reported in women: two-thirds of Dieulafoy's cases were in the female sex. The most common age seems to be between thirty and forty, but it may occur in infants (Saint Philippe),[4,5] though some of the reported cases really belong to the next category.

Treatment.—The associated condition must be treated, such as mucous colitis or constipation. Copious drafts of water, or the mineral waters of Plombière, Vittel, Kissingen, Homburg, etc., have been recommended. Bicarbonate of soda and salicylate of bismuth have been given. For the crises of abdominal pain, antipyrin, opium, and morphin hypodermically have been given, but it is not advisable to give opium or morphin if it can be avoided, as the drug-habit may be acquired, the disease being chronic.

2. False Intestinal Sand (Food Residues).—The constituents under this heading may be of various kinds, but are usually the remains of indigestible vegetable food, especially the sclerenchymatous particles in pears, which may or may not become encrusted with earthy salts. Duckworth and Garrod refer to a description of pear sand by Marcet[6] in 1817. The seeds of figs, the residue of barley meal (Delé-

[1] Duckworth and Garrod, *Medico-Chirurg. Trans.*, vol. lxxxiv., p. 389.
[2] Mathieu and Richaud, *Bull. et Mem. de la Soc. Méd. des Hôp.*, Paris, 1896, p. 473.
[3] Thomson and Ferguson, *Jour. Path. and Bacteriol.*, vol. vi., p. 334.
[4] R. Saint Philippe, *Jour. de Med. de Bordeaux*, 1901, No. 49.
[5] Shattock, *Trans. Path. Soc.*, vol. xlviii., p. 124.
[6] A. Marcet, *Essay on Calculous Disorders*, 1817, p. 132.

pine[1]), gooseberry jam (Georg[2]), or even the vertebræ of sardines (Rapin[3]) have also been found in false intestinal sand. In a three-months-old baby yellow sandy particles in the stools were associated with attacks of colic and green stools, and were found, by Moussous,[4] to be composed of fatty acids.

A rather different condition, inasmuch as it is due to drug rather than to food residues, may be mentioned. Olive oil, when taken in large quantities for cholelithiasis, may, as the result of partial digestion, appear in the motions as small masses which may possibly be regarded as the remains of biliary calculi, and so give rise to an erroneous idea of the efficacy of this form of solvent treatment for gall-stones. Salol, when taken by the mouth, may give rise to calculi in the bowel (*vide* enteroliths). False intestinal sand is not often associated with colic or pain, and when there is pain, it is probably due to constipation, for which pears, figs, and other fruits are often taken. The dependence of pear sand on the ingestion of pears can be shown by stopping the intake and then beginning it again (Thomas[5]).—Ed.]

SLUGGISHNESS (CONSTIPATION) OF THE BOWELS
(Obstipatio Alvi).

In a perfectly healthy individual the bowels act once in every twenty-four hours, and, when the conditions of life are regular and uniform, usually at about the same time of day. The essential causes of this rather striking periodicity are still unknown, but it probably depends on the nervous system.

Observation leads irresistibly to the conclusion that the nervous stimuli influencing peristalsis of the large intestine under normal conditions do not act in the same manner as the nervous influences causing peristalsis of the small intestine. The contents of the small intestine are propelled onward quite rapidly, and reach the ileocecal valve in the course of a few hours. The passage of the bowel contents through the colon and the rectum, on the other hand, is slow. The nerves of the rectum become educated, as it were, by long habit, so that in most persons the contents of the intestine pass from the main reservoir—*i. e.*, the sigmoid flexure and the upper part of the rectum—every twenty-four hours into the ampulla of the rectum, where they produce the peculiar sensation which leads to the act of defecation and is spoken of as tenesmus if it is pathologically exaggerated.

The physiologic evacuation of the rectum may vary in many respects in perfectly healthy individuals. Some persons do not empty the bowels more than once in every two, three, or four days, and this without any resulting derangement of health ; others again evacuate the bowels two or three times a day. Still greater deviations from the usual rule can-

[1] S. Delépine, *Trans. Path. Soc.*, vol. xli., p. 111.
[2] C. Georg, Jr., *Physician and Surgeon*, May, 1900.
[3] Rapin, *La Méd. Mod.*, April 10, 1897.
[4] Moussous, *Jour. de Med. de Bordeaux*, April 4, 1897.
[5] D. Thomas, *Australasian Med. Gaz.*, November, 1891.

not be regarded as physiologic, for the majority of the individuals in question present certain abnormal symptoms. If the intervals between the evacuations of the bowels are longer than those just mentioned, the act of defecation is rarely accomplished physiologically, and artificial help must often be employed. In addition a number of distressing symptoms may appear, and finally, as a rule, definite anatomic or other abnormalities supervene. In cases of this kind, therefore, retention of the feces is a symptom of disease.

Reichmann has recently announced that many persons who feel perfectly well, and who, on examination, appeared normal, defecate but once in two or three weeks or still less frequently. He attributes this anomaly, which requires no treatment, to a deficient formation of feces. We shall leave this matter as undecided at present.

In popular parlance constipation (obstipatio alvi) is that condition in which feces are not passed sufficiently often. Another form of retention of feces is recognized in which defecation occurs daily, but the motions are insufficient in quantity. Many instances of this condition are recorded in the literature. I remember the case of a public official who asserted most positively that he had always made a point of having a daily evacuation of the bowels, and yet he died of peritonitis following a perforation caused by a large number of enormous fecal masses as hard as stone occupying the intestine.

It is hardly possible to specify a definite quantity as the normal average amount of feces that should be evacuated a day. Much will depend on the nature of the food and its quality, but these are not the only factors involved. Woodward first expressed the opinion that a large proportion of the normal bowel contents passed at stool is not, as is usually assumed, composed of remnants of food, but consists of innumerable micro-organisms. My own investigations are in entire agreement with Woodward's statement. L. Hermann has reported an interesting observation that bears out this view. He placed two ligatures around a loop of intestine in a dog, the intervening portion being completely empty. The ligatures prevented the entrance of any food into this loop, but a lumpy mass was nevertheless found within it, resembling ordinary feces in some respects, and consisting of products derived from the intestinal wall (mucus and epithelia), together with micro-organisms. Voit has also shown that in cases of prolonged starvation a considerable amount of excrementitious matter—the so-called feces of inanition—is given off from the intestine.

These facts explain the peculiar phenomenon that patients who eat very little may, nevertheless, pass a large quantity of feces, and they also show that reliable conclusions in regard to the adequate evacuation of the bowels cannot be arrived at by merely making separate estimations of the amount of food ingested and the amount of feces passed.

ETIOLOGY.

Insufficient evacuation of the bowels is one of the commonest occurrences. Its interpretation varies with each case, and its causes are

manifold. Not only for convenience, but also for scientific and practical purposes, I deem it wise to arrange all these etiologic forms synoptically. It will lighten the task of the practitioner, who must study each individual case etiologically, anatomically, and clinically, to determine his course of treatment. To proceed, then, we distinguish three chief, etiologically different forms of constipation :

(*a*) A group in which sluggishness of the bowels is dependent on the perversion of certain functions that still remain physiologic.

(*b*) A group in which sluggishness of the bowels is the direct result of certain pathologic states and constitutes a more or less important symptom associated with other abnormal conditions.

(*c*) A group in which sluggishness of the bowels is the essential feature of the disease and forms an apparently (or, perhaps, really) independent pathologic type (habitual sluggishness of the bowels).

[Illoway[1] gives the following classification of chronic constipation according to the mode of production :

(*A*) Chronic constipation produced by well-defined morbid processes.

    (1) Obstruction of the lumen of the bowel—strictures, growths of bowel, tumors pressing on the bowel.

    (2) By impairment of secretions poured into the intestines—in chronic disease of liver and pancreas.

    (3) By inhibition of peristalsis through the nerve centers—in chronic diseases of brain and spinal cord, in chronic insanity, diphtheric paralysis, tabes, etc.

    (4) Chronic venous engorgement of portal system.

    (5) By voluntary abstention from stool on account of the pain it causes by reason of a diseased condition of the rectum—in piles, fissure, ulcer, etc.

    (6) By changes in the mucous membrane which impair its irritability and render it incapable of performing the physiologic function in digestion—chronic catarrh, mucous colitis, atrophy after catarrh.

    (7) By atony of the intestinal muscles produced by morbid conditions of stomach and bowels.

(*B*) Chronic constipation from foreign bodies.

(*C*) Chronic constipation from—(1) Malformations—abnormally developed colon ; undue size or length of sigmoid flexure ; diverticula of large bowel ; diaphragms in large bowel.

    (2) Defective development or essential primary atrophy of the colon.

    (3) Dislocation or enteroptosis.

(*D*) Chronic constipation from impaired physiologic function.

    (1) Perverted action—enterospasm ; spasm of sphincter.

    (2) Imperfect performance of physiologic function, to which cases to be considered in this section chiefly belong.—Ed.]

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[1] Illoway, *Constipation in Adults and Children.*

(1) The character and the quantity of the food, the amount of bodily exercise, and numerous other factors influence the evacuation of the bowels in many ways. Both qualitative and quantitative changes in the food may cause sluggish peristalsis. Individual powers of reaction are concerned in this process—that is, the bowel may react in a certain way to a change in the diet in one person and not in another. In many persons an exclusive milk diet causes sluggishness of the bowels, while in others it causes diarrhea. On a diet consisting chiefly of coarse vegetable food the movements are usually more frequent to start with, but subsequently constipation supervenes. This can probably be explained on the hypothesis that at the outset the intestine is irritated mechanically, with the result that peristalsis is intensified, but that eventually the bowel becomes accustomed to this irritation, or even becomes, so to say, fatigued. If the excretion and exhalation of water through the skin, the lungs, and the kidney are increased, whether artificially or as the result of certain physiologic or pathologic processes, constipation may result. The same may occur if the amount of fluid ingested is reduced.

Active physical exercise promotes regularity in the evacuation of the bowels, no matter what the character of the exercise may be, whether walking, riding, gymnastics, or exertion of a similar nature. If this physical exercise is stopped, as, for instance, when a railway journey of twenty-four or forty-eight hours is taken, constipation usually occurs, while sedentary habits or confinement to bed during illness usually have the same effect. It is probable that in physical exercise the exaggerated respiratory movements, the more marked excursions of the diaphragm, and possibly the stimulation of peristalsis due to the energetic circulation of the blood through the intestinal walls, are all factors in producing the effect.

Extremely corpulent persons occasionally suffer from sluggishness of the bowels, probably from not taking sufficient exercise. Obesity *per se*, however, does not necessarily produce a sluggish peristalsis, for there are plenty of patients with a degree of obesity amounting to a disease, whose bowels, nevertheless, act every day with the greatest regularity.

To enumerate all the various possible forms of constipation of the character discussed in the preceding paragraphs would be superfluous. Many other forms occur under physiologic conditions and in disease. The observant practitioner, remembering the leading physiologic principles concerned in the normal action of the bowels, will readily recognize them.

(2) Sluggishness of the bowels in various degrees, amounting ultimately to complete retention of the feces, is exceedingly frequent as a concomitant symptom in a number of the most diverse pathologic conditions, of which only a brief summary can be given here. When the various diseases of the intestine are under consideration, the relations that exist between these several pathologic states and the symptoms of constipation will be described.

Constipation is of frequent occurrence in a number of diseases of the stomach and intestine, such as round ulcer of the stomach, pyloric stenosis, dilatation of the stomach, and obstruction to the entrance of bile into the intestine. In the latter class of cases constipation is, at least in part, due to a decrease in the amount of fluid passing into the intestine, although unquestionably other and probably more important factors are also concerned in the process. Retention of feces in any of the numerous forms of stenosis or occlusion of the lumen of the intestine is, of course, readily understood. The phenomena of defecation in chronic catarrh of the intestine will be discussed in detail in the section on this disease, but as there is a wide-spread belief that chronic catarrh of the intestine is a very frequent cause of habitual constipation, and as this question is exceedingly important, it may here be stated emphatically that this view is erroneous. The anatomic lesions characteristic of chronic intestinal catarrh by no means always lead to sluggish peristalsis. It is certainly wrong to make a prompt diagnosis of chronic catarrh of the intestine in cases of chronic constipation, and to send the patient to Carlsbad, Marienbad, or some other similar spa. This error, however, is of somewhat frequent occurrence. The case is somewhat different in chronic venous engorgement of the intestine, which may involve large or small portions of the bowel and may be the result of a variety of primary conditions. It is seen in certain diseases of the heart and lungs which lead to obstruction of the circulation in the systemic veins, and consequently to engorgement in the area of distribution of the inferior vena cava. It is also seen in different diseases of the liver which entail engorgement in the area of distribution of the portal vein. In these cases it is only venous engorgement of the intestine that is in question, a condition which may predispose to the development of catarrh of the intestine, but does not of itself in any way exhibit the physiologic changes and functional perversions characteristic of such a catarrh. In venous hyperemia of the intestine sluggishness of the bowels may develop, but this symptom is by no means so constant as is sometimes believed. There are many patients suffering from heart disease and advanced degrees of venous engorgement whose bowels act quite regularly every day.

Peritonitis, both circumscribed and general, may lead to constipation—in the former case from adhesions and in the latter from implication of the muscular structure of the intestine.

Constipation occurs in a number of acute febrile diseases, a variety of factors being probably concerned in its causation under these circumstances. For instance, the patients' ordinary mode of life is suddenly changed; they remain in bed and their food is different from what it was. Besides, the nerves that inhibit the movements of the intestine are irritated by the increased temperature of the body (Bokai), and in many instances the special toxin of the disease itself may possibly exert a direct influence on the bowels. In many chronic diseases a like effect is produced by similar influences, and in addition by some other factors, such, for instance, as the general wasting of all the tissues of the body.

Some diseases of the central nervous system are occasionally complicated by constipation, which may be very obstinate. The following nervous diseases produce constipation : cerebrospinal meningitis, chronic hydrocephalus, cerebral tumors and hemorrhage, and many forms of myelitis, etc. In all these conditions the nerves that inhibit intestinal peristalsis are probably irritated. Constipation is a well-recognized condition in persons who suffer from some depressing emotion or from mental disorder, and may become exceedingly intractable.

(3) Habitual constipation has for a long time been recognized as a definite morbid state in which sluggishness of the bowels is neither (*a*) the result of recognizable physiologic irregularities nor (*b*) a concomitant symptom of any well-marked disease, but in which it is, or at least is considered to be, the essential and primary feature of the clinical phenomena. The great practical importance of this subject justifies a more extensive discussion.

HABITUAL CONSTIPATION.

From the early misconceptions of this affection we have gradually come to understand that habitual constipation may be caused by many varying conditions. Some are anatomic causes, but such that their existence may at times be merely surmised, at other times cannot be ascertained at all. Others are rather functional disturbances. Some of these can be distinctly recognized, others must be accepted hypothetically. Yet difficult as the determination of the etiology may be, it must, nevertheless, be attempted in all cases, because it is indispensable to the choice of successful therapeutic measures. A careful anamnesis, paying attention to the minutest details, and a painstaking examination of the entire organism, with particular attention to the intestines and their functions, will in most cases give sufficient information. Of course, all cases cannot be narrowed down to fit the symptomatology of a single cause. In fact, in numbers of cases there are many causes.

Habitual constipation may be congenital or acquired. This applies to the form of anatomic origin as also to that of functional causation.

The etiologic factors of purely functional nature, without discoverable anatomic changes, are numerous and varied. Of these particularly, a number are frequently found in an individual case.

Of late, much stress has been laid upon the importance of the kind of nourishment taken (alimentary constipation—Boas). This factor was recognized some time ago, but not at its true value. Westphalen in particular states positively that diet containing mainly albumin (meat and eggs) will eventuate in habitual constipation, while a vegetable diet, rich in cellulose, strongly favors normal evacuation. This is explained as follows : The meat diet is thoroughly converted, leaves little scoria, and forms less feces. With the vegetable diet, these conditions are reversed. Mechanical irritation, a palpably necessary factor for the excitation of peristaltic movements, is, therefore, less with animal than with vegetable diet. Add to this that with the former diet chemic irritation is less than with the latter. The fermentation products of vegetable

food rich in carbohydrates (carbonic, lactic, acetic, formic, and tartaric acids, hydrocarbonic and hydrosulphuric gases) excite peristaltic movements more strongly than the products of albumin decomposition (phenol, indol, skatol). According to Rubner, carrots appear in feces after four hours; brown bread in fourteen and white bread in seventeen hours. Animal foods, on the other hand, may not cause defecation until three or four days after ingestion, on account of the scant feces formed therefrom.

In a general way these figures may be regarded as decisive. But we must not lose sight of the fact that, aside from individual peculiarities, food is not the only important formative factor of feces. For instance, Westphalen states that in St. Petersburg, among the intelligent classes, lack of exercise and great mental effort must be added to the factor of animal foods. *Per contra*, the Russian peasant eats mainly vegetable food, does but little mental work, but a great amount of physical labor.

As before stated, regular and sufficient bodily activity exerts a decidedly favorable influence on peristalsis and evacuation of the bowels. A short period of inactivity will cause temporary constipation. If the inactivity is continued for a long period or indefinitely, habitual constipation may be the result. This applies equally to mental work, inasmuch as such is practically always sedentary.

In all cases as the preceding the development of habitual constipation is readily explained by the continued action of the causal factor.

Moreover, there is, I am convinced, a form of habitual constipation possessing all the characteristics of a primary and independent morbid condition. I regard as primary coprostasis all those cases in which the cause of the infrequent evacuation of the bowels is some perversion of the normal nervous mechanism of the peristalsis of the colon and the rectum. As yet it is uncertain whether the same condition may exist in the small intestine. At all events, in habitual constipation the stasis of fecal matter is always limited to the large intestine, or at least originates in this portion of the bowel. This abnormality of intestinal innervation may be congenital, hereditary (there are families the members of which for several generations suffer from constipation), or acquired. Many individuals have been constipated from their earliest childhood, while in others constipation begins at a definite time as the result of some special cause. Sometimes, for instance, an interruption of the previously regular action of the bowels by some chance circumstance may constitute the starting-point of habitual constipation. Unavoidable interruption of the regular daily evacuation of the bowels, as, for instance, during a long railroad journey, or conversely an attack of acute diarrhea, or some acute febrile disease may all lead to constipation. Depressing emotions occasionally give the first impulse to habitual constipation. Cases are on record in which prolonged and tiring marches produced habitual constipation. Possibly in the latter instance perspiration was so free that the contents of the intestine were to some extent deprived of water. A mistaken regard for appearances and the circumstances of social intercourse frequently serve as the exciting cause of

habitual constipation, especially in females, as, for instance, the efforts to suppress the desire of relieving the bowels which are made in boarding-schools and at social gatherings. These circumstances seem to be precisely analogous to those sometimes observed in the initial stages of quite a different malady—namely, protracted insomnia; a casual interruption of the normal alternations of rest and activity may give the first impulse to the development of a disturbance of the functional periodicity of the rectum, on the one hand, or of the brain, on the other. In these cases no other factors can be discovered which in general either lead to or aggravate sluggishness of the bowels. This form of habitual constipation may be seen in individuals who take active and regular physical exercise—they may, for instance, be farmers—are of strong constitution, eat ordinary food and are not exposed to mental shocks or emotions or in the least degree nervous, and are normal subjects with the one exception of constipation. That this form of habitual constipation cannot be explained on anatomic grounds is shown by autopsies on cases of this kind dying from some intercurrent disease; further, it cannot be shown that the condition depends on purely functional perversions of normal physiologic processes. The conclusion is, therefore, inevitable that the essential cause of this habitual constipation is a perversion of the normal function of the intestinal nervous system, the nature of which is quite unknown.

This explanation, which I made not to fit all cases, but still covers a great number, has met with criticism. The facts are simply these: An otherwise healthy individual, whose stools had always occurred regularly, becomes afflicted with habitual constipation from some merely incidental occurrence. Neither during life nor postmortem can the slightest anatomic change be observed. In such a case I have no doubt one is forced to the conclusion that as a result of the past and gone incidental occurrence, the nervous stimuli, which had theretofore occasioned a defecation once every twenty-four hours, have been so altered that they cause the act but once in several or many twenty-four-hour periods. I, at any rate, cannot at present see any other explanation. In hereditary constipation the abnormal peristalsis and evacuation are congenital; compare with it the congenital individualities in the frequency of the pulse. Whether the abnormal innervation applies to the entire colon or only to the act of defecation itself may at times appear uncertain, yet, as a rule, the colon as a whole seems to be involved.

Federn assumes a circumscribed atony of the intestines which may be combined with general intestinal atony, but is not at all identical with it. In this condition he claims some portion of the colon, perhaps only a very small part, is not capable of fully expelling its contents. He considers the condition to be the cause of a great variety of pathologic appearances. Improvement is said to follow faradization of the atonic part.

In another series of cases that must also be regarded as an independent form of habitual constipation the primary cause of the malady is not an impairment of the action of the intestinal nervous system,

but a weakening of the intestinal muscular fibers. Although the condition of the skeletal muscles in general has little bearing on peristalsis, the state of the unstriated muscles of the intestine itself is undoubtedly important. Erroneous ideas are prevalent in regard to the frequency with which the powers of these unstriated muscles are impaired. When a person becomes constipated after an attack of typhoid fever or some other exhausting and chronic disease, it is often assumed that this is due to weakness of the intestinal musculature, or, it may be, to an actual loss of muscle tissue. This conclusion, however, is based on only *a priori* reasoning. As a matter of fact, I have been able to prove by direct measurements [1] that no atrophy of the intestinal musculature occurs even in cases of advanced cachexia with great wasting of the striated skeletal musculature. It is, of course, possible that there may be functional impairment of the intestinal musculature, although it has never been proved, even though this tissue remains to all appearances anatomically intact. Conversely I have described cases in which the skeletal muscles were very strong, whereas the muscular coat of the large intestine was underdeveloped and below the normal average or even the normal minimum (instead of measuring from 0.5 to 1.0 mm., the fibers measured only from 0.12 to 0.25 mm.). This condition of the muscular structure of the large intestine occurs without any other lesion of the bowels, and is probably congenital. It may be assumed without hesitation that in these cases the peristaltic action of the large intestine was impaired, and, as a matter of fact, the patients under my observation in whom this condition was discovered suffered from habitual constipation. During life, of course, it is impossible to diagnose either congenital atrophy of the musculature of the large intestine or abnormalities of its innervation. It is obvious, moreover, that atrophy of the intestinal musculature, which occasionally follows other pathologic conditions, such as catarrh or overdistention, as well as that rare disease, fatty degeneration of the musculature of the intestine seen in consumptives and chronic alcoholics, will impair the vigor of the peristaltic movements. However, diseases of the intestinal musculature are responsible, on the whole, for only a small fraction of the cases of primary habitual constipation.

The foregoing has by no means exhausted the list of causes of habitual constipation. Great importance attaches to a large number of cases known as spastic constipation, in contradistinction to the previously described atonic variety. The diagnosis "primary constipation" is very frequently made in cases where, in reality, it is only a symptom. Dunin and Bouveret called attention to the fact that constipation is a frequent concomitant symptom of a general neurosis or of neurasthenia. This view had been expressed before they published their investigations, but it was Dunin and Bouveret who clearly established that in such cases constipation is a result of these neuroses, and not, as was erroneously believed, a cause of them. This view is undoubtedly correct, as may be shown both by the clinical analysis of the cases and by the effect of remedies, for

[1] *Loc. cit.*, p. 210.

treatment directed exclusively against the general neurotic state, without any special attention to the constipation, frequently leads to a cure of the latter symptom. Further reference will be made to this subject later on. Fleiner believes that this form of retardation of the bowel action is "spastic," and is due to the fact that certain portions of the intestine become spasmodically contracted, and in this way retain their contents. His view, therefore, is different from those just described, for in contradistinction to atonic constipation he speaks of "spastic" constipation. Dunin, in my opinion, goes too far in regarding the great majority of cases of habitual constipation as symptomatic and as dependent on a general neurosis—neurasthenia or hypochondriasis. Every medical man must, however, know of a large number of cases of chronic constipation in which there is not the least trace of any neurosis. Patients of this kind may never at the time have presented any nervous symptoms. Constipation, being in fact their only recognizable ailment, must be regarded as a primary disease and as complying with the definition of the group described above.

Formerly one of the chief causes of habitual constipation was believed to be some mechanical impediment to the onward progress of the intestinal contents, and the majority of cases of apparently primary independent constipation were thus explained. Special stress was laid on peritoneal adhesions around the intestine, and it was thought that these adhesions interfered with the free movement of the bowel. Virchow is often quoted as the authority for this mechanical theory, but in his original essay on this subject he expresses himself as follows : "The question arises, can fecal accumulation give rise to this form of local peritonitis ? It appears to me that this question must be answered in the affirmative." In the course of his remarks on this subject Virchow proceeds to show how fecal accumulation may lead to inflammation of the serosa ; in other words, he considers the accumulation of fecal matter to be the primary factor, and the peritonitis secondary. In reality anchoring of the intestine by peritoneal adhesions is relatively rare as compared to the extraordinary frequency of habitual constipation. This fact alone would militate against any causal connection between the two conditions. There is, however, another factor : there can be no doubt that fixation of the intestine, particularly if it leads to partial kinking of the bowel, may obstruct the passage of the intestinal contents ; but fixation of the intestine is more likely to produce very definite narrowing or complete occlusion of the lumen of the intestine than a comparatively harmless condition, such as simple chronic constipation.

Pennington has recently attributed importance to the Houston's folds in the rectum in the etiology of habitual constipation. Ebstein favors this view.

A few cases of congenital hypertrophic dilatation of the colon have been observed in chronic constipation of children. More will be said of this later under Intestinal Anomalies.

Congenital dislocations of the intestines, particularly such as are

caused by anomalies of the mesentery (excessive or insufficient length), have often been regarded as etiologic factors in habitual constipation. Earlier authors (Vötsch and others) laid great stress upon this point. Leichtenstern has acknowledged its correctness in some cases, but his splendid work has considerably limited the scope of this factor. He has shown that decided flexions and abnormal positions of the intestines need not necessarily interfere with the passage of the contents, provided only that there are no adhesions causing kinks in the canal. Frequently conditions are reversed, inasmuch as the abnormal position of the intestine is the result of excessive retention of feces following primary sluggish peristalsis (*vide supra*). It is evident that the causal relations between intestinal displacements and habitual constipation have been constructed on largely *a priori* reasoning. Dunin is right when he says that no one has yet demonstrated such connection by a careful analysis of clinical material and adequate anatomic proofs. The frequent constipation met in enteroptosis is at times regarded as atonic, oftener as spastic, as before described. Nervousness is well known to be an almost regularly occurring symptom in this disease (see p. 339).

Finally, under certain circumstances, the striated skeletal musculature may be a factor in habitual constipation. Of course, the general musculature of the body is wholly unimportant, as the weakest individual may have regular defecations and the strongest may suffer from obstinate constipation. But we must not underestimate the coöperation of the muscles of the abdominal wall, as they aid the consummation of peristalsis—the expression of feces from the rectum. If the action of the abdominal muscles is too feeble, evacuation may be only partial and mechanical obstruction occurs. This is particularly so in emaciated individuals and in pregnant and multiparous women. A familial type is evidenced at times. Thus, Ebstein reports the case of three brothers who had been afflicted since earliest childhood with diastasis of the abdominal muscles and considerable constipation. According to Pincus, women who have suffered traumatism of the musculature of the pelvic floor (levator ani) during child-birth experience increasing difficulty in evacuation and ultimate habitual constipation.

SYMPTOMATOLOGY.

A veritable army of symptoms, disturbances, and sequelæ have been attributed to constipation. Popular parlance and methods of treatment bear this out in a very conclusive manner. Since erroneous and exaggerated views on this point are so generally accepted by the laity, and to some extent by many medical men, it is necessary to consider carefully and in detail the question of paramount practical importance—namely, What are the consequences of habitual constipation? The following paragraphs will be exclusively devoted to elucidation of this subject, and will not treat of the symptomatic forms of constipation.

In the first place, it must be remembered that even exaggerated degrees of sluggishness of the bowels need not necessarily produce any

disturbances. Aside from freaks (see a collection of curious cases in Leichtenstern's book—he reports, for instance, cases of constipation in individuals with an extraordinarily long colon who evacuated the bowel contents only once every few weeks), subjects are frequently met with who evacuate the bowel contents only once in every three or every five days and still feel perfectly well and experience no inconvenience whatever. This fact alone proves conclusively that habitual constipation is not always so serious a matter as it is so generally believed to be.

It is true that, as a rule, some disturbances appear in prolonged constipation, but in the majority of cases these disturbances are insignificant, are seldom of importance, and only in exceptional cases dangerous. The symptoms are, as a rule, referred to the alimentary canal or are local in character. It is important to establish this fact before discussing the symptomatology of chronic constipation, since these symptoms are the only essential clinical features of habitual constipation. In the first place, therefore, the derangements of health which are really and undoubtedly due to habitual constipation will be considered, and then the symptoms which have been erroneously attributed to it will be briefly reviewed.

Every practitioner knows that certain mild functional disorders may occur in primary independent sluggishness of the bowels. It is quite clear that these disturbances are due to retention of feces, for as soon as the contents of the bowel are evacuated, these functional disorders disappear. They chiefly consist in a feeling of fulness, tension, and discomfort in the abdomen ; at times these sensations are referred directly to the stomach ; at the same time there is well-marked loss of appetite, and occasionally there is belching or a disagreeable taste in the mouth.

If defecation is habitually insufficient, and fecal matter is retained for a long time, the stools become very dry, so that they form small, roundish balls (scybala) which collect in the pouches of the colon. Occasionally they may form large lumpy masses which accumulate in the ampulla of the rectum.

Fleiner was the first to observe that the form assumed by the feces served to an extent to indicate the etiology of the constipation. Boas, Rosenheim, and Westphalen observed it later, and my own experience also coincides with theirs. The small, spheric, fecal masses (like sheep-dung) occur in the atonic and spastic constipations. He also believed that rod-shaped masses of fecal matter of small caliber—about as thick as a lead-pencil or the little finger—are particularly characteristic of the latter form of constipation. These pencil-shaped dejecta may be long or short. This form of stool will be discussed in the section on Enterostenosis, but it should be stated here that they may originate independently of any general neurosis or of any local spasm of the intestine ; as a matter of fact, they occur during starvation as soon as the intestine becomes contracted ; they are also seen in some forms of inanition—for instance, in carcinomatous cachexia—in which the intestine also contracts.

Westphalen has called attention to some other clinical methods of

differentiating atonic and spastic constipations. In the atonic form the patients underestimate the relative quantity of feces to ingesta, and in the true spastic form the reverse obtains. In the latter the subjects have a feeling after defecation, frequently accomplished only after great effort, that there is still feces in the rectum, though this is really not the case. In the atonic cases the patients after defecation experience a sensation of satisfaction and relief.

If constipation is not too persistent or too obstinate, the bowels may act spontaneously after a few days' interval, vigorous straining efforts being a usual accompaniment of the process. In these cases the hard fecal masses are covered by a thin layer of flaky mucus, a circumstance which may lead to the erroneous diagnosis of " chronic catarrh of the intestine with constipation," whereas in reality the case was the very reverse, the increased secretion of mucus being due to the irritation exerted on the mucous membrane of the colon by the accumulation of fecal matter. In other cases the bowels become loose after a few days. This occurs when the irritation of the hard fecal masses is so marked as to produce hyperemia of the mucosa with secretion of fluid and consequent liquefaction of the intestinal contents ; in addition there is usually an abnormal development of flatus, and, lastly, increased peristalsis. In the third place, spontaneous defecation may become quite impossible, so that an action of the bowels can only be obtained with the aid of purgatives or enemas. In extreme cases it is necessary to remove the fecal masses from the rectum with the fingers.

Arrest of the onward movement of the feces may have more important results than the production of transitory diarrhea or a slight increase in the secretion of mucus, for it may be the cause of definite structural lesions, such as catarrh of the intestine, ulceration of the intestinal wall, and finally local peritonitis due to irritation. This will be referred to below when the various morbid conditions are under consideration.

When exceptionally large masses of fecal matter accumulate, a group of symptoms called fecal colic may appear. The patients are seized with violent colicky pains that may be so severe as to produce syncope ; at the same time the abdomen sometimes becomes distended with gas, the passing of which gives temporary and comparative relief. The symptoms never completely disappear until the accumulated feces are passed. A rapid diagnosis in these cases is frequently a matter of difficulty. The history of prolonged constipation is of importance in this respect, but I have repeatedly seen attacks of fecal colic occur even though the bowels acted regularly every day ; the detection of fecal accumulations on palpation of the abdomen is of greater diagnostic significance. The colicky pain in these cases is produced by the energetic tonic contractions of the intestine above the point where the fecal masses are lodged.

In cases of obstinate and persistent constipation a very alarming symptom-complex (with pronounced collapse and vomiting) occasionally develops which resembles those of intestinal obstruction. This occurs when internal remedies have been taken which, while not powerful

enough to open the bowels, merely produced vigorous intestinal contractions and violent colic. The symptoms in these cases disappear as soon as the rectum is emptied of fecal masses either by irrigation or by the finger. Occasionally very prolonged fecal accumulation may actually lead to paralysis of the colon, the result being that the patient dies with symptoms of complete intestinal obstruction. This condition is most frequently seen in the insane and in old and very weak persons, particularly in those whose lives are sedentary; it is also seen in other cases in which the feces are retained too long for some reason, such as painful fissure of the anus or hemorrhoids.

Habitual constipation occasionally leads to the formation of so-called fecal tumors—namely, accumulations of inspissated, more or less hardened masses of feces (coproliths). These may occasionally attain an astonishing size. They always originate in the large intestine, from the ampulla of the rectum up to the ileocecal valve. Sometimes several of them are found in the same individual.

[Gersuny [1] lays stress on a new sign of fecal tumor, which consists in a feeling of separation of the wall of the bowel from the tumor when, on palpation, the pressure is somewhat relaxed.—ED.]

These fecal tumors frequently render the diagnosis of abdominal disease difficult, and many cases are on record in which these tumors have led to remarkable errors in diagnosis. They have been taken for peritonitic exudates or for neoplasms, particularly carcinomata and sarcomata of different organs (the liver, the stomach, the spleen, the kidneys, the ovaries, the uterus, and, especially, the omental, mesenteric, or retroperitoneal glands, and the bones of the pelvis). It is by no means always easy to diagnose fecal tumors; the position, the form, the resistance, the mobility, the tenderness, even the general symptoms, or the functional disturbances connected with the intestine vary greatly in individual cases, so that mistakes can easily be made. Fecal tumors, for instance, situated in the transverse colon may drag this portion of the bowel down as far as the symphysis pubis. They may be nodular, beaded, elongated, angular, or may be shaped like bricks. They may be either fairly soft and impressionable (in a positive case, of course, of great diagnostic importance), or of stony hardness. Their range of movement may be so exceedingly small that they are apparently fixed. They may set up secondary inflammation of the intestine and local peritonitis, and as a result become very tender on pressure. They may cause digestive disturbances leading to impairment of the general nutrition of the patient. The most misleading feature, however, in many cases is the fact that the patient does not appear to be constipated, but has a regular evacuation of the bowels every day. The amount of feces passed is, however, less than it ought to be, for the first feces accumulate in the pouches of the large intestine or in some diverticulum, leaving a central passage that remains free. In many instances a diagnosis can be made only after repeated examinations of the patient, and especially by observing whether treatment directed

[1] Gersuny, *Wien. klin. Wochenschr.*, Oct. 1, 1896.

toward unloading the bowels causes any change in the character of the tumors in question. Gersuny has recently called attention to a palpation phenomenon ("adhesion symptom") at times to be obtained in fecal tumors. When the abdominal wall over a prominent part of the tumor is gradually deeply depressed with the finger-tips, the pressure very gradually diminished, and the fingers slowly withdrawn, we can feel the mucous coat of the intestine loosening itself from the tough feces forming the tumor. [Examination under an anesthetic often gives valuable information.—ED.]

Fecal tumors are a source of danger in many ways. The production of stercoral ulcers and local peritonitis have already been mentioned. Even gangrene of the intestinal wall and perforation followed by diffuse peritonitis may result. Another dangerous sequel is partial blocking of the lumen of the intestine, which may finally lead to complete obstruction. Intestinal obstruction is also occasionally due to the fact that an overloaded portion of the colon sinks down out of its place or becomes twisted on itself (especially at the sigmoid flexure). In this way kinking of the gut or compression of the intestine is produced (compare Fecal Colic above).

Finally, another local sequel of fecal accumulation—*i. e.*, the development of hemorrhoids—must be mentioned. As, however, the subject of hemorrhoids will be discussed in a separate section, a mere allusion to it will be sufficient at present.

While the above-mentioned morbid conditions may undoubtedly follow chronic constipation, the case is different with regard to a great many other so-called sequelæ, which it is or has been usual to regard as connected with constipation. Even now there is a great deal of controversy as to many of these secondary symptoms. Only a few years ago (1874) Voetsch published a monograph in which he claimed that a number of the most serious diseases (presenting the clinical picture of typhoid fever, pyemia, cholera, and brain lesions) were the consequence of coprostasis, and as late as 1890 Feyat attributed the majority of mental diseases to constipation. In 1898 von Gölder stated that there are cases of coprostasis in whose wake an acute psychosis occurs, running the course of an acute delirium of eight to fourteen days' duration, in which time death may take place, with concurrent weakness of the heart. Most medical men at the present time have abandoned views of this character, and the days in which the motto "*Qui bene purgat bene sanat*" was an important therapeutic rule are happily over. Among the laity, however, speculation as to the influence of "confined bowels" on the general health is even now by no means rare. Even metritis, compression of the iliac and femoral veins, with thrombosis and edema from stasis, compression of the nerves of the pelvis producing neuralgia and paralysis, and many other conditions that are considered etiologically related to pressure symptoms within the area of the lower bowel are now rarely attributed to constipation. It is really almost unnecessary to enter into a detailed discussion of all these matters. [In

a paper on 48 cases of postoperative crural thrombosis Schenck [1] concludes that constipation and the use of enemas play a doubtful part in the causation of this condition.—ED.]

We cannot omit some mention of the so-called "fecal fever," the occurrence of which, in both children and adults, is attested by such reputable authors as Lauder-Brunton, Edlefsen, Ebstein, Périer, Ewald, and others. This fever lasts usually only two or three days. Its connection with constipation is proved, by those who believe in it, by the prompt dissipation of the fever by an artificially procured evacuation of the bowels. It is undoubted that constipation in countless instances causes no rise in temperature. Hence, it cannot be considered a direct cause of fever, but only the very remotest factor. We must, therefore, seek another, more immediate, factor, and probably this is to be found in abnormal putrefaction of the intestinal contents, leading to autointoxication (Mathieu, Edlefsen). In isolated cases, we believe in nervous individuals, cardiac symptoms appear, such as a feeling of anxiety, palpitation, at times, too, an accelerated pulse and arhythmia, all of which disappear after evacuation of the bowels.

The belief has long been prevalent and is widely accepted even now, that certain disorders of the central nervous system are causally related to habitual constipation. This applies not merely to some of the milder nervous symptoms, such as vertigo, stupor, etc., but also to certain forms of hypochondriasis and even pronounced psychoses. As a matter of fact, there is a so-called "copro-psycosis." Although the ridiculous exaggerations of many authors hardly deserve serious consideration, it must still be remembered that some psychic alterations may undoubtedly be of "abdominal origin." The literature contains numerous animated discussions as to the connection between "abdominal disturbances," particularly dyspepsia, and constipation and hypochondriasis. Virchow, some forty years ago, published a very lucid critical essay on this subject, and it must be confessed that even after the expiration of all these years we hold the same opinion that he defended. At that time a number of authors, among whom I may mention Romberg, emphatically declared that hypochondriasis might be directly caused by abdominal disturbances. At the present day, on the other hand, Dunin and others hold a contrary opinion, and believe that hypochondriasis is not the result of constipation, but its cause, and that even if this is not always the case, hypochondriasis and constipation are certainly two concomitant symptoms of the same disease—namely, of neurasthenia. This view is certainly strengthened by a phenomenon common in subjects who are prone to attacks of periodic melancholia. In these individuals it often happens that the bowels act quite regularly in the intervals between the attacks, and that constipation comes on as soon as melancholic symptoms appear.

Attention has already been called to the fact that habitual constipation occasionally leads to mild cerebral symptoms (in addition to well-known gastro-intestinal symptoms) in subjects who are perfectly healthy

[1] Schenck, *New York Med. Jour.*, vol. lxxvi., p. 401.

and not in the least neurotic. This is particularly prone to occur when constipation has lasted for several days. The chief cerebral symptoms complained of are a feeling of pressure, heaviness, and stupor in the head, occasionally, though not often, violent headache, vertigo, and sometimes a feeling of heat in the head. These head symptoms are unquestionably due to constipation, for when the bowels act they disappear; the patient may continue to feel perfectly well for a few days, but the same symptoms reappear as soon as constipation has returned and lasted for some days.

It is impossible to determine with absolute certainty the cause of these mild cerebral symptoms, and possibly several factors are concerned in their production. The older authors were inclined to attribute them exclusively to disturbances of respiration and circulation, which, in their turn, were thought to be due to upward displacement of the diaphragm by the distended intestine. They were chiefly led to this view by the fact that serious respiratory disturbances were occasionally noticed in these cases. Later, when the laws of reflex irritation became known, the cerebral symptoms were attributed to reflex stimulation of the cerebral blood-vessels. No direct proof of this assumption, however, has so far been forthcoming, nor has any definite statement ever been made in regard to the exact path that the reflex irritation is supposed to travel, nor as to the nerve-fibers and nerve-endings that are particularly irritated. Leube recently reported a case of intestinal vertigo in which the vertigo was produced by pressure on the lowest portion of the intestine, whether the pressure was exerted by fecal masses, accumulated gas, or by the palpating finger in the rectum. In this way the intestinal wall was irritated, and Leube believes that pressure on the hemorrhoidal plexus of the sympathetic nerve produced the feeling of vertigo. Senator has advanced another theory. He calls attention to the fact that in some digestive disorders a number of well-known gases, such as sulphureted hydrogen, may be absorbed and cause mild symptoms of intoxication. I believe that in some cases this may be the correct explanation, but it does not apply universally. I have advanced the theory that during constipation certain ptomains may possibly be absorbed. Dunin objects to this view on the ground that an excessive formation of ptomains could only occur when the retained feces were liquid. He supports his objection by referring to Bouchard's observation that the toxic fecal ptomains are absorbed and excreted in the urine chiefly during diarrhea. Another possible cause of the mild cerebral symptoms that have been described are certain aromatic products of intestinal putrefaction, for we know that these are formed in large numbers in constipation (see below). Von Pfungen has demonstrated an increase in bacterial decomposition of derived albumin in cases of simple chronic constipation similar to that proved to exist in mechanical occlusion of the rectum and large intestine. The last was demonstrated, among others, by Kast and Boas. Although, therefore, we are unable to formulate an exact explanation of the occurrence of

cerebral symptoms in constipation, the fact must be recognized that such symptoms may appear in this condition.

What is the true connection between hypochondriasis and constipation? A great deal has been written on the relation between the two conditions. My own view, based on personal experience, is as follows: Habitual constipation *per se* and alone causes no psychic changes in individuals otherwise normal. It can never be held responsible for hypochondriasis and still less for any form of psychosis. At the same time the fact remains that symptoms of psychic depression occasionally develop in habitual constipation. In all these cases, however, a second factor must be considered. Psychic depression occurs only in individuals with a neuropathic disposition, and it is only in these individuals that perversions of intestinal function (just as mild perversions of other functions) lead to the development of nervous disturbance. Chronic constipation alone cannot produce hypochondriasis in a healthy person, though it may in a neuropathic subject. Virchow, in discussing the views of Cullen, Brachet, and others, expresses himself as follows in regard to this matter : " Disturbances of abdominal organs produce symptoms only in subjects suffering with a certain irritability (lack of resisting power, predisposition) of the nervous system. These symptoms assume the character either of exaltation in the sensory or of depression in the motor sphere." Although, therefore, the popular belief as to the injurious effect of constipation on the brain is exaggerated and untenable, it is, nevertheless, justified within the limitations imposed above.

Some observers report neuralgias resulting from chronic constipation, not only of the ischiatic and crural regions, but even of the trigeminus (Gussenbauer).

A number of authors, particularly English writers, have recently put forward the view that habitual constipation produces the symptom-complex of chlorosis. [It can hardly be said that this view of the late Sir Andrew Clark has any supporters now.—Ed.] They assume that possibly the absorption of ptomains is the primary cause, and that these bodies impair nutrition and interfere with the regeneration of the blood. They attempt to prove this assertion by calling attention to the fact that such cases of chlorosis are usually cured by the administration of laxative remedies. Personally I have never been so fortunate as to obtain this result, and I do not believe that it has ever been proved that constipation can produce this disease.

TREATMENT.

It is impossible to describe all the various laxatives and other means that have been employed for many years with more or less success in the treatment of the symptomatic forms of constipation. This description will be confined to the means and remedies that are indicated in the treatment of habitual constipation alone. Sufferers from this condition are frequently a source of great annoyance, trouble, and care

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to their medical attendant ; they go from place to place, from one medical man to another, seeking relief, and their condition seems to remain the same for years, notwithstanding all the remedies that are employed. This obstinacy of the symptoms may be due to three causes : In the first place, unsuitable remedies or methods of treatment may have been employed ; in the second place, appropriate and suitable methods of cure may have been imperfectly carried out ; in the third place, some anatomic-mechanical condition may be present that cannot be relieved.

As a rule, patients of this character are treated in a routine manner, and the measures devised are generally devoid of value, and occasionally directly harmful. The routine prescription of laxative remedies and the application of other measures that are intended to cause evacuation of the bowel contents rarely lead to the desired result. In order really to benefit a case of habitual constipation the peculiarities of each individual case must be carefully analyzed and a plan of treatment devised on the basis of the conditions found in each patient.

In the first place, it must be remembered that evacuation of the bowel contents every second or third day does not necessarily mean that the patient must be treated for constipation, particularly if this evacuation occurs spontaneously and a sufficient quantity of feces is passed. If the subject feels perfectly well, the case belongs to the category of physiologically retarded intestinal peristalsis described above. The chief duty of the medical man in cases of this kind is to warn the patient against all treatment.

If the symptoms in any given case are so distressing or so serious that therapeutic measures are indicated, an attempt should be made at once to determine what the etiology of the chronic constipation is. Above, in group 3, we have described the different possible etiologic factors that may lead to the disease. In many cases it will be very difficult, or even impossible, to decide whether habitual constipation is originally due to some abnormal nervous perversion of the peristalsis of the colon or to some congenital weakness of the intestinal musculature, or finally to displacement or abnormality in the course of the intestine. Frequently it will be possible to ascertain that the constipation is acquired, and to determine what particular features of the mode of life and of diet have caused it. Even if the exact underlying cause is not determined in every case, careful investigation will prevent gross errors ; laxatives should not be given where physical methods of treatment are necessary, and a neurasthenic subject with secondary constipation should not be sent to Marienbad or Kissingen.

Therapeutic measures in habitual constipation may be classified as follows : (1) Dietetic ; (2) physical ; (3) medicinal. Choice of these must be governed by the etiology and clinical presentations of individual cases.

1. Diet.—A patient with true habitual constipation, without any complications, can, as a rule, eat and drink the majority of foods and beverages. Nevertheless it will be best to countermand some of them, while others are well suited to promote intestinal activity. As a gen-

eral rule, vegetables will form a greater part of the diet than meat and eggs. The reasons for this have already been given (p. 101). Various fruits, chiefly the juicy kinds, as plums, peaches, pears, apples, oranges, mandarins, gooseberries, currants, strawberries, raspberries, and grapes, are to be taken in plenty. Whortleberries must be avoided. Vegetables rich in cellulose, as lettuce and cucumbers, and those of the cabbage and tuber varieties, are generally indicated, except that the last two must be omitted in cases with a decided tendency toward flatulence. Honey, syrups, milk-sugar, coarse brown breads that readily acidulate (rye, Graham, bran, and ammunition breads, pumpernickel), are all useful. In the same category may be placed very fatty and highly seasoned foods. Of the beverages, red wines, tea, chocolate, and cocoa must be avoided. The following may be taken, as their effect is somewhat favorable to intestinal action : White wines, champagne, cider, young beer, lemonade, buttermilk, sour milk, carbonized waters, and cold water.

Experience has shown that any single article of food may have the most varied results on different subjects. The best-known example is milk, which in some cases is constipating, in others laxative, and in others without appreciable effect either way. We must, therefore, be guided in such matters by the experience and observations of our patients, and from these decide our dietaries. Some authors have compiled diet-lists, among others Penzoldt, Rosenheim, and Wegele. Naturally, we must always take into consideration the general health of the patient (obesity, anemia, etc.), and must pay due attention to any physiologic condition.

In very simple cases the dietetic treatment alone often suffices. In fact, the simplest measures at times effect cures—as, for instance, a glass of cold water in the morning on an empty stomach ; a few dried plums, a teaspoonful of milk-sugar, and others equally homely.

It is true, on the other hand, that some laxative remedy or an enema must occasionally be administered when these patients are at home, but this need not necessarily be done every day ; the real and important object in treatment is to act on the intestine in such a way that it gradually acquires the power of performing its function in a normal manner. This is the most important feature in the treatment of habitual constipation.

2. Physical Treatment.—One of the most important measures is massage of the abdomen. In order to do good, massage should be given by an expert and should be applied for a long time. Both doctors and patients frequently err in not continuing the treatment for a sufficiently long period of time ; we can really expect nothing, or at best very little, from a three or four weeks' course of massage ; the only way in which we can expect to benefit our patients is to employ massage for a long time. In many cases the treatment must be persisted in for many months ; this will be followed by the gratifying result of permanent improvement of the patient and regular, spontaneous evacuation of the bowels. At the commencement of this treatment, and before any direct effects from massage can be expected, a laxative must occasionally be given symptomatically. The massage treatment should be entrusted only to expert

hands. The chief feature to be observed is careful manipulation of the large intestine. The object is to stimulate the peristaltic action of the colon by short tapping and punching movements, by kneading and rubbing—that is, by purely mechanical irritation. Massage in habitual constipation should be directed only to the stimulation of the peristalsis of the bowel and not to the direct propulsion of the intestinal contents, so that it is immaterial whether these movements are performed in an ascending or a descending direction. It need hardly be mentioned that the rest of the abdomen must, of course, also be massaged, and that the manipulations should not be limited to the colon.

A number of other measures must be regarded as valuable adjuvants to massage, among which faradization of the abdomen may be specially mentioned. The application of the faradic current in constipation is intended to cause short contractions of the colon ; for this reason no attempt to produce long tetanic contractions should be made, but the thoroughly moistened electrode should be pressed deep down into the abdomen and moved rapidly from place to place. Leubuscher has also recommended galvanic electricity, which is applied by introducing one electrode into the rectum, while the other is placed upon the abdomen. Electric treatment is not so effective as massage, nor is it so generally applicable. Older clinicians attach special importance to increasing the contractions of the abdominal muscles. The contraction of these muscles certainly aids the individual act of defecation, but it exerts no influence on the peristaltic movements of the intestine.

Another very valuable method of treating chronic constipation is by physical exercises, gymnastics, and so-called Swedish movements and indoor exercises.[1] When the patients happen to live in a city in which special institutions exist for carrying out these gymnastics, they should be recommended to attend these gymnasiums ; if not, they should be advised to purchase hand-books containing all the necessary directions. In order to be successful this form of treatment must be carried out methodically and for many months ; if this is done, permanent results are occasionally obtained. Henoch states that laxatives should still be given to these patients when starting the treatment. The masseurs usually advise against this, and claim to be able to accomplish everything by their manipulations. As a matter of fact, however, the patients frequently suffer greatly from the consequences of obstinate constipation if the evacuation of the bowels is not promoted by drugs in the first instance. Other forms of physical exercise, as walking, long marches, and walking tours, horse-riding, rowing, lawn-tennis, bicycling, etc., are merely adjuvants to other measures, and probably chiefly act by making the patient lead a more regular life. I have never seen a case of habitual constipation improve even though walking and horse-riding were energetically practised for a long time.

[1] Occasionally most surprising conditions are met with. I remember the case of an anemic and very delicate lady who told me that the sluggishness of the bowels disappeared spontaneously and without any manipulation or treatment immediately after she remained in bed permanently and began to take iron on account of the anemia she was suffering from.

Massage, faradization of the abdomen, and gymnastics are, in my experience, the most effective remedies for the form of habitual constipation which I have called the primary independent form. Some hydropathic measures, however, are also of considerable value, either alone or in combination with the procedures mentioned above. Hydrotherapeutic treatment, however, should be carried out only in a special institution. The most effective procedures are frictions, cold douching or alternating warm and cold fan and Scotch douches of the abdomen, short, cold sitz-baths, and the wet binder (Neptune's girdle) applied during the night. Boas recommends the ether douche in obstinate cases. In this about 100 c.c. of pure ethyl ether are played upon the abdomen in a fine stream. The procedure lasts about five minutes, and is repeated once or twice daily. Richardson's apparatus is used. By far the most favorable results are obtained by the combination of massage, faradization, gymnastics, and hydrotherapy.

Trousseau, many years ago, emphasized the fact that one of the most important features of the cure is regularity. He instructed his patients to attempt an evacuation of the bowels daily at exactly the same hour, and to do this regularly, even though the attempt was at first unsuccessful. I am very much in favor of this plan and can indorse Trousseau's advice, for I have seen many cases in which this methodic education of the intestinal function was beneficial.

3. Medicinal Treatment.—While I advise limiting the administration of laxatives proper as much as possible in cases of genuine habitual constipation, I realize that it is impossible to do without them altogether. If they are given at all, it is important that the proper remedy should be selected. It is impossible to discuss the whole class of purgatives here, and the remarks will be limited to those remedies that should be selected in cases in which drugs have to be administered for a long time in order to combat chronic constipation. The leading remedy of this group is aloes; this drug is one of the oldest, and at the same time one of the most effective, remedies that we possess, and is the chief ingredient of a large number of preparations in the market. One of the chief advantages of aloes is that its effect is not lost even when the drug is administered for a long time. In addition, the feces evacuated retain their normal consistence, and defecation is painless. As a rule, the evacuation of the bowels occurs from eight to twelve hours after the administration of the remedy. In the past medical men have opposed the use of aloes, on the ground that it disposes to hemorrhoidal and uterine hemorrhages. Aloes in some of the more common prescriptions is often combined with rhubarb, and in some persons the latter drug alone is very effective.

[Sir T. Lauder Brunton [1] gives the following formula of a pill, taken before dinner with success for forty years without losing its effect :

Pil. colocynthi comp. . . . . gr. j
Pil. rhei comp.   . . . . gr. j
Ext. hyoscyam.   . . . . gr. ss.—Ed.]

[1] T. Lauder Brunton, *Allbutt's System of Medicine*, vol. iii., p. 708.

Another useful remedy is jalap; the chief advantage of this drug is that it does not produce a tendency to constipation. Among more recent preparations, two have become very popular, and justifiably so, particularly in those cases where it is desired to treat habitual constipation symptomatically for a time; these two remedies are podophyllin and the extract of cascara sagrada. I am particularly impressed with the value of the former remedy, and am in the habit of frequently prescribing it in the following form:

> Podophyllini 0.5
> Ext. aloes
> Ext. rhei . . . . āā 3.0
> Ext. taraxaci . . . . q. s. ad pilul. No. xl.
> Sig.—To be taken in the evening before going to sleep, 1 to 4 pills, according to individual requirements.

[Purgatin is a new and synthetically prepared substance, allied to the active principle of aloes, rhubarb, and cascara sagrada. It is anthrapurpurin diacetate. It is tasteless, and is a slow but sure purgative, with no serious after-effects. It colors the urine red, and in large quantities irritates the kidneys, and, therefore, should not be given in renal disease. The dose is from 15 to 30 grains (C. R. Marshall[1]). Purgin or phenolphthalein, another synthetic purgative, has been investigated by Vamossy,[2] Unterberg,[3] and Tunnicliffe.[4] It is a useful and safe purgative for children in $\frac{3}{4}$ to $1\frac{1}{2}$ grain, and in adults $1\frac{1}{2}$ to 15 grain doses. It has no disagreeable secondary results and does not irritate the kidneys.—Ed.]

In many persons a good evacuation can be produced by a sauce made of tamarinds, by manna, or by a dessertspoonful of the compound powder of licorice. Another prescription that has been recommended is a mixture of the extract of calabar bean and glycerin (0.05 to 10.0), six drops to be given every three hours for atony of the intestine and its sequelæ. This drug, however, is not universally employed in practice. [The use of its active principle, eserin, has given good results in obstinate constipation (F. A. Packard[5]); and the salicylate of physostigmin has been employed subcutaneously (Vogel[6]) with this same object. —Ed.] The exhibition of belladonna at times gives good results in the spastic forms of constipation. Trousseau recommended this remedy warmly.

Calomel and castor oil, on the one hand, and so-called drastic purgatives, such as senna, colocynth, rhamnus, on the other, should be used only in habitual constipation, when the obstinate retention of the stools must be overcome at once; in other words, they are to be given only occasionally. The same applies to the saline cathartics. The salines, it is true, are frequently given in a special form—namely, in the waters that are taken in certain spas, such as Marienbad, Carlsbad, Homburg,

[1] C. R. Marshall, *Scottish Med. and Surg. Jour.*, May, 1902, p. 402.
[2] Vamossy, *Therapie der Gegenwart*, May, 1902.   [3] Unterberg, *ibid.*
[4] Tunnicliffe, *British Med. Jour.*, 1902, vol. ii., p. 1224.
[5] F. A. Packard, *Trans. Coll. of Phys.*, vol. xxiv., p. 120, Philadelphia, 1902.
[6] Vogel, *Annals of Surgery*, December, 1902.

Kissingen, and many others. An erroneous impression exists with regard to the effect of these waters, whether taken at home or at the spa. It is true that they produce an evacuation of the bowels, but they act merely like any other ordinary laxative; the water, if a certain number of glasses are taken every morning, produces an evacuation of the bowels for that day, but its effect is not felt after that—in other words, the administration of these waters is merely symptomatic and in no way leads to a cure of the disease nor in any way influences the primary factors underlying habitual constipation. It is possible that in a few isolated cases in which catarrh of the intestine (in an anatomic sense) is the primary cause of constipation, a course of these saline waters may lead to permanent improvement—merely, however, by curing the catarrh. A course, for instance, in Carlsbad may do this. While these waters, therefore, do not cure the majority of these cases, there is no doubt that patients suffering from habitual constipation feel comparatively well while stopping at one of these watering-places. This is easily explained by a number of reasons, and many sufferers from constipation frequent certain watering-places regularly every year. While there is no doubt that many of these patients feel extremely well while they are actually taking the course of waters, it is just as certain that the old condition returns as soon as they stop taking the waters. "In X. Y. I enjoyed a regular daily evacuation of the bowels. Four days ago I left the place and now I have not had a stool for two days." This is a story we frequently hear. All the factors enumerated in this paragraph must be remembered in attempting to estimate the true effect of a course of mineral waters in genuine and habitual constipation.

Rectal injections or enemas are an important method of the symptomatic treatment of constipation. It matters little how the fluid is injected—whether we employ an ordinary syringe, a "Clyso pump," or an irrigator. The exact quantity of fluid to be injected and its composition must be determined in each individual case—that is, we must see whether cold, cool, or lukewarm water or a mixture of water and soapsuds, vinegar, common salt, or castor oil is most effective. Occasionally, particularly in the spastic form, injections of oil alone produce excellent results. I usually employ from 150 to 500 gm. of the best olive or poppy oil of the first expression. Lately glycerin has become very popular, administered in quantities of from 2 to 3 gm. with a small syringe, or inserted into the rectum in the form of suppositories. Glycerin stimulates peristalsis by withdrawing water from the intestinal wall.

The different kinds of injections all have the advantage of acting purely locally on the large intestine and of stimulating the peristalsis of this portion of the bowel without affecting the stomach in any way. The same objections, however, obtain in the case of this method of treatment as in the case of all the laxatives—namely, that sooner or later it loses its power. It is advisable, therefore, in cases in which the evacuation of the bowels must be artificially stimulated for a prolonged

period of time and cannot be avoided, to alternate between the different remedies and methods at our disposal.

[Washing out the stomach has been recommended by O. Ziemssen [1] as a means of stimulating peristaltic action of the bowel. Brunton speaks of it in conjunction with enemas as of use in removing fecal masses.—ED.]

Dunin has recently formulated the rule that habitual constipation is merely a symptom of neurasthenia. Acting on this view, he deprecates all measures and remedies intended to promote defecation. He argues that remedies of this kind should be employed only in exceptional cases, and then only symptomatically. He advocates treatment of the neurasthenic condition alone, and claims that cure of neurasthenia will lead to the disappearance of the chief symptoms of this condition—namely, habitual constipation. Dunin's view is extreme, is too much of a generalization in its original form, and is incorrect; but at the same time it certainly does apply to a number of cases. Neurasthenics, therefore, suffering from chronic constipation should be treated on the following plan :

The use of laxatives and of measures intended to promote the evacuation of the bowels should be avoided. Only in cases of very prolonged constipation should any kind of laxative be given symptomatically. Kussmaul and Feinberg specially recommend oil enemas for these cases. They believe that massage is useless and even harmful in "spastic" constipation. The attention of the patient himself should be diverted as much as possible from his digestive organs. Psychic treatment should be carried out in the same way as in any other case of neurasthenia. The patient should be induced to give up his business or profession and be advised to live in some suitable climate. He should undergo hydropathic treatment, with careful regulation of the different procedures, such as the temperature of the water; his mental and bodily activity should be carefully regulated, and a sufficient amount of sleep insisted upon. In addition, preparations of bromin should be given, and, above all, "moral treatment" should be continuously carried out by the medical attendant. I can testify to the fact that occasionally all intestinal symptoms disappear in these cases as soon as the general nervous symptoms begin to improve.

If habitual constipation is due to abnormally sluggish peristalsis, the treatment should be on completely different lines. The first point to determine is whether the sluggish peristalsis is due to some disorder of the musculature or of the nervous apparatus of the intestine. In the treatment of this condition physical methods are of paramount importance. Laxatives of any kind should be employed only symptomatically. All that can be accomplished by the administration of a purgative or laxative is a momentary and transitory effect. The primary disorder is never modified by this means. Every medical man has undoubtedly often seen patients of this kind who travel from spa to spa, going to a different one every year, who feel comparatively well

[1] O. Ziemssen, *Berlin. klin Wochenschr.*, August 13, 1900.

while they are away from home, but are forced to have recourse to laxative pills, etc., as soon as they return. It is undoubtedly good for some of these cases of habitual constipation to leave the city and to spend a few weeks in the pure air of a health resort, such as Marienbad, for instance, and to drink the waters, but they should never expect a permanent cure from such a vacation.

[Hemmeter[1] speaks highly of the effects of Bedford Springs, Pa., and says that it is one of the few mineral waters that can be taken for a long time without harm ; he explains this by the slight purgative action due to the small amount—ten grains to the pint—of magnesium sulphate present in the water.—Ed.]

In the treatment of fecal colic and of fecal tumors very energetic drastic purgatives and large quantities of laxative salts or waters must occasionally be given. In other instances the evacuation of the bowel contents must be brought about by manual manipulation—that is, by skilful kneading of the fecal masses within the bowel ; at the same time the bowel contents must be softened by injections of water or of large quantities of oil. In manipulating the intestine for this condition it should always be remembered that the intestine may be weak and easily ruptured, owing to the presence of fecal ulcers, and great care should consequently be taken. Occasionally large, hard masses of fecal matter accumulate in the ampulla of the rectum and cannot possibly be propelled through the anus by any peristaltic movement of the bowel. In these cases the hardened masses must be removed directly with the fingers, sometimes after preliminary breaking up with instruments.

The treatment of fecal colic, of course, consists in the removal of the primary cause of the colic—namely, of the stagnating masses of fecal matter. At the same time it is unwise to proceed energetically in this direction unless the diagnosis is definitely established. Evacuation should be postponed for a while and opiates be administered to combat the pain in all those cases in which the diagnosis cannot be positively made at once. While a little time is lost in this way, this is not so dangerous in fecal colic as the immediate administration of eccoprotics would be if the diagnosis should happen to be wrong.

[Believing that micro-organisms, by putrefaction and fermentation, exert a definite influence on peristalsis, Roos[2] has treated constipation by the administration of living cultures of the colon bacillus in keratin capsules and has obtained good results. Dead bacilli had no effect, though dead yeast acted satisfactorily.—Ed.]

DIARRHEA.

THE clinical conception of diarrhea entails two factors—viz., soft or liquid consistence of the dejecta and frequent evacuations of the bowel contents ; at the same time it must be remembered that many individuals habitually pass solid dejecta two or three times daily, which,

[1] Hemmeter, *Diseases of the Intestine*, 1901, vol. i., p. 392.
[2] Roos, *Münch. med. Wochenschr.*, 1900, No. 43.

of course, is not diarrhea. Conversely, under certain conditions, a single solid dejection may possess some pathologic significance, or, again, a very pultaceous evacuation is not necessarily pathologic. The conditions in each individual case settle whether the evacuation is to be regarded as pathologic and diarrheic.

In a preceding section (p. 81) the causes of a pultaceous stool were explained. Diarrheic stools are caused by the presence of a large amount of water in the feces, which may depend on the liquid contents of the small intestine being so rapidly hurried into the colon that very little absorption is able to occur in the small intestine, or, again, on very free transudation of water from the intestinal wall into the lumen of the bowel, either from the blood-vessels or from the glands. In the former case a distinct reaction for bile-pigment may occasionally be obtained in the stools with fuming nitric acid, whereas this reaction is never seen in diarrhea solely due to involvement of the large intestine. A positive reaction for bile-pigment, therefore, indicates that diarrhea depends on the small intestine, but a negative reaction does not exclude this origin for diarrhea. The causes leading to an excessively watery condition of the bowel contents will be described in the section on the different forms of diarrhea.

More rapid peristalsis, either in both the small and the large intestine or in the large intestine alone, is a second factor of importance in the production of diarrhea. Occasionally, as in many cases of diarrhea produced artificially by certain purgatives, this is the only factor at work, for in these cases evacuation of the contents of the small intestine is entirely due to increased peristalsis and not to any chemic or physical changes of the bowel contents or to any structural changes in the bowel-wall. This accelerated peristalsis must always involve the large intestine as well as the small intestine, for when it is limited to the jejunum or to the ileum,—and this occasionally happens,—the evacuations are not necessarily diarrheic in character.

Pathologic acceleration of intestinal peristalsis may be produced in a variety of ways. In the great majority of cases it is caused by some anatomic disease of the intestine, but it may occur without such change. For example, increased peristalsis occurs as the result of irritants in the contents of the bowel or when the bowel contents are perfectly normal, but the irritability of the nervous elements of the intestinal wall or of the sensory nerves is increased. Finally, increased peristalsis may occur when the muscular coats of the intestine are stimulated as the result of an irritant affecting the central nervous system or circulating in the blood. Occasionally several of these factors are operative at the same time.

The macroscopic and microscopic appearances of diarrheic evacuations vary greatly according to the etiology of the disease and the anatomic changes that constitute the primary cause of the diarrhea. For some of the general features of diarrheic stools the reader is referred to the section on feces, while further details will be given in the accounts of various diseases. In estimating the probable conse-

quences of an attack of diarrhea (not to speak of the cause and the
nature of the process) it is important to know whether it is caused
merely by abnormal transudation and exudation and increased peristalsis
of the large intestine, or whether, at the same time, the peristaltic move-
ments of the small intestine are greatly increased. In the latter case,
and especially when the acceleration of peristaltic movements begins
very high up in the small intestine, large quantities of unchanged diges-
tive fluids and digestible, but not digested, food-remnants will be evacu-
ated *per anum*, and general nutrition is naturally greatly impaired. In
rare instances the ingesta rush through the whole intestine from the
stomach to the anus with enormous rapidity and reappear in the stools
almost unchanged. The older physicians called this condition lientery,
which imitates a direct communication between the stomach and the
transverse colon—such as occurs when carcinoma of the stomach or
transverse colon sets up adhesions and then perforates into the other
viscus.

I do not think we are justified in dropping the old-established, purely
clinical, symptomalogic conception of diarrhea in favor of the concep-
tion of catarrh and other anatomic changes in the intestine and this
view has recently been again abandoned. Cases of diarrhea are fre-
quently met with in which no lesions of the intestinal wall can be
discovered, or at least in which these lesions are altogether out of pro-
portion to the intensity of the diarrhea. This group of cases will be
specially discussed in this section, while those forms of diarrhea due to
anatomic changes in the intestinal wall will be dealt with in the sections
devoted to these lesions and their consequences.

Those forms of diarrhea in which there are no discoverable changes
in the intestinal wall may be classified etiologically as follows. Those
due to :

1. Irritants present in the bowel contents.
2. Irritation transmitted through the nervous system.
3. Irritants present in the blood.

Irritation from the Bowel Contents.—Diarrhœa Cathartica.
—Although diarrhea produced by purgatives does not properly belong
to the subject of clinical pathology, it may be discussed here. Certain
drugs, such as colocynth, aloin, and cathartic acid, may, it is true, also
act as purgatives when administered by subcutaneous injection, but,
practically speaking, act exclusively from the intestine. As a detailed
description of purgatives is outside of the scope of this work, a few
general remarks only will be made.

The majority of purgative drugs act by stimulating the peristaltic
action both of the small and the large intestine—chiefly, however, of
the latter. Aromatic laxative drugs produce diarrheic evacuations in
this way exclusively. The dejecta, after the administration of the
latter class of remedies, are thin and liquid, not on account of the
transudation of liquid from the blood and hypersecretion of the inflamed
mucous membrane, but owing to the fact that increased intestinal peris-
talsis interferes with the absorption of the intestinal secretions (of the

gall-bladder, pancreas, and intestinal glands) which are normally poured into the upper portions of the intestine.

The action of laxative alkaline salts is more complicated, and the diarrheic character of the stools after the administration of these drugs seems to be due to different factors. Alkaline salts primarily increase peristalsis, although to a slighter degree than the first-named class of remedies. In addition, however, they act as concentrated saline solutions, and, in accordance with the laws of osmosis, withdraw water from the blood, since it is the less concentrated of the two fluids. When very dilute solutions are introduced into the intestine, the diarrheic character of the stools is due to the retention of this fluid in the bowels; Glauber's salt, for instance, which is not very diffusible, prevents the absorption of these fluids. Lastly, it is claimed that these salts are capable of stimulating the secretion of intestinal secretions. According to the careful analyses of Radziejewski, the diarrheic stools produced by purgative remedies differ from normal stools only in the increased amount of water contained in the former.

Diarrhœa Dyspeptica (compare also section on Intestinal Dyspepsia).—Various articles of diet may produce pultaceous or even liquid evacuations, especially fresh fruit, pickles, and some kinds of cabbage and turnips. In some persons milk possesses this power, whereas in others it has the opposite effect and produces constipation. The effect of these articles of food may be due to certain inherent qualities or to the fact that they are eaten in excessive quantities, or, finally, because they are mixed with certain liquid articles of diet (water, beer) that prevent proper gastric digestion and cause them to enter the intestine in an undigested state and so produce increased peristalsis. The latter effect may be due to the formation of acids, to the development of gas, or may be purely mechanical. Bokai has proved, by a number of experiments, that sulphureted hydrogen, carbon dioxid gas, and carbon disulphid gas all possess the power of energetically stimulating intestinal peristalsis.

Catarrhal changes in the intestinal wall are rare—at any rate, in the early stages. Later the continued irritation of the abnormal intestinal contents may give rise to changes typical of catarrh. In the dyspeptic or alimentary forms of diarrhea, therefore, catarrh, as in the case of laxatives administered by mouth, is caused by an irritant present in the bowel contents. The increased peristalsis is the best remedy both for the diarrhea as a symptom and for its primary cause, because as soon as the peristalsis is increased the bowel contents are rapidly evacuated and the irritation of the intestinal mucosa ceases. The administration, therefore, of remedies that produce constipation artificially would manifestly be a therapeutic error in these cases.

Many of these patients in addition to diarrhea frequently complain of abdominal pain, which must be considered the expression of increased spastic peristalsis. There are, in addition, some dyspeptic symptoms, nausea, belching of gas, which is often offensive, loss of appetite, and a bad taste in the mouth. The evacuations themselves are usually first

pultaceous and then liquid; they are generally mixed with considerable quantities of offensive gas (carbon disulphid and hydrocarbons).

[Under the name of the celiac affections Gee[1] described a form of chronic indigestion met with in persons of all ages, but especially in children of from one to five years of age. The feces are loose, not formed, more bulky than the food taken would seem to account for, pale in color, as if devoid of bile, yeasty, frothy, and very offensive, and resembling oatmeal porridge or gruel in appearance. He speaks of it as being the same as diarrhœa alba of India (which is the same as sprue or psilosis), but does not correlate any definite morbid change with the celiac affection. The course of the disease is slow; emaciation, pallor, cachexia, and frequently dropsy supervene; death is common, and recovery is often followed by a peculiar weakness of the legs which shows itself in that the child is unable to jump.—Ed.]

Diarrhœa Gastrica.—Einhorn and Oppler were the first investigators to call attention to this important form of chronic diarrhea. Martius, Rosenheim, Ewald, A. Jones, Boas, and R. Schütz are others who have described it. The last-named author rightly called it *chronic dyspeptic* diarrhea, but I suggest *diarrhœa gastrica* to avoid confusion with the form described in the preceding section. The latter term, furthermore, serves to suggest at once the etiologic relations of the condition. All cases coming under this head show a primary deterioration in the gastric digestion, manifested usually as achylia gastrica (Einhorn, Oppler), sometimes as hyperchlorhydria with very high acidity, and, at times, motor insufficiency of the stomach.

When the insufficiently digested food reaches the small intestine, demands far in excess of the normal are made upon the peptic powers of this organ. A healthy intestine is equal to the demands, and thus it is that in achylia gastrica and hypochlorhydria the stools may remain normal indefinitely or the action of the bowels may be merely sluggish. But when the intestine is not healthy, when its resisting powers are insufficient, the irritation caused by the incompletely digested or undigested stomach-contents causes a diarrhea that is independent of anatomic lesions in the intestine itself. Gradually, however, there arises a true catarrh with a pathologic, increased secretion of mucus. Rosenheim and Schütz believe this to be the case in the majority of subjects, many of whom become nervous and neurasthenic.

It will be seen that the practical importance of this form of diarrhea is great, inasmuch as many cases of diarrhea present no gastric symptoms, the entire clinical picture being dominated by the enteric symptoms. Passages occur from 1 to 10 times daily, in some cases always immediately after taking food. Flatulence, borborygmus, and colicky pains coexist. The stools have a sour, not feculent, odor, consistency watery to thick and pasty, impregnated with gas-bubbles. They contain considerable undigested food—meat, connective tissue, and carbohydrates. When intestinal catarrh has already developed, mucus can

[1] S. Gee, *St. Bartholomew's Hosp. Reps.*, 1888, vol. xxiv., p. 17.

be demonstrated in the stools. This form of diarrhea may last for years, even to a score or more.

Diarrhœa Stercoralis.—When constipation occurs in persons whose bowels usually act regularly and normally every day, diarrhea frequently follows. The interruption of the normal bowel evacuation in these cases may be due to many causes, but usually depends on some interruption of the ordinary mode of life. The diarrhea, which is usually insignificant, is accompanied by rolling and gurgling in the intestine, by more or less severe colicky pains, and by the evacuation of fecal matter that is first solid, then pultaceous, and lastly liquid; at the same time offensive gases (CH_4 and H_2S) are expelled. These patients also complain of a feeling of swelling and distention, of eructations, and of mild dyspeptic disturbances. These symptoms all disappear without any treatment when the diet is properly looked after.

This form of diarrhea is caused by the development of CH_4 and H_2S in the intestinal contents as a result of the prolonged stagnation of fecal matter, which, as has been shown, are capable of stimulating peristalsis. This form of diarrhea, therefore, is essentially the same as the dyspeptic diarrhea just described.

In habitual chronic constipation intercurrent diarrhea occasionally occurs and is also probably due to the irritation of flatus. It is more probable that the mucosa of the intestine is really occasionally in a state of catarrhal irritation owing to the retention of stagnating fecal masses.

Diarrhœa stercoralis, like diarrhœa dyspeptica, may, under unfavorable conditions—improper treatment and errors in diet—lead to catarrhal changes in the intestinal wall. In the early stages, however, this form of diarrhea is never accompanied by such catarrhal changes, and therefore I feel justified in discussing it here.

Diarrhœa Entozoica.—The question naturally occurs, Can intestinal parasites cause diarrhea without changes in the intestinal mucous membrane? We cannot answer definitely, and as the subject is fully considered in special works dealing with animal parasites, and we shall pass it without discussion in this place.

Irritation of Nervous Origin.—Diarrhœa Nervosa.—Diarrhea, like constipation, may depend on nervous disturbance, without any morbid change of the intestinal wall. The original description of diarrhœa nervosa by Trousseau is very clear and concise. It may originate in two ways : either from excessive stimulation of the nerves governing peristalsis (it is impossible to prove whether paralysis of fibers inhibiting peristalsis would produce the same effect) or from the transudation of serous material into the lumen of the intestine, brought about by certain nervous influences. It is probable that in some cases both factors are in operation at the same time.

It is clear that occasionally these stimuli originate from the nerve-centers and are transmitted along the fibers of the vagus, the sympathetic, or the splanchnic nerves. Whether nervous diarrhea may depend on irritation of the peripheral nerves in the intestinal wall has not yet

been established; as a matter of fact, this proposition would be very difficult to prove; at the same time it does not seem impossible.

The prototype of nervous diarrhea is that in which some violent psychic shock, usually fear or fright, causes the repeated and very fluid evacuations, which, on examination, will be found to be the liquid contents of the large intestine. In these cases the peristaltic action of the intestine is accelerated, and at the same time there is an abundant transudation of fluids into the lumen of the intestine. It is clear that the primary irritation must start from the cerebral centers. Another proof of this is the following: patients with chronic intestinal catarrh, who, by careful regulation of the diet and other treatment, succeed in limiting the evacuation of the bowel to one a day, find that the evacuations of the bowel are increased by any emotional shock. Leyden reports a number of cases in women with perfectly normal digestion, in whom any emotional change produced sudden attacks of diarrhea. No doubt every practitioner has had experiences of this kind, some of them most unique.

In all these cases we are dealing with acute conditions which are transitory. There is, however, another form of nervous diarrhea presenting very various clinical manifestations, which becomes a chronic symptom and even an independent chronic disease (Nothnagel, Peyer). The only way in which to gain a clear understanding of this protean affection is to study the details of a number of histories of such cases. I have, for instance, seen individuals in whom gurgling and abdominal pain, tenesmus, and diarrhea occurred regularly whenever they could not have access to a water-closet, and only under these circumstances. In others again merely seeing a closet immediately produces an urgent call to stool; further, in another group of cases this desire occurs only at definite hours and without any relation to their environment. At the same time these patients present nervous symptoms, such as vertigo, stupor, congestion of the head, reddening of the face, a feeling of hot flushes all over the body, fear, or a feeling of oppression, particularly in the chest, rapid breathing, palpitation, etc. All these symptoms frequently disappear rapidly after a few diarrheic evacuations. In other instances again diarrhea is the only symptom.

The number of stools varies greatly: there may be two or four, or there may be as many as twelve or fifteen. The material evacuated usually consists of thin, liquid intestinal contents. Mucus is rarely present, or at best only in minimal quantities. In a case described by Wick his patient passed at intervals a total of 150 to 200 gr. of an almost clear liquid, containing neither albumin nor sugar, but the ash of which showed considerable potash and sodium salts. Occasionally the first evacuation is solid, the second one pultaceous, and all the later ones liquid. Defecation is occasionally accompanied by great peristaltic unrest, with violent rolling and gurgling in the intestine and very severe tenesmus, and a large amount of gas is frequently expelled. I have also known patients otherwise perfectly well who evacuated the bowel contents only once every three to six days. As soon as these patients

became nervous for any reason they had an evacuation of the bowels daily, and quite regularly, without having to employ any artificial measures to bring about this result. In these cases, therefore, the solitary daily evacuation is an expression of a nervous form of increased peristalsis.

There are many different causes for chronic nervous diarrhea. Beard described this form of diarrhea as a frequent symptom of neurasthenia, and claims that it is seen in general nervousness and hysteria. It is not yet explained why in these neuroses there is sometimes nervous diarrhea and in other instances nervous constipation. In exophthalmic goiter, diarrhea, apparently of nervous origin, is occasionally met with. In migraine the patients sometimes suffer from attacks of diarrhea, particularly toward the end of a paroxysm, which may or may not be accompanied by colicky pain (Möbius). Peyer speaks of a "reflex" form of nervous diarrhea seen in certain conditions of irritation or weakness of the sexual system both in man and woman—for instance, catarrh and ulceration of the uterus, in morbid emissions and spermatorrhea, and in various forms of sexual excesses. In some instances diarrhea persisted for several years and resisted all treatment, and finally disappeared after the cure of some sexual disorder—such as the cure of an ulcer of the uterus or the replacement of a retroflexed uterus (Fischl). Condio describes obstinate vicarious diarrheas in pregnant women with a neuropathic tendency, the diarrhea supplanting the vomiting. Bieganski's cases of nervous diarrhea occurred in excessive smokers, and he is, therefore, inclined to attribute some importance to the abuse of tobacco. Möbius, under the name of "nervous digestive weakness of the intestine," described a peculiar condition met with in neurasthenics. In this form patients with a good appetite, who eat an abundant quantity of food and have no subjective digestive disturbances, begin to emaciate greatly. The stools are apparently normal, except that defecation occurs several times a day, and that each motion is excessively large. If the general nervous condition is treated and cured, the abnormal character of the fecal evacuations and the emaciation disappear. I have also had some nervous patients in whom the abnormality in the stools consisted in far too copious evacuations of feces, partly of normal consistence, partly fluid. Kelling has reported a familial type, grandmother, mother, and child being affected with a periodically recurrent nervous diarrhea. All were subject to dizziness, fainting, vomiting, and edema. I wish, however, to insist on the fact that diarrhea possessing all the characteristics of so-called "nervous diarrhea" may occur in individuals who otherwise present no symptoms of nervousness whatever.

The diagnosis of nervous diarrhea is based on the following points: In the first place, the character of the dejecta and certain peculiarities in the time of defecation are almost bizarre. This is the most important symptom. In addition there are symptoms of nervousness and an absence of all morbid symptoms referable to impaired digestion or lesions of the intestinal tract.

The following very simple case, selected from many similar observations, may serve as an illustration. The patient, a merchant, thirty-five years of age, without any hereditary nervous predisposition, began, six years previously, without appreciable cause, to suffer from diarrhea whenever he was exposed to the slightest mental excitement. When free from all mental excitement or psychic perturbation, he was perfectly well for several weeks and had a daily evacuation of a solid, well-formed stool, and enjoyed an ordinary mixed diet. As soon, however, as the slightest psychic disturbance occurred, diarrhea supervened. In these attacks defecation became frequent and liquid feces were evacuated. This occurred whenever he became frightened, depressed, or worried by any insignificant detail in the course of his daily life. For instance, if a child fell down while playing in the same room, or if a chair was overturned, he immediately developed diarrhea. In fact, he once had an attack in which he passed three liquid stools in rapid succession when a doctor prescribed a laxative for one of his children. When he entered the clinic for examination I ordered him to evacuate his bowel contents, but he declared that he was unable to do so at the time. As soon as we suggested giving him a laxative he immediately and spontaneously had an action of the bowels, which was perfectly normal in consistence. During the attacks of diarrhea this patient suffered no pain, and no mucus or blood was ever seen in the stools. While defecating, however, he frequently complained of a feeling of oppression in the neck, and of oppressive frontal headache; he also believed that there was some loss of memory and intelligence (in talking to the patient I could not verify this opinion); he also frequently complained of pain in the hypogastric region. Nothing abnormal on examination, except some tenderness on pressure over the third and fourth thoracic vertebræ and hyperalgesia of the skin over these points.

Diarrhea from catching cold deserves some consideration here. It is an established fact that diarrhea may occur after a sudden and severe chill or wetting of the surface of the whole body, or of certain portions of the body, particularly the abdomen and the feet. Many individuals develop diarrhea on exposure to cold, just as other persons develop coryza, sore throat, or bronchitis. The exact genesis of this diarrhea is more or less obscure—probably, however, to judge from analogous processes when a person " catches cold," the blood-vessels of the intestinal mucosa become hyperemic, owing to the reflex irritation transmitted from the nerves of the skin. As a result of this vascular hyperemia serum transudes into the lumen of the intestine and an increased quantity of mucus is secreted. Whether the accelerated peristaltic movements that occur are also due to reflex stimulation from the nerves of the skin, or whether it is secondary to the hyperemia of the intestine, cannot be decided.

This form of diarrhea is usually very transitory and ceases after a few rapidly succeeding evacuations of the bowels; in other cases, however, as shown by the clinical course, this diarrhea from cold undoubtedly assumes the character of a true intestinal catarrh.

3. Irritation Transmitted through the Blood.—Diarrhea due to the hypodermic injection of certain drugs must be included in this category. This form has been already mentioned above.

Further, some cases of nephritic, or better uremic, diarrhea occasionally met with must be included under this heading, since it is produced by the action of carbonate of ammonia, which is derived from urea excreted into the lumen of the bowel from the blood. Diarrhea is frequently seen in patients with nephritis whose intestinal

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mucosa is found pale and unchanged after death. As a rule, however, the irritation of carbonate of ammonia rapidly leads to anatomic changes of the intestinal wall that may be catarrhal, diphtheric, or ulcerative in character.

Hirschler believes that in the vagus we have the motor medium for the stimuli acting directly through the blood-channels.

I believe that those typical forms of diarrhea occasionally seen in malaria which disappear on the administration of quinin, must also be included in this group. The same applies to the diarrhea seen in cholera. The profuse evacuations in cholera are not due to anatomic changes of the intestine proper, but to the action of the cholera toxin, which causes hypersecretion of all the glands of the intestine, and at the same time inhibits absorption in the lower portions of the intestinal tract. For the details, reference should be made to the section on Cholera; here attention is merely directed to the fact that probably the cholera poison, formed by the comma bacillus, is absorbed into the blood-vessels from the lumen of the intestine and reaches the intestinal glands through the blood, in this way stimulating their secretion to an enormous degree. The attacks of diarrhea occasionally seen in septicemia, which can be experimentally produced by injections of the putrid toxins, are frequently due merely to violent stimulation of peristalsis (Cohnheim), for in many of these cases no histologic changes can be found in the intestinal wall. There are many other instances of diarrhea due to some infectious disease without visible lesions of the intestine itself—for instance, in croupous pneumonia, in erysipelas, and in influenza. Even in typhoid fever diarrhea often occurs long before intestinal ulcers develop and before there is a catarrh of the intestine.

TREATMENT.

A review of the different forms of diarrhea enumerated shows at once that no universal method of treatment is possible in this disease : each form must be treated in a special manner, and it is essential that the etiologic factors and the primary cause of the diarrhea must be taken into account in the treatment. It would be a gross error to endeavor to combat every diarrhea with emplastics.

When the administration of an excessive dose of some laxative remedy has produced undue purgation, opium and heat (see below) are indicated. This is probably the only emergency in which opium should be given for diarrhea. As a general rule, it is best to allow this form of diarrhea to cease spontaneously and even to neglect the subsequent constipation that may follow and last for a short time afterward.

The dyspeptic and stercoral forms of diarrhea should be treated on similar lines. As a matter of fact, laxatives (castor oil, calomel, and salines) may be required in these cases even though the diarrheic evacuation of the bowels is quite abundant. This method of treatment should be adopted when putrefactive processes are manifestly going on in the evacuated masses and when there is considerable development of gas,

since these signs show that the intestinal contents are in a state of active decomposition. The early prevention, by artificial means, of the evacuation of the bowels under these conditions would be a serious error.

The statements made above hold true with diarrhœa gastrica. In this also emplastics are useless, oftentimes even harmful, and are indicated only in exceptional cases of profuse intercurrent diarrhea. The treatment must always be governed by the gastric processes causing the condition. Hydrochloric acid and lavage are useful, as are also injections, high up into the large bowel, of 2 or 3 liters of lukewarm water or normal salt solution. Regulation of diet requires the greatest care. Schmidt commends milk with addition of salicylic acid. Meats must be reduced to a minimum, and when again permitted, must be taken in finely divided condition. Easily digested egg-preparations, beef-tea, dry-toasted white bread, and similar foods are permissible (see p. 197).

The treatment of diarrhea due to entozoa naturally consists in removing the cause, and reference should be made to special works for the best methods of doing this.

The success of treatment in nervous diarrhea, particularly when chronic,—and this is probably the only form that comes to the medical man for treatment,—is frequently surprising. When a correct diagnosis is not made, the numerous and varied kinds of treatment, which are eminently and rapidly successful in other forms of diarrhea, are quite useless. This applies particularly to constipating remedies and courses of mineral waters. Even when the origin of the trouble has been made out, treatment should be carefully adapted to the requirements of each individual case. In the " reflex " form of diarrhea the primary focus of irritation must be removed. The diarrhea of neurasthenia and hysteria calls for methodic treatment directed toward the disease of the nervous system. Occasionally nervous diarrhea seems to baffle all treatment, however carefully it is considered and planned ; under these conditions it is worth while trying a number of methods at random, for occasionally the appropriate remedy is thus hit upon. Thus, I remember a case of diarrhea in a neurasthenic subject that was cured by the administration of arsenic after a great many methods of treatment had been employed in vain and after the diarrhea had existed for over six years.

No general therapeutic rules based on a study of the etiology can be applied to the treatment of diarrheas due to the action of irritating substances carried to the intestine by the blood. In all these cases the primary toxic focus must be removed or the treatment must be symptomatic.

The regulation of diet is not so important in the forms of diarrhea described in this section as in diarrhea due to anatomic lesions of the intestine. It is immaterial what the patients eat in cases of nervous diarrhea. In order to avoid repetition reference should be made to the section on Catarrh of the Intestine for the directions as to diet in diarrhea.

Here, however, the various methods of treatment and drugs that can be employed in the symptomatic treatment of diarrhea must be briefly noted. These methods of treatment have no reference to the etiology of the disease or to the anatomic changes that may be present; and, as already indicated, are purely symptomatic and more or less empiric.

Among the drugs to be employed opium occupies the first place and is the best remedy we possess. It has been tested millions of times and has been found reliable in cases of diarrhea due to the most diverse causes. At the present day morphin is used instead of opium in almost all conditions where this remedy is indicated; in diarrhea, however, opium and preparations made directly from opium are still in use. Experience shows that this method of administration is the best; it is true that opium and morphin, qualitatively speaking, have the same effect on the intestine; possibly the more marked effect of opium in diarrhea and in colic depends on the presence of certain resinous constituents in crude opium. These constituents of the drug possibly cause the active ingredients of opium, which are in intimate combination with these resinous bodies, to be more slowly absorbed, and consequently to exert a more prolonged local effect on the intestine. Another advantage of opium over many antidiarrheic remedies is its power of stopping pain, in addition to inhibiting peristalsis. Opium, of course, merely acts symptomatically; indirectly, however, it aids in the cure of diarrhea by reducing the violence of peristalsis and thus keeping the tissues at rest for a time and giving them an opportunity of spontaneously returning to their normal state. I have mentioned that opium should never be given in the dyspeptic or stercoral forms of diarrhea.

Apart from opium, I know of no remedy that can be considered a general antidiarrheic; no other drug has the power of arresting peristalsis whatever the primary cause of the increased movements of the bowels. It is true that many other remedies are employed in the treatment of diarrhea (subnitrate of bismuth, preparations of tannic acid, etc.), but they are employed only in diarrhea due to definite causes and definite pathologic conditions. Their administration will be discussed in the sections on the Anatomic Lesions of the Intestine. There is, however, one other valuable measure in general use in diarrhea—namely, heat. In the majority of cases of diarrhea heat applied to the abdomen, in the form either of dry or of moist warm compresses, acts beneficially. It is difficult to explain this action. Horvath, Lüderitz, and Bokai have published a number of investigations on the effect of changes in the temperature of the body on the movements of the intestine, but these physiologic experiments cannot be utilized in explaining the effect of heat on diarrhea. Pal, however, found that the irritability of the intestine (irritation of the vagi after division of the splanchnics) was greatly reduced when the intestine was placed in warm (103° F.—39.5° C.) physiologic salt solution and thus explains the well-known fact that heat seems to quiet the increased peristalsis due to irritation of the intestine.

DYSPEPSIA OF THE INTESTINE (Dyspepsia Intestinalis).

UNDER the heading of intestinal dyspepsia all the abnormal proc-
esses of digestion occurring in the intestine are included. [Hemmeter
points out that the term intestinal dyspepsia is inappropriate, inasmuch
as peptic digestion is not involved, and that dystrypsia is a better term,
since the pancreatic ferment is usually defective.—ED.] Faber, I
think, has recently used this term incorrectly from an etymologic
standpoint. He has included under it gastric symptoms arising not in
the stomach, but in the intestine. Dyspepsia intestinalis literally means
impaired peptic activity of the intestine.

The normal processes of intestinal digestion, in the widest sense,
must naturally be disturbed when the walls of the intestine are dis-
eased, and especially when the morbid change involves the most im-
portant physiologic part of the wall of the bowel—namely, the mucosa.
Secretion and absorption are greatly altered in catarrh of the intestine,
in multiple ulceration, etc.; as a result, the processes of disassimilation
in the intestine become perverted, so that those constituents of the intes-
tinal contents that would normally be absorbed, as well as those nor-
mally expelled by rectum are changed. This series of pathologic
changes will be discussed in connection with the various anatomic
diseases of the intestine.

Again, intestinal digestion must naturally be thrown out of gear
when the necessary secretions entering the intestine from without are
excluded. This applies to the bile and the pancreatic juice. In the
absence of bile intestinal digestion becomes very greatly perverted.
This deviation from the normal is a well-established fact, and has been
made the subject of exhaustive clinical, chemic, and experimental in-
vestigation, with the result that it is thoroughly understood. Fried-
rich Müller has recently published a monograph giving a clear review
of the present state of our knowledge on this subject. The effect
of obstruction of the pancreatic duct is not so well understood, and the
abnormalities and perversions of intestinal digestion that follow this
accident are more or less obscure. This is chiefly due to the fact that
clinically simple cases of atrophy of the pancreas or obliteration of the
duct of Wirsung are comparatively rare. Intestinal dyspepsia depend-
ing on absence of the bile or pancreatic juice will not be considered
here, inasmuch as this subject is fully dealt with in the volume dealing
with Icterus and Diseases of the Pancreas.

Disturbances of intestinal digestion may, however, be due to other
causes. The digestive processes in the intestine must naturally become
abnormal when the condition of the intestinal contents is no longer
normal and physiologic. This may occur even when the mucosa re-
mains intact and a sufficient amount of bile and pancreatic juice enter
the intestine. I refer particularly to cases in which the quality and the
quantity of the ingesta are normal, but in which certain special microbes
gain an entrance into the intestine; and also to cases in which the con-
tents of the intestine are so abundant as to overtax the powers of the

digestive secretions and render normal digestion impossible ; in the same category must be placed those cases in which the food is indigestible or in which different and incompatible kinds of food prove too much for the powers of the digestive ferments. Proteid digestion may be more disturbed than the digestion of carbohydrates, or, conversely, the digestion of carbohydrates may be abnormal, while that of proteids remains normal. On the slightest provocation the bacteria and hyphomycetes that are always present in the intestine develop in an abnormal manner and lead to increased fermentation of carbohydrates and the formation of organic acids, particularly acetic acid and lactic acid, and of certain gases. This form of dyspepsia is very important, more especially in children and in breast-fed infants. Bednar and Henoch believe that a large proportion of the cases of infantile diarrhea are due to fermentation and decomposition processes in the contents of the stomach and intestine without any material changes of the mucous lining of these organs. A number of investigators have recently been occupied with a study of the details of these processes. Escherich and Baginsky, in particular, have carefully studied the rôle of bacteria in this form of diarrhea.

Here attention will briefly be called to the following points of view : Primary acid fermentation in the intestine must naturally influence the functions of the digestive fluids—namely, the bile, and especially the pancreatic juice—poured into the intestine, inasmuch as the latter can act only when the contents of the intestine are alkaline. As a result of this inhibition of the action of the pancreatic juice and the bile, the elaboration of the chyme is still further impaired, and finally the intestinal contents become so abnormal that irritation of the intestinal wall is set up. The most important factor in the whole process, however, is the formation of organic acids.

This form of intestinal dyspepsia is also important in adults, in whom its harmful influence is twofold, as in children. Very frequently this form of diarrhea, especially when the diet is not well chosen and rational treatment is not instituted at once, constitutes the chief etiologic factor in the development of genuine catarrh of the intestinal mucosa. This is due to the fact that the abnormal acids that are formed in the intestine and the other anomalies of the intestinal contents act as irritants and are the direct cause of anatomic changes in the intestinal wall. This very important point will be referred to in the section on Catarrh of the Intestine. In the second place, this form of intestinal dyspepsia *per se*, even if it does not present the same clinical aspect as definite catarrhal enteritis, may give rise to a definite group of symptoms. This syndrome of intestinal dyspepsia is very frequently seen in young children and infants, and is also met with in adults. As a rule, these patients develop pronounced symptoms of gastric dyspepsia—loss of appetite, acid eructations, and vomiting—and at the same time suffer from diarrhea. The increased peristaltic action of the intestine is caused by the irritation exerted by the abnormal acid present. Pathologic fermentation of the contents of the gastro-intestinal tract occurs both

in the stomach and in the upper portions of the small intestine; while
the abnormal peristaltic movements begin in the jejunum. At the same
time the rapid peristalsis, extending from above, drives the acid con-
tents into the lower portions of the bowel. In this way the intestinal
contents irritate and cause increased peristalsis in every part of the in-
testine with which they come in contact, and the contents of the jejunum
are rapidly moved downward. The evacuations in these cases at first
consist of the contents of the lower portions of the bowel, but the con-
tents of the jejunum rapidly follow, and possess special features which
may be considered almost characteristic of acid dyspeptic processes going
on in the upper portion of the small intestine. When the evacuations
present these characters (see following paragraph), the condition may be
considered jejunal diarrhea.

Normally, the contents of the jejunum are gelatinous, tenacious, and
semifluid. The acid stools derived from the jejunum present the same
appearance. The gelatinous consistence of these stools is shown by
chemic examination to be due to the presence of mucus. It appears,
however, that the mucin present is not the result of catarrhal inflamma-
tion of the intestinal mucosa, but that it constitutes a physiologic admix-
ture to the contents of the jejunum. Even in health the higher portions
of the small intestine contain a large percentage of mucus, which, when
peristalsis is increased, appears in the stools.

A large amount of mucus in the stools is usually regarded as evi-
dence of some anatomic catarrhal change in the intestinal wall. The
question arises, whether it is possible to distinguish between mucus from
this source and mucus derived from simple dyspepsia of the small intes-
tine. In the first place, the formed elements, such as epithelial or
isolated round cells, which are so abundant and so common in catarrhal
mucus, are absent from the mucus in cases of dyspepsia of the small
intestine. This point is of particular importance. In addition, the
stools of a patient suffering from dyspepsia of the jejunum are often
intensely green and give a very pronounced bile-pigment reaction, and
are, moreover, usually markedly acid in reaction. This is natural when
the pathogenesis of the disease is taken into account. In the later stages
of the disease the stools become catarrhal or alkaline, and it then be-
comes difficult to decide whether the diarrhea is purely dyspeptic in
character or whether catarrhal changes in the intestinal wall have de-
veloped. Finally, the stools in cases of jejunal diarrhea present the
other properties of the contents of this portion of the bowel—that is,
the different articles of food are only partially digested and no fecal
odor can be detected. A consideration of these various points will
usually make it possible to differentiate without much difficulty between
a catarrh of the intestine and simple jejunal diarrhea. It must never
be forgotten, however, that the latter condition may rapidly lead to
catarrhal changes, so that in later stages of the disease there is often
true catarrh of the intestine.

Ad. Schmidt and Strasburger have recently studied a form of func-
tional disturbance in the small intestine, which they term " intestinal

fermentation-dyspepsia," which closely resembles jejunal diarrhea. The following is an abstract of their description of the clinical features: " The subjective symptoms are rather vague, but abdominal pains, chiefly in the umbilical region, fatigue, and discomfort are the most prominent. Abdomen frequently symmetrically distended, usually tender on palpation either universally or limited to umbilical region or to the left of it. Examination of stomach rarely gives any pathologic indications. Feces (after test-meal) evacuated somewhat more frequently, but no actual diarrhea. Stools are often foamy, light yellow, acid, with the odor of butyric acid. Other indications usually absent, particularly noticeable being the lack of mucus, and often of starch, but after ingestion of potato there occur plentiful empty potato cells." Schmidt and Strasburger believe that a part of such cases are in all likelihood due to catarrh, but that others are independent of organic changes, being purely functional and some of nervous origin. They believe that intestinal fermentation dyspepsia depends upon insufficient digestion of starch, and that test-meals and fermentation-tests are of the highest diagnostic value.

Finally, there is another origin for intestinal dyspepsia, which also depends on abnormal decomposition of the intestinal contents—viz., putrefactive decomposition of proteids. This occurs in prolonged fecal accumulation. The diarrhea accompanying it, however, is really a secondary symptom, and has no further clinical significance. It will be discussed in detail in the sections on Coprostasis and Enterostenosis. One aspect of the clinical picture presented by this perversion will be discussed in the section on the Urine (compare p. 165).

TREATMENT.

Following the general plan explained in the preceding paragraphs, the treatment of one form only of intestinal dyspepsia will be dealt with here,—namely, the form that is primary in character and directly due to bacterial acid fermentation of the contents of the upper portion of the small intestine,—in other words, acid jejunal diarrhea. Baginsky has written a very careful dissertation on this subject in which he has elucidated the different points of view that must be considered in the treatment of this disease, and has laid special stress on the fact that antibacterial treatment, which at the present time is so popular, is not necessarily indicated in all these cases. He showed that under certain circumstances the formation of acetic acid by the Bacterium aceticum may even be a protective process and defend the organism against invasion by other more dangerous forms of pathogenic microbes reaching the intestine from without. In actual practice it will be found a difficult matter to differentiate between the different forms of intestinal dyspepsia, and to determine whether we are dealing with fermentative decomposition of carbohydrates or putrefactive decomposition of albumin, etc. At all events, this decision cannot be rapidly made, and in general, therefore, treatment must be essentially empiric.

One of the most important features in the treatment of these cases is the regulation of the diet. As we have to deal almost entirely with conditions of acute onset and course, it follows that the inhibition of food to as great an extent as possible is indicated. These patients may be allowed to take a little weak tea (without sugar, possibly with a very little brandy and skimmed milk). If there is much flatulence, they should be given some carminative, preferably in the form of a tea. Subsequently gruel, chicken or beef bouillon, and a little meat jelly as solid food may be allowed. After two days it will usually be easy to determine whether carbohydrate fermentation or albuminous putrefaction predominates, so that the diet can then be regulated accordingly.

The best remedy is calomel, a drug that has stood the test of long experience. The dose should be from 0.03 to 0.05.

[According to Van Valzah,[1] calomel should be given in doses of $\frac{1}{40}$ grain one to four times a day, and if it has a tendency to hurry food through the intestines, smaller doses should be given. Some patients can bear $\frac{1}{20}$ grain several times a day. If patients are sensitive to calomel, protiodid of mercury should be given. Ichthyol in 4-minim doses one to four times a day on an empty stomach is also useful. Many intestinal antiseptics interfere with the fermentation processes of digestion, as well as inhibit bacterial growth.—ED.]

The chief effect of calomel is to inhibit the fermentation of carbohydrates. Other remedies that may be administered are organic acids, particularly if the abnormal fermentative processes in the intestine are due to the action of pathogenic microbes. To meet this indication lactic acid has been frequently recommended and seems to be very popular. Resorcin, creasote, boric acid, and possibly menthol are all so-called direct antibacterial remedies and may all be tried.

On pages 165 to 171 the diagnostic significance of the analysis of the urine in cases of intestinal dyspepsia will be considered, and some general regulations will be given as to the choice of a diet and the remedies to be prescribed.

THE ACCUMULATION OF GAS IN THE INTESTINE
(Meteorism; Tympanites).

THE older authors spoke of the abnormal accumulation of gas in the intestine as meteorism when it occurred acutely, and as tympanites when the condition was chronic. At present both terms are used without distinction. The term *flatulence* is used to indicate great formation of gases that are removed by eructations and flatus.

Physiologically, the intestine always contains a certain amount of gas. Some of these gases, the oxygen and nitrogen of the atmospheric air, are swallowed with the saliva and the food, and enter the stomach and intestine. Oxygen is rapidly absorbed, and is therefore not found in the large intestine. Carbon dioxid in the intestine is in part derived by diffusion from the blood-vessels of the intestinal wall, and partly from

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[1] Van Valzah, *Medical News*, January 17, 1903.

fermentation in the intestinal contents.   Hydrogen, ammonia, methane, and sulphureted hydrogen ($H, NH_3 CH_4, H_2S$) are entirely due to intestinal fermentation and putrefaction.

Although gas is continuously entering the intestine of healthy human subjects, and is practically always being formed there, meteorism, does not develop, since any excess of gas is immediately expelled.   In this way the average tension and the average volume of the gases present in the intestine under normal conditions fluctuate within very narrow limits.   Only a small amount of the gas that is formed every day, or that is added to the amount already present, forming a surplus, is expelled in healthy adults either upward or downward (eructations and flatus); the greater proportion of this excess is absorbed, and the tension and volume of the gases in the intestine thus regulated.   Traube points out that this can be recognized by the fact that few healthy individuals suffer from eructations or flatus.

Regnault and Reiset were the first to demonstrate that a certain proportion of methane leaves the body through the lungs in the expired air, and that this gas originates in the intestine.   Since their first publications a number of other authors have worked at this subject.   Tacke discovered that rabbits exhale from 10 to 20 times more of the intestinal gases through the lungs than they expel through the anus.   Zuntz subsequently tried to make out whether the same applies to human subjects; he failed to arrive at positive results, but his experiments render it rather doubtful whether the same conditions obtain in human subjects. At the same time there is no doubt that a large proportion of the gases of the intestine is absorbed into the blood, and that this process plays an important part in regulating the volume and tension of the gases present in the intestine.   This modern view directly contradicts the older idea that the intestinal gases leave the bowel almost exclusively *per anum.*

ETIOLOGY AND PATHOGENESIS.

The abnormal accumulation of gas in the intestinal canal is essentially due to two chief causes : in the first place, too much gas is developed within the intestine, or is introduced from without; in the second place, a normal or an excessive amount of gas is formed, but too little is removed by the normal channels.

(*a*) **The Introduction and Formation of Abnormal Quantities of Gas.**—Cases in which the accumulation of abnormal quantities of gas occurs acutely from swallowing large quantities of air are quite rare, and may almost be considered curiosities.   The gases, moreover, in these cases accumulate chiefly in the stomach.   The same applies to the accumulation of carbonic acid gas following the ingestion of aërated beverages.

[Aërophagy, or the swallowing of air, has been carefully studied in 8 cases, 3 of whom were males, by Lyonnet and Vincent.[1]   It usually occurs in hysterical women, and may come on suddenly from shock or

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[1] Lyonnet and Vincent, *Lyon médical*, February 10, 1901.

emotional disturbance. It may be voluntary—a physiologic curiosity —or involuntary and pathologic, due to spasm of the pharynx. The symptoms are distention after food, loss of appetite, desire to sleep, but not infrequently insomnia, and frequent noisy eructations. Vomiting is rare. The symptoms described by Bouveret as neurasthenic gastro-intestinal atony may be present. Constipation is usually present, while mucous colic, enteroptosis, and floating kidney may coexist. Occasionally there is considerable wasting, so that new growth is suspected. Before the eructations there are rapidly repeated deglutition movements. If these are overlooked, an erroneous diagnosis of flatulent dyspepsia may be made. Treatment consists in—(1) Combating hysteria ; (2) counteracting pharyngeal spasm by keeping the mouth widely open, painting the pharynx with cocain, blisters to the front of the larynx, bromids, valerian, and belladonna ; hypnotic suggestion has had an extremely marked effect in some cases ; (3) the muscular tone of the stomach may be stimulated by strychnin and ergot ; food should be concentrated and not bulky. The duration of the condition is variable— sometimes it is cured by very simple means ; on the other hand, relief may only be temporary, and frequent relapses are prone to occur.—Ed.]

The formation of an abnormal quantity of gas in the intestine depends on two conditions : there must either be large quantities of fermentable substances in the intestine, or bacteria or fungi capable of causing various forms of fermentation—*e. g.*, lactic acid, butyric acid, acetic acid, and alcoholic fermentation ; and the decomposition of fats and the conversion of starch and cellulose must be present. In the former process hydrogen and carbon dioxid are developed, while in the latter a greater proportion of the methane is found in the intestine. (For details on these points the reader is referred to pages 17 to 64.) Since the majority of the ferment organisms concerned in these processes are always present in the intestine of healthy subjects, the abnormal development of gas must be referred to the introduction of an excessive amount of fermentable material, such as unfermented grape-juice and beer, a number of vegetables containing much cellulose, such as varieties of cabbage, turnips, peas, beans, lentils ; baked foods prepared with yeast, fresh bread, etc. In some individuals large quantities of milk also cause an abundant development of gas in the intestine.

Schmidt rightly emphasizes the fact that the quantity of carbohydrates is not the only factor, and that their assimilability must also be considered. The extent of intestinal fermentation is in about an inverse ratio to the assimilable qualities of the amylaceous foods taken.

The development of gas is also occasionally encouraged by abnormal conditions of the digestive tract and perversions of the processes of digestion, particularly by some forms of catarrh. This is probably due to the fact that in catarrhal inflammation the intestinal contents are abnormal and provide a suitable culture-medium which favors the growth of certain forms of bacteria and hyphomycetes.

(*b*) A variety of factors may be referred to as being responsible for the fact that occasionally **smaller quantities of gas than normal**

are expelled. Among these may be mentioned mechanical obstruction of the intestine, a reduction or inhibition of the muscular power of the intestinal wall, and any conditions that interfere with the absorption of gas from the intestine by the blood. These factors may be operative either when a normal amount of gas is present in the intestine or when an abnormal quantity is developed there. Occasionally all the factors enumerated act together. Zuntz, relying on the experiments of Tacke, was at one time inclined to attach greater importance to the absorption than to the expulsion of gas in regulating the quantity present in the intestine, but later he changed his views somewhat. As a matter of fact, clinical experience and observation show that the expulsion of gas *per anum* is at least as important in this regulatory process as the absorption of gas by the blood.

The occurrence of meteorism in stenosis of the intestine can readily be understood. Intestinal obstruction, however, does not necessarily produce meteorism at once, and several days may elapse before the condition becomes pronounced; apparently a great deal depends on the constitution of the intestinal contents and their power of producing gas at the time when obstruction of the intestine occurs. These factors, of course, determine the rapid or the slow development of gaseous fermentation products. If the production of gas is slow at first, the surplus may be removed from the intestine by absorption into the blood. Later, stagnation occurs and the contents of the intestine undergo rapid decomposition, and the production of gas, of course, is greatly increased. As soon as the amount of gas present exceeds a certain limit, the intestine becomes distended, and this inhibits the absorption of gas by the blood-vessels of the intestinal wall. In addition the excessive distention impairs the contractile powers of the intestinal musculature, as I have been able to prove experimentally. All these factors in their turn lead to the further accumulation of gas in the intestine. In this way a vicious circle which cannot be interrupted results, and causes the colossal meteorism occasionally seen in stenosis of the intestine.

Meteorism in acute diffuse peritonitis is due to impairment of the muscular powers of the intestine. Loss of muscular tone is also responsible for the meteorism, occasionally enormous, sometimes seen in infectious fevers, particularly in typhoid fever, and also in pneumonia, sepsis, pleurisy, etc. I agree with Mannaberg, that the meteorism in these cases, which almost always run a severe course, is due to toxic paralysis of the intestine. This subject is more fully considered under Paralyses of the Intestines. Leyden calls particular attention to the fact that relaxation of the general muscular tone is responsible for the accumulation of gas so frequently seen in collapse. The general rule may be formulated that all conditions that impair the energy of the intestinal contractions favor the development of meteorism.

[Acute abdominal distention, apart from disease of the intestines and peritoneum, is particularly liable to occur in children under three years of age in severe bronchopneumonia; it may also occur in acute

cerebrospinal and posterior basic non-tuberculous meningitis (Still).[1] In some cases it appears to be a phenomenon of dying, but this is not necessarily so. The distention comes on rapidly and may seriously embarrass respiratory movements and the action of the heart. Usually there is only time for local measures, such as the passage of a soft tube into the sigmoid flexure ; minim doses of creasote may be of use. In some of these cases there may be diarrhea before the onset and a catarrhal condition of the intestine may be associated with and perhaps responsible for the distention.—Ed.] Acute meteorism after operations on the intestines deserves considerable attention. It is thought to be due to paralysis of the intestine, and will be considered at length under that head.

Zuntz, as has been pointed out, regards interference with the absorption of intestinal gases by the blood-vessels of the intestinal wall as the chief factor in the production of meteorism, and believes that this absorption is interfered with when more or less severe circulatory disturbances occur in the intestinal wall. According to this view, the occurrence of meteorism in collapse, in febrile diseases in which the general circulation is depressed, in peritonitis, and in puerperal processes, but chiefly in all acute and chronic obstructions of the portal circulation, is invariably due to interference with the circulation of the blood through the wall of the intestine. That the absorption of gas by the intestinal blood-vessels is an important factor in regulating the volume of gas present in the intestine has been definitely determined by physiologic experiments. In pathologic cases, or rather clinically, it is impossible, however, to determine what share in the production of meteorism must be attributed to the perversion of this function, as the conditions in any given clinical case are usually so complicated that several factors act at once.

A peculiar form of meteorism is occasionally met with in nervous individuals which deserves special notice here. This form of meteorism is most striking and commonest in the hysterical, and usually occurs as a diffused distention of the abdomen (tympanites hystericus), but also, though far less frequently, as a circumscribed swelling—the so-called phantom tumor. The pathogenesis of this form has been much discussed. The cases of this type do not apparently present any uniform clinical history, different cases showing individual peculiarities and etiologic possibilities. Magendie emphasized the pathogenicity of swallowed atmospheric air. Ebstein explained its passage from stomach to bowels by insufficiency of the pyloric valve, originating in the central nervous system. He was able to demonstrate this, as inflation from carbonic acid always immediately increased the existing (gastric and) intestinal tympany. It is very doubtful whether the gas, of which there is often a considerable amount, can pass from the blood into the intestine as rapidly as the appearance of meteorism would suggest. Talma attributed hysteric tympanites to a continuous contraction of the diaphragm. This may be diffuse, with coexisting relaxation of the other abdominal

[1] G. F. Still, *Pediatrics*, September 15, 1897.

muscles, or partial, with concomitant contraction of individual abdominal muscles—such as partial contraction of the transversalis abdominis. According to this, partial and diffuse tympanites would have a common pathogenesis. Talma denies that there is any increase in the gas of the intestines, and cites as reasons that in narcosis the abdomen at once becomes flaccid, while with returning consciousness meteorism promptly recurs, and in neither case can air be detected entering or leaving the body. Percussion is the guide to the state of the diaphragm. Finally, no doubt, a number of varying conditions of contraction in the muscular walls of the intestine must be considered. Valentiner has advocated a general acute paresis, of sudden onset, passing off with equal rapidity, analogous to numerous other hysterical paralyses. It may take the form of a local intestinal tetanus or a circumscribed paresis of the intestinal musculature, and is also occasionally seen in hypochondriasis. Other authors are inclined to attribute the rapid disappearance of meteorism to absorption of the accumulated gas by the blood. Jolly, however, argues that it is quite unnecessary to invoke this factor, because it is quite possible that the accumulated gas, which is usually odorless, might escape through the anus, and that this expulsion might occur unnoticed and noiselessly.

SYMPTOMATOLOGY.

In most cases of meteorism the shape of the abdomen is altered. In general meteorism it is distended quite symmetrically, and is readily distinguished from ascites, for in this affection the sides of the abdomen become most prominent, while in meteorism it is chiefly the anterior portion that is protruded. When gas collects in parts of the intestine only —partial or local meteorism—the affected parts stand out in bold relief. In severe cases of this kind errors are easily made in recognizing the distended parts; for example, the descending colon may extend far to the right and the transverse colon may occupy most of the middle of the abdomen. It is, therefore, wise to exercise great caution in the localization of partial meteorism. The coexistence of visible peristalsis (as in intestinal stenoses) simplifies diagnosis, as its shape may sometimes indicate whether the part is the large or the small intestine. (Other points bearing on this matter will be found under Intestinal Obstruction and Stenosis, p. 367.)

The degree of distention of the abdomen in general corresponds to the amount of gas present. A second factor determining the distention of the abdomen is the resistance offered to the expansion of the accumulated gases both by the muscular tissue of the intestine itself and by the muscles of the abdominal wall. The weaker the muscles and the more reduced their tone, the greater the degree of distention. This fact explains the high degree of distention seen in peritonitis, and the occurrence in puerperal peritonitis in particular, of the most extreme degrees of abdominal distention; for in the latter condition there are, in the first place, paresis of the muscular coat of the intestines, and, in the second place, great weakness of the muscles of the abdominal wall as a direct

result of the distention of these parts during the preceding period of pregnancy.

In contradistinction to these cases distention of the abdomen is occasionally prevented, or at least inhibited, by increased tonicity of the abdominal muscles. In these cases the diaphragm is forced upward by the gases accumulated in the intestine, and dyspnea results. This form of meteorism is easily overlooked and may endanger the life of the patient.

On palpation of the abdomen of a case of this kind it will be found that the abdominal walls are very tense and that the whole abdomen presents a peculiar resistance to the fingers, which has been called air-cushion resistance. The feeling imparted may best be characterized as tense and elastic, and at the same time very hard.

The percussion-note is changed ; as a rule, it is abnormally low and loud. The tympanitic ring normally present is also usually lost. These peculiarities of the percussion-sound constitute the so-called meteoristic note. On forcible percussion (with the hammer and pleximeter) a metallic sound is occasionally elicited. With combined percussion and auscultation this metallic sound is heard very distinctly. Limitation of the metallic sound to one portion of the abdomen may in some cases indicate the seat of a stenosis of the intestinal canal, but any conclusion of this kind should be made with great caution, since metallic sounds due to this cause are sometimes heard over wide areas of the abdomen in combined percussion and auscultation. The peristaltic propulsion of liquid intestinal contents may also produce gurgling sounds with a distinct metallic ring.

The liver dulness is reduced and finally disappears altogether, so that the pulmonary note merges directly into the tympanitic note heard over the intestines. This is due to the fact that the liver is tipped backward in such a manner that finally the only part of the organ in contact with the anterior thoracic wall is its lower sharp margin, which is so thin that it does not modify the percussion-sound over the region of the liver.

When the accumulation of gas in the intestine is considerable, the abdominal walls are usually pale and peculiarly shiny. In contradistinction to ascites, the umbilicus is rarely obliterated, although this may occasionally occur.

The distended loops of intestine force the diaphragm upward ; the lower and posterior portions of the lungs are compressed and become collapsed. The percussion-note over the lungs becomes dull ; the lower margin of the pulmonary resonance begins anteriorly in the nipple-line at the level of the fourth or fifth rib ; the apex-beat of the heart is correspondingly displaced and is found in the fourth or the third intercostal space, somewhat outside the nipple-line. The movements of the diaphragm are interfered with, and respiration finally becomes purely costal, shallow, and rapid. Subjective and objective dyspnea develops, and the patients become cyanotic. Finally the jugular veins may swell so that the bulbi appear as prominent nodules. When meteorism, espe-

cially the fulminating form, becomes very marked, symptoms of asphyxia and cardiac failure develop, the pulse grows weaker and weaker, and death finally ensues. Several cases of this kind have been reported.

The accumulation of gas *per se* and the resulting intestinal distention may produce a number of disagreeable sensations, and in very sensitive people actual pain. At the same time true colicky pains never develop as a result of meteorism (compare the section on Intestinal Pain); when colicky pains develop together with the accumulation of gas in the intestine (colica flatulenta), it must always be assumed that tonic contractions of the intestine occur at the same time. As soon as some of the gas is expelled (with or without fecal matter), the subjective and objective symptoms become ameliorated.

All the other symptoms occasionally observed in any given case are not due to meteorism alone, but to the primary disease, or rather to the factors that produce the disease, or, lastly, they may be presented by chance in certain individuals, thus a variety of nervous symptoms may occur in the course of meteorism in neuropathic subjects.

The more rapidly meteorism develops, the more severe the symptoms and the more dangerous the condition. Meteorism is particularly dangerous if it develops as a complication in severe diseases in which the respiratory system is involved—for instance, in typhoid and pneumonia, and may then directly lead to death from asphyxia.

Hysteric meteorism deserves special description. When general, it resembles physically the same condition due to other causes. But the manner of its onset and course are more remarkable than even its intensity. In many women the abdomen at the menstrual period is distended in a manner resembling peritoneal tympanites. Changes in disposition may cause tympanites in the hysterical. The condition may last hours, days, or even weeks, and disappear spontaneously or form fresh psychic stimuli. It may disappear after gentle rubbing or faradization of the abdomen or after tight bandaging. At other times the meteorism accompanies hysteric convulsions, and again it appears without recognizable cause at a definite time of the day.

Apart from this widespread hysteric meteorism, there appears also a circumscribed form that may present the aspect of tumors, hence the name *phantom tumors*. These are not to be confounded with the *apparent tumors* (scheinbaren Tumoren) of the abdomen. Under this head Einhorn includes abnormal positions of the viscera or abnormal conditions of parts of the abdominal wall, simulating new growths. Spencer Wells has given an exhaustive description of such cases in which the phantom tumor presents the appearance of an ovarian or uterine tumor, or of a bilateral ovarian cyst, and in which the rectus muscle formed a sharp line of demarcation between the two halves of the abdomen. The phantom tumor may occupy the epigastrium or other part. These partial meteoristic prominences are sometimes exceedingly hard and resistant. Powerful contractions of abdominal muscles may give an impression of hard growths, but there is this diagnostic peculiarity, that their percussion-note is always resonant, and that they disappear under narcosis,

reappearing with returning consciousness. In fact, all that has been said concerning the characteristics of hysteric meteorism applies to them.

TREATMENT.

Treatment should be primarily directed toward the removal of the etiologic factors responsible for meteorism. This, of course, is only possible within certain limits, as many of the causes of meteorism are not amenable to treatment. Articles of diet known by experience to produce flatulence in a given individual must be avoided. In constipation the bowels must be kept open. Catarrh of the intestine, if present, must be treated by appropriate means. In peritonitis, typhoid fever, sepsis, and other acute febrile diseases the treatment of meteorism coincides with the treatment of the primary disease. The same applies to meteorism occurring in stenosis of the intestine. Distention of the intestine following cardiac failure calls for stimulating treatment carried out on the recognized lines. The treatment of meteorism in hysteric subjects will be discussed below.

A number of drugs and of different methods are employed in the treatment of meteorism itself. Unfortunately, the effect of the majority of these measures is very unreliable, particularly in severe cases. No method is known of promoting the absorption of intestinal gases by the blood; this object in any given case can probably be attained by counteracting any concomitant weakness of the heart, by reducing the high temperature in certain fevers, etc. The most important practical object, however, is to promote the expulsion of gas *per anum*.

Since vigorous peristalsis favors the expulsion of gas, purgatives seem to me the most effective method of treating meteorism. It is essential, of course, in giving these drugs that the bowel should not be obstructed and that conditions contraindicating purgation, such as typhoid fever, should be absent. The choice of the laxative will depend on the peculiarities of each individual case.

Carminatives are also useful in the treatment of meteorism; they are closely related to laxatives, and have a similar though slighter and less energetic physiologic effect, hence they are used exclusively in mild forms of flatulence. Among the most useful carminatives mention may be made of caraway seed, peppermint, mint, thyme, cinnamon, cloves, nutmeg, anise-seed, sassafras, etc. All these remedies may be administered in a variety of forms and combinations. The best way to give them is in the form of a tea, prepared in the ordinary way in the kitchen. Brunton and Cash performed a number of experiments with carminative drugs (asafetida and oleum caryophyllorum) and arrived at the conclusion that they promote actual absorption of some of the gases in the intestinal tract (CO_2 and H_2S). They are chiefly employed in cases of flatulent colic following simple indigestion or " catching cold." Oil of turpentine was formerly given *per os* or *per anum*, but in my experience this remedy is quite unnecessary in simple cases, and useless in more complicated and severe ones.

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In cases in which laxatives are suitable the peristaltic action of the bowels may be stimulated by rubbing the surface of the abdomen. In intestinal obstruction, or when the presence of marked structural change in the intestines is known to be present, massage is, of course, either counterindicated or quite useless. If it is employed at all, simple methodic massage may be given, or rubbing with cloths, or with spirituous, aromatic, and ethereal substances (linimentum camphorato-saponatum, olei carbi, cajeputi, terebinthini, balsamum nucistæ, etc.). The application of cold, either externally or internally, is also occasionally useful. Faradization exerts a similar physiologic effect to massage, but is less important from a practical point of view.

Occasionally the introduction of a tube into the rectum will aid the escape of gas by stimulating the peristaltic action of the bowel. Enemas are still more effective in this direction, particularly injections of cold water. An attempt may also be made to draw off gas directly through a long tube introduced into the intestine, but its results are usually very doubtful.

Certain drugs have been given internally for the purpose of absorbing gas, but the effect of these remedies is at best very slight, and probably altogether fictitious. Calcined magnesia, lime-water, prepared oyster-shells, and animal charcoal are utterly valueless in severe cases, and unnecessary in mild ones. Subnitrate of bismuth possibly possesses the power of dissociating a limited volume of sulphureted hydrogen, but this gas is probably the least important in meteorism.

Recently von Noorden has recommended physostigmin. Should his good result be confirmed by general experiences, it would be of greatest importance. Physostigmin in his hands gave remarkable results in a case of meteorism with reflex intestinal paralysis (without peritonitis) following an operation for inguinal hernia ; in severe cases of obscure origin ; and in grave meteorism in typhoid fever. He gave doses of 0.0005–0.00075 to 0.001 gm. of salicylate of physostigmin, the smallest dose three or four times daily, the largest twice, internally as powders.

In desperate cases, in which the distention of the abdomen threatens the life of the patient, an opening into the intestines with the knife or trocar may be made. This procedure, as we know, is frequently carried out by veterinary surgeons on some domestic animals prone to abdominal distention. If the primary disease is not incurable, puncture of the abdomen may save the life of the patient.

[A very full account of this subject, with a collection of cases, will be found in J. W. Ogle's [1] pamphlet on "The Relief of Excessive and Dangerous Tympanites by Puncture of the Abdomen." The subject is also referred to later in the section on the Treatment of Occlusion of the Bowel, p. 650.—Ed.]

Meteorism in hysteric subjects often passes away without any treatment, and even suddenly ; in other cases again it may resist all treatment. The internal administration of carminatives, like valerian and

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[1] J. W. Ogle, *On the Relief of Excessive and Dangerous Tympanites by Puncture of the Abdomen*, Churchill, 1888.

asafetida, is advisable; or these drugs may be given in the form of enemas. Massage and mild friction treatment or faradization of the abdomen should all be employed. In the Middle Ages the abdomens of the hysteric St. John's dancers, who developed meteorism, were tightly laced with bands of cloth.

INTESTINAL PAIN (Enterodynia; Colica; Enteralgia).

SEVERAL kinds of intestinal pain can be distinguished according to their mode of origin and their clinical characteristics—namely, (*a*) Pain originating from inflammation, either of the intestinal wall or of the peritoneum covering the intestine; (*b*) the pain of genuine nervous enteralgia; (*c*) the typical pain of colic. The inflammatory forms of pain will be considered among the other symptoms of the various anatomic forms of intestinal disease.

THE PAIN OF COLIC.

Colic is the painful stimulation of the intestinal nerves which is accompanied or caused by severe tonic contractions of the intestine. Intestinal colic is pathologically related to biliary, renal, and uterine colic, for in all these cases the pain is produced by spasmodic contractions of the unstriped muscle of the parts.

Traube has defined the old conception of the word colic as follows: Colic is the pain experienced whenever the wall of a viscus, which contains involuntary muscle-fibers, undergoes violent peristaltic contractions. I agree with Traube that the pain of colic (the word is used in this place only to designate intestinal colic) is produced by the irritation of the nerves of the intestinal wall following violent contractions of these walls. Genuine peristaltic movements, however, do not irritate the nerves of the intestinal wall in this way. For the production of colicky pain, violent tonic contractions of the intestine are necessary. This distinction is quite clear; in the former case the intestinal contractions are always of the continuous, progressive type, while in the latter the contractions persist for a shorter or longer time, but are very violent and lead to rigidity of the intestinal tube in the affected area; in this way the nerves in the intestinal wall are mechanically irritated by the pressure thus exerted; this is analogous to the irritation of the nerves of the muscles of the calf in so-called cramps. The tetanic form of contraction may be so severe that the lumen of the intestine is completely obliterated. Such advanced degrees do not, however, occur in all cases, for it is often possible to see, when the abdominal walls are thin, that loops of intestine, more or less distended with gas, are converted into rigid tubes during tetanic contraction. This is usually seen above some obstruction, and the tetanic contraction of the muscular coat generally lasts for a very short time only. Simple peristalsis of the intestine never produces pain, even when the movements are very violent and cause most energetic expulsion of the intestinal contents and loud gurgling sounds (borborygmi). Traube inclined to the belief that the

presence of an obstruction in the intestine which prevented the onward passage of the bowel contents always produced violent peristaltic contractions of the intestine, which in their turn produced the pain of colic; but this view is erroneous, for the contractions that originate under these circumstances do not possess the character of peristaltic movements, but constitute true tonic contractions of the intestinal wall. That this is the case can readily be seen when the abdomen of an animal is opened and the intestine exposed (it should, of course, be kept in warm physiologic salt solution); it may also, as mentioned above, occasionally be seen through the abdominal walls in human subjects.

The question arises whether the opposite condition, namely, great overdistention of the intestine, is capable of producing colicky pain. That this is not the case is shown by the symptoms produced by meteorism, for in this condition a disagreeable and even slightly painful feeling of distention may be experienced, but genuine colicky pain, with all its characteristics, is never complained of. Colic may, of course, be accompanied by an accumulation of gas in the intestine, but in this instance the distention of the intestine is not the direct, but only the indirect, cause of the colicky pain, for the accumulation of gas irritates the intestine and causes tonic contractions of the intestinal wall in certain places. That this sequence of events actually occurs can be shown experimentally.

ETIOLOGY.

Tonic contractions of the intestine producing colicky pain may, physiologically speaking, occur under two conditions: either when some qualitatively or quantitatively abnormal irritant stimulates the intestinal wall (its muscles or its nerves), or when the intestinal wall is hypersensitive to normal stimuli. Both factors may, of course, be active at the same time. In both cases peristalsis may simply be increased or tonic contractions of the intestine may develop; or, finally, and this is the most frequent event, both the changes may occur together. When tetanic contractions of the intestinal wall occur, the irritant or the condition of sensibility must be greater than in the case of increased peristalsis.

From a practical point of view the most important point is to make out the cause of the pain. As a preliminary step it must be determined whether the abdominal pain really possesses the characteristics of intestinal colic. When this has been done and the cause of the condition has been discovered, treatment should be started, and should be directed exclusively to the primary cause of the condition. Clinically speaking, the following causes of intestinal colic must be considered:

The ingestion of certain articles of food that the patients are not accustomed to. Here the idiosyncrasy of the patient is of great importance. In some people new beer, some kinds of fruits, or certain coarse and indigestible articles of diet produce this effect; in others again articles of diet that the patients can usually digest with impunity— in other words, normal articles of food—produce colic when eaten in

excessive quantities (colica saburralis). Occasionally unusually cold drinks produce colic (ice, ice-cold beer, and mineral waters cooled in ice).

Substances which give rise to excessive gas-formation when taken into the intestine irritate the intestinal wall by distention and set up secondary tetanic contraction of the bowel (colica flatulenta).

Fecal accumulation is a frequent cause of a very violent form of colic (colica stercoralis—compare the section on Constipation) ; in exceptional cases large gall-stones or other foreign bodies which have passed into the intestine have this effect.

The presence of large quantities of mucus, as seen in the syndrome called colica mucosa (see this disease).

The presence of entozoa, teniæ, and ascarides.

Certain laxatives—for instance, senna.

It is impossible to prove absolutely that in all these cases the pain of colic is really dependent on tetanic contractions of the intestine, but I feel justified in making this assumption on the grounds that it can be positively demonstrated that such contractions of the intestine actually occur in another form of colic due to another cause—namely, in inflammatory colic (colica inflammatoria sive catarrhalis). I have been able to demonstrate experimentally that in acute inflammations of the bowel tonic contractions of the intestinal wall occur in certain areas in addition to violent peristalsis. These tonic contractions are so severe that the involved area of the intestine is converted into a solid cord. These tetanic contractions directly cause the violent gnawing pain of colic occasionally experienced in acute catarrh of the intestine, and in addition other more continuous painful sensations. On the other hand, cases of acute diarrhea are occasionally met with in which increased peristalsis produces loud borborygmi audible at a distance, but does not cause colicky pain. So called colica æstiva and biliosa also probably belong to this group, and will be described in the section on Catarrh of the Intestine.

Another prolific cause of colic is exposure to cold (colica rheumatica). This form develops either when the whole body or some parts of it, particularly the feet, become wet. Whether or not these cases are due to true inflammatory processes or to some vasomotor disturbance in the intestine induced by reflex irritation from the cutaneous surface, or whether, finally, the muscular coats of the intestinal wall are reflexly stimulated to contraction by the same influences, cannot be definitely decided. This form of colic may or may not be accompanied by diarrhea. The factor determining the presence or absence of diarrhea is the extent of intestine involved. When the small intestine and colon are involved, there is diarrhea, but not when the small intestine alone is affected.

It is very important to remember that pain of a colicky character may also occur in some structural diseases of the intestine other than catarrh. Thus, colic occasionally occurs as a precursor of perityphlitis (compare the section on this disease), and is probably produced by spasmodic contractions of the walls of the appendix. Colicky pain is also

frequent in various forms of ulcer (Bamberger has reported colic in typhoid ulcers).

[T. McCrae [1] has analyzed 500 cases of typhoid fever : in 206, or 41 per cent., there was no pain at any period of the attack ; in 61 there was pain only at the onset ; in 161 there was pain during the course of the disease ; and in 72, tenderness but no pain. Pain may be due to a number of various factors, both connected and unconnected with the disease, but in 70 cases pain occurred without any discoverable cause ; in 14 cases perforation was to some extent imitated. As there were leukocytosis and local abdominal symptoms, two of these cases were operated upon, but nothing was found in the abdomen.—ED.]

It occurs in strangulation of external hernias, in internal strangulation of the intestine, and in stenosis of the bowel. [Colic may for a long time be the only sign of carcinoma of the colon (Crämer).[2] Attacks of colic may occur in association with Henoch's purpura and angioneurotic edema (Osler).[3]—ED.]

Occasionally patients apparently perfectly healthy in every other respect are attacked several times a year with genuine colic accompanied by constipation and possibly vomiting. If no other cause for these attacks can be found, fecal colic as the result of habitual constipation is usually thought of, but if in the interim between the attacks the action of the bowels is perfectly regular, the existence of some other condition that can occasionally produce fecal accumulation and so colic must be considered. Under these circumstances the diagnosis of cicatricial narrowing of the bowel or of narrowing of the intestinal lumen by peritoneal adhesions is a very probable one, particularly if the pain always starts in the same part of the abdomen or if a certain circumscribed area of the abdomen is always tender on pressure.

It is a well-known fact that very young children, and particularly breast-fed infants, show a great predisposition to colic ; colica infantum plays a large rôle in medical practice among children. Colds and indiscretions in diet in children produce catarrh of the intestine which is accompanied by colicky pain ; in some instances colicky pain is present alone, without catarrh of the intestine. Sometimes the mother's milk undergoes a change as the result of emotional disturbance, and this factor alone is frequently sufficient to produce colic in the infant.

[Purgatives or unsuitable food taken by the nursing mother set up colic in the suckling infant. Paroxysmal attacks of toothache in the mother appeared to be the cause of colic in her infant in a case quoted by Illoway.[4] In infants renal colic, as pointed out by Gibbons,[5] may easily be overlooked and regarded as intestinal colic. Intestinal sand is said to be a cause of colic in infants which is sometimes overlooked (R. Saint Philippe).[6]—ED.]

[1] T. McCrae, *New York Med. Jour.*, May 4, 1901.
[2] Crämer, *Münch. med. Wochenschr.*, June 17, 1902.
[3] W. Osler, *Jacobi's Festschr.*, 1900 ; *Practice of Medicine*, 1901.
[4] Illoway, *Phila. Med. Jour.*, February 2, 1901.
[5] R. A. Gibbons, *Med.-Chir. Trans.*, vol. lxxix., p. 41.
[6] R. Saint Philippe, *Jour. de Bourdeaux*, 1901, No. 49.

Lead-poisoning occupies a very important position in the etiology of colic. As this form of poisoning will be discussed in detail in another part of this series, the reader is referred to a section in Vol. I., for an account of lead and copper colic, as well as for a description of colica endemica and vegetabilis, two diseases which should probably be considered as lead colic.

SYMPTOMATOLOGY.

Lead colic is excluded from the following description. Intestinal colic is characterized by two factors—first, by certain peculiar features of the pain; second, by the time at which it appears. The pain of colic is peculiar: it is pinching and boring. These peculiarities of the pain are not noticed by the patients in severe cases because they are only conscious of the extreme pain. Colic occurs in paroxysms which may last only for a few seconds, or may be very prolonged and persist for several hours. Colicky pain usually appears suddenly and disappears just as rapidly. The intensity of the pain varies, and all degrees of suffering are seen. In the more severe and the extreme forms of colic a variety of reflex symptoms are developed; some of these must be interpreted on the same lines as the symptoms developed in Goltz's test.

An attack of colic presents the following clinical aspect: more or less suddenly the patient experiences pain in the abdomen, frequently starting at the umbilicus, and either remaining localized there or radiating in different directions; occasionally, particularly if the pain is due to circumscribed structural changes, it begins in other localized areas of the abdomen. The facial expression of these patients shows their suffering. The attack passes away in a short time. In violent attacks the patients twist and turn in various positions, draw the legs up toward the body, or lie on the abdomen; occasionally the pain seems to be somewhat relieved by remaining absolutely quiet. Sometimes pressure over the abdomen causes relief; in other cases it increases the pain. Drawing and straining sensations are felt in the bladder and the rectum; and occasionally borborygmi can be heard. If the abdominal walls are thin, peristaltic movements, spasmodic contractions of certain portions of the intestine, and inflation of others with gas can be seen. The testicles are occasionally drawn up owing to spasm of the cremasteric muscles, and the abdomen may be retracted. The facial expression resembles that of collapse. The patients are frightened and vomiting may develop, or the patients may feel faint and sore all over. The extremities are cold, and the pulse small and varying in rapidity. In very violent attacks the patients may, in exceptional cases, become unconscious. Young children cry and scream and draw their legs up, and an infant with colic lets go of the nipple or the bottle.

A paroxysm of this kind may persist for many hours with remissions and exacerbations. The general picture presented will, of course, vary somewhat according to the primary cause. In colica flatulenta, for instance, the picture differs from that in colica stercoralis. In

the former the expulsion of gas usually terminates the attack ; in the latter the evacuation of feces.   In colica catarrhalis and rheumatica symptoms of catarrh of the intestine are always present and complicate the picture.   The course of the attacks is also more or less dependent on the primary cause ; if the primary cause persists, the attacks will be repeated, and disappear only when the cause is removed.   Occasionally cases presenting very peculiar characters and not conforming to the ordinary type are met with.   I remember the case of a perfectly healthy man who drank a glass of ice-cold Giesshübler water on a hot August day while travelling by rail ; half an hour later he developed a most violent attack of intestinal colic lasting several hours.   Afterward attacks of colic returned once or twice a week for several months without any appreciable cause, the patient being perfectly well in the intervals between the attacks.

The diagnosis of intestinal colic may occasionally be extremely difficult ; it is often necessary to decide whether the case is one of gastric, biliary, uterine, or renal colic, or whether the symptom is not due to some inflammatory process, such as enteritis, and especially peritonitis. I will content myself with merely calling attention to these difficulties. The final decision in these cases must be made after careful consideration of many different factors which are too widely divergent to be arranged in diagrammatic form ; the only way to make a positive diagnosis is by careful analysis and grouping of the symptoms in each individual case.   I wish, however, to call attention here to one symptom which is frequently misleading—namely, increased tenderness on pressure over the abdomen which does not necessarily indicate the presence of some inflammatory process. and may be seen in colic.

TREATMENT.

The sovereign remedies in the treatment of colic are opium and morphin, which relieve the pain and stop the contractions of the intestine, or rather prevent their return.   Occasionally these drugs alone will bring about a cure ; this applies particularly to the rheumatic form of colic that can be " cut short," so to say, by a full dose of the tincture of opium (10 to 20 drops) given once or twice.   The same to a certain degree applies to the colic accompanying acute catarrh of the intestine, as well as to that form of colic produced by drinking extremely cold liquids.   In addition heat should be applied in various forms (baths, dry fomentations, or poultices, etc., applied to the abdomen) ; warm drinks (Chinese tea, camomile tea, peppermint tea, caraway-seed tea, etc.) ; in addition the diet, of course, should be light.

The same method of treatment should be employed in colica saburralis ; in this form, however, the question of diet requires even greater care.   In the treatment of colica flatulenta the special cause of the formation of gas should be determined and dealt with.   Colic from worms calls for the administration of the various anthelmintics.   Colica stercoralis should be treated by laxatives (see the section on Constipa-

tion). Colic depending on structural changes in the intestine must be treated according to special methods; and symptomatic treatment should be instituted as above.

NERVOUS ENTERALGIA.

I believe that colic may be correctly defined as that peculiar form of pain produced in the bowels by tetanic contractions of the muscular coats of the intestines. This definition in itself refutes the view held by some authors that colic is merely hyperesthesia of the mesenteric plexus, or neuralgia meseraica.

Occasionally, however, a form of pain is met with in the abdomen that is certainly not due to tonic contractions of the intestine, for all symptoms of that condition are absent; at the same time no symptoms indicating inflammation are present, so that we are forced to the conclusion that these cases of colic are of nervous origin. For this form of colic, then, it is desirable to retain the name nervous enteralgia.

The prototype of these cases is the attacks of abdominal pain occasionally seen in hysteric subjects. The picture presented, as Valentiner rightly remarks, occupies an intermediate position between peritonitis and colic. Henoch has published a very exhaustive description of such a case, and I have also occasionally seen the picture of " pseudo-peritonitis " in hysteric subjects; violent spontaneous attacks of pain are experienced in the abdomen that appear suddenly, occur at short intervals, and may be so severe that they are almost unbearable. Associated with attacks of pain there is frequently pronounced collapse, with all the accompanying symptoms, so that the diagnosis of peritonitis may easily be made, particularly as strangury may be present in addition to the severe spontaneous pain, with great abdominal tenderness on very light pressure. One symptom, however, that is rarely absent in cases of peritonitis with a very acute onset—namely, vomiting—is always absent; in addition the skin of the abdomen is hyperalgesic in many of these cases (although not always). This is evident when a fold of skin is carefully raised so that all deep pressure is avoided; it will be found that the skin of this fold is hypersensitive to the slightest touch; this symptom is never seen in peritonitis. The functions of the intestine and the stomach remain undisturbed, and the patients feel perfectly well in the intervals between the attacks. Analogous attacks of pain are occasionally met with in neurotic men. In this category a neuralgic symptom-complex that I have termed pseudoperityphlitis must also be included.

This condition undoubtedly depends on functional excitation implanted on the general neurosis; these colicky pains and peritonitic symptoms are analogous to other forms of pain that are experienced by nervous and hysteric subjects in many different nerve areas. Nothing definite can be said in regard to the nerve-tracts involved in this process.

The following sketch of a case delineates yet another picture of intestinal colic: The patient was a man of advanced age who for a number of years (without knowing anything definite in regard to the

primary cause and the onset of his symptoms) suffered from pain in the abdomen that usually began in the region of the umbilicus and radiated downward toward the left. At the present time attacks of pain occur nearly every day and seem to decrease when the patient moves about or is engaged in some occupation that distracts his attention from the symptoms; as soon as he rests and leaves off his work the pain increases and may even interfere with sleep. The appetite and digestion are good; the stool, if anything, is slightly constipated, but perfectly normal in consistence and composition. The passage of flatus seems to relieve the pain. If, however, a laxative is taken,—as, for instance, castor oil,—and the patient is in the habit of doing this as rarely as possible, —the pain is greatly increased. Pressure over the abdomen is soothing when the pain is violent; in general, however, it is without effect except when the aorta is pressed upon through the thin abdominal walls; this seems to cause pain. Careful palpation and other physical methods of examination reveal nothing abnormal in the abdomen or elsewhere.

Cases of this kind are very difficult to interpret correctly. There is not the slightest reason to assume that any anatomic changes, like neoplasm, catarrh, ulceration, stenosis, or circumscribed peritonitis, are present; further, no definite etiologic factor can be discovered. The diagnosis colica flatulenta is hardly probable, since no definite symptoms of this condition are present and no definite cause can be discovered. Cases of this kind almost force us to the conclusion that there is such a thing as chronic neuralgia of the intestine that persists after the primary cause, whatever it may have been, has disappeared. This would be analogous to the chronic forms of neuralgia seen in other nerve-tracts without any anatomic cause; cases of this kind should, therefore, be regarded as chronic nervous enteralgia proper.

Cases resembling the one just described are frequently encountered in practice. It is impossible to sketch all these features. The only way to arrive at a correct interpretation of each individual case is to analyze carefully each symptom and group the different symptom-complexes, and to study them in their entirety.

A mere reference will be made here to the neuralgic pain of the intestine that is recognized as a symptom of tabes dorsalis, and perhaps of other diseases of the spinal cord. These attacks of pain are known under the name of abdominal crises.

The treatment of nervous enteralgia in hysteric, neurasthenic, or in other nervous subjects coincides with the treatment of the primary disease. The physician will encounter many difficulties in treating cases that are altogether atypical. The only way to treat cases of this kind is to institute measures and administer remedies according to general principles; for instance, a change of air and climate, hot springs, sea-bathing, hydrotherapy, general and local massage, electricity in its different forms, and particularly moral treatment may all be tried. I have repeatedly seen good results in cases of this kind from a long-continued course of arsenic. Strictest attention to each and every symptomatic indication (stools, etc.) is a matter of course.

INTESTINAL HEMORRHAGE (ENTERORRHAGIA).

ETIOLOGY AND ANATOMY.

Loss of blood from the intestine may be due to a great number of morbid conditions, especially anatomic lesions of the intestine. The occurrence of hemorrhage, its frequency or its rarity, its character and violence, will be described separately in the sections on each of the anatomic diseases of the intestine, and for this reason the remarks here will be limited to a general summary of the symptoms of this accident. In order to avoid repetition the reader is referred for the details to the different sections mentioned. Hemorrhage, for instance, may occur in different kinds of ulceration, in acute catarrhs, in toxic inflammation of the intestine, in irritation of the intestine by the excessive use of laxatives, in injury, neoplasm, hemorrhoids, in embolism of the mesenteric artery, in general venous hyperemia of the intestine due to diseases of the heart or lungs, in stasis of the portal system, most frequently in cirrhosis of the liver (*vide* E. Stein's latest collected work on this subject), in volvulus and intussusception, and, in rare instances, after the reduction of a strangulated hernia (first described by Schnitzler; latest complete work on the subject by Kukula and Preindlsberger). In chronic constipation with dried scybala the latter are occasionally covered with streaks of blood. This is due to the fact that they mechanically injure the mucosa in their passage through the intestine. Enterorrhagia caused by ankylostomum is considered in the section on this parasite in the sixth volume of the series.

Occasionally rare local lesions of the intestine cause hemorrhage and must be briefly mentioned. Thus, isolated venous varicosities, that are occasionally present in the small intestine, may produce enterorrhagia. This accident may occur when the mucous membrane of the intestine is otherwise perfectly normal, so that the origin of this form of hemorrhage is very difficult to determine, and is clinically very puzzling, as the patients suffer from no other disturbance of the digestive apparatus and there is not the slightest interference with the general health. The same accident occasionally occurs in small arterial aneurysms of the intestinal wall. Aneurysm of the branches of the hepatic arteries may also occasionally rupture, so that the blood is poured into the bile-passages and causes evacuation of blood by the rectum. Naunyn has collected a number of cases in which it was established that cholelithiasis was the immediate cause of the rupture of an artery.

Still more important, since it occurs more frequently than in other accidents, is the following event: I have on former occasions repeatedly reported cases of this kind, and repeat my description here because the clinical importance of this syndrome is not sufficiently appreciated, although cases of this kind are by no means rare.

The patient was a woman suffering from phthisis. For weeks there was diarrhea, and the stools presented a peculiar brown color resembling that of changed blood. The existence of intestinal ulcers was, there-

fore, not improbable. On autopsy tuberculous ulcers of the ascending colon, the cecum, and the lowest portions of the ileum immediately above the valve were found, but at the same time the contents of these portions of the intestine contained no blood whatever. In the lower portions of the jejunum and the upper portion of the ileum, however, far above the ulcers, the bowel-contents were dark blood and chocolate-colored. The submucous and the muscular coats of this portion of the intestine were macroscopically and microscopically absolutely normal. There was no abnormal development of blood-vessels, but the mucosa was atrophic and the villi and glands were lost. The upper part of the jejunum was also quite normal. In this case, therefore, there was no pronounced hyperemia although intestinal hemorrhage occurred.

In a man suffering from phthisis the stools were thin and the bowels acted only every other day. No blood could be found in the stools. At the autopsy a considerable number of fresh blood-clots were found in the colon. This portion of the bowel was empty, perfectly pale, and like the rest of the intestine was quite free from ulceration. Above and below the transverse colon no trace of blood could be found.

Not only in the bodies of phthisical subjects, but also in the bodies of subjects who have died from other diseases, large or small quantities of blood are occasionally found in the intestine, and are passed with the stools during life. Müller, in particular, has called attention to the fact that enterorrhagia occasionally occurs in prolonged fasting. The only way in which this occurrence can be explained in these cases, especially in the absence of anatomic changes, is to assume that some alteration of the vessel-walls of the intestine has occurred. The same pathogenesis—namely, nutritional disturbances of the vessel-walls—must also be assumed in another class of intestinal hemorrhages that is clinically as important and quite as frequent as the above. I refer to the hemorrhages that occur in a variety of constitutional diseases, in many infectious diseases, and in some intoxications—for instance, in grave anemia, leukemia, scurvy, morbus maculosus, septicemia, severe icterus, yellow fever, phosphorus poisoning, and severe intermittent fever. Bayer has described a fatal case of enterorrhagia occurring immediately after the disappearance of an attack of facial erysipelas. Grainger Stewart observed intestinal hemorrhage in amyloid degeneration of the blood-vessels.

Charcot had already observed that enterorrhagia and other hemorrhages cannot all be attributed to mechanical agents. Leichtenstern, and others, believe that the reason is a nutritional disturbance in the vessel-walls, caused by failure of one of the most important factors in metabolism—the functions of the liver. Huismans reports a case in an otherwise healthy boy of twelve years. A profuse intestinal hemorrhage recurred in six months without any other clinical appearances. The second attack caused extreme anemia and death. The colon, from cecum to sigmoid flexure, was filled with tarry masses, but both macroscopically and microscopically the entire intestine appeared normal.

Some authors assume that there is a vicarious form of intestinal hemorrhage that takes the place of menstruation in women. It is

possible that this occasionally occurs, but if it does, it is certainly very rare.

Bamberger has arranged a scale of frequency, from which it can be seen how often hemorrhage of the intestine occurs in different diseases. The most important information to be gleaned from this table, practically speaking, is that enterorrhagia is most frequent in dysentery, typhoid fever, and carcinoma of the colon, not including, of course, hemorrhages from piles, which, as every physician knows, are the most frequent of all. Other etiologic factors are less important, both as regards their significance in intestinal bleeding and the frequency with which they produce hemorrhage.

The character of the hemorrhages may vary; it may be capillary, and may be due either to intense arterial congestion or to severe degrees of venous hyperemia from stasis; in other cases the mucous membrane may be found perfectly pale. Sometimes some of the larger venous or arterial branches become eroded, chiefly in various forms of ulceration.

Markwald reports an interesting observation of Traube's, of very profuse intestinal hemorrhage in a case of typhoid, but only found minute ulcers, and no eroded or ruptured blood-vessels in them. He considers this finding a proof that it is often impossible to determine whether the hemorrhage is capillary or comes from some large blood-vessel, even though the etiology of the bleeding seems to be quite clear.

[The tendency to excessive hemorrhage during typhoid fever is correlated by Wright and Knapp[1] with diminished coagulability of the blood. Other writers consider that it is due to alcoholism or to bacterial activity (Nicholls and Learmouth).[2] In the light of some other hemorrhagic fevers the latter view seems probable.—ED.]

Hemorrhage may occur in a great variety of other conditions (diapedesis, congestion, increase of blood-pressure, changes in the vessel-walls, etc.), but it would entail too detailed a description to deal fully with all these different kinds of hemorrhage.

The anatomic changes in any given case naturally vary greatly according to the primary etiologic factors of the different diseases. The pathologic anatomy will be considered in the sections on the different lesions of the intestine. All intestinal hemorrhages, however, have the following features in common :

After very severe hemorrhages the well-known symptoms of general loss of blood appear in the body. The primary cause of the hemorrhage will determine whether or not the intestine also becomes anemic. In hemorrhage from stasis, for instance, the vessels of the mesentery and of the intestinal wall may still appear hyperemic, even though there is general anemia. Occasionally blood may be found in the lumen of the intestine, even though the hemorrhage was very insignificant. The character of this blood will depend on the date of the hemorrhage; when the hemorrhage is recent, the blood will be fresh and red; if old,

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[1] Wright and Knapp, *Lancet*, 1902, vol. ii.
[2] Nicholls and Learmouth, *ibid.*, 1901, vol. i.

dark and tarry. Under some conditions it is fluid; under others, lumpy and coagulated.

[Hemorrhage from the rectum without hematemesis may, of course, occur in ordinary gastric ulcer, but it is interesting to note that Bériel[1] has described hematemesis from the lower bowel in a case of hour-glass stomach in which there was a large gastric ulcer on the distal side of the contraction. On one occasion the patient passed, by the rectum, a clot of blood of a deep-red color weighing nearly 8 ounces.—Ed.]

SYMPTOMATOLOGY AND DIAGNOSIS.

The leading symptom of enterorrhagia is the passage of blood *per anum ;* at the same time the presence of blood in the feces does not necessarily always indicate enterorrhagia, for blood may enter the intestine from the stomach; conversely, blood poured into the upper portion of the duodenum in certain cases may be propelled backward into the stomach and be got rid of by hematemesis.

The quantity of blood passed in the stools may vary greatly. Enormous quantities may be passed and rapidly lead to death from loss of blood, or only minute traces may be evacuated. Occasionally the amount of blood in the stools is so small that it cannot be detected with the naked eye. Microscopic examination of the dejecta, however, shows the presence of red blood-corpuscles or of crystals of hematin. When such minute quantities of blood are passed in the feces, the clinical picture of hemorrhage from the intestine is not presented; but these traces of blood in the feces are of great diagnostic importance (compare the section on Ulceration of the Intestine). The quantity of blood in the stools does not by any means justify conclusions being drawn in regard to the severity of the intestinal hemorrhage, for a large proportion of the blood poured into the lumen of the bowel may remain there; death in fact may occur without any passage of blood by the rectum (death from internal intestinal hemorrhage).

The general clinical picture of profuse intestinal hemorrhage corresponds to that of other severe hemorrhages. The action of the heart and the pulse become weak, the surface of the body becomes cool, and the extremities in particular feel cold; there is pallor of the skin and mucous membranes; symptoms of acute cerebral anemia appear (vertigo, nausea, buzzing in the ears, visual disturbances, and fainting). If all these symptoms appear suddenly under circumstances that favor intestinal hemorrhage (for instance, in the course of typhoid fever or of some severe diseases of the blood), the diagnosis of enterorrhagia can usually be made before the passage of blood by the bowel, and even if death occurs before the blood can be passed with the stools. Insignificant bleeding from the intestine exercises no influence on the general clinical picture of the primary disease.

Occasionally there is a single evacuation of blood; in other instances a number of consecutive fecal evacuations are blood-tinged; in the latter case an attempt must always be made to determine whether fresh

[1] Bériel, *Lyon médical*, vol. xcii., p. 109.

blood is constantly being passed or whether darker masses of blood derived from the primary hemorrhage which has merely been temporarily retained in the bowel are being gradually passed. Intestinal hemorrhage is rarely accompanied by any sensations in the bowel, and, as a rule, the accident is not detected until the hemorrhagic character of the dejecta or some of the general symptoms enumerated above are discovered. Blood is passed either alone or mixed with intestinal contents or with pathologic intestinal secretions. These differences are of some diagnostic importance, which will be referred to when discussing the methods of localizing the exact spot where the hemorrhage occurred. Reference should be made to the sections on the various diseases of the intestine (ulceration, catarrh, neoplasm) for the conclusions that can be drawn from the presence of blood, together with pus, mucus, or shreds of tissue in the feces.

As a rule, it is possible to determine by simple inspection whether or not a suspicious color of the stools is due to the presence of blood. The color of blood cannot be confounded with anything else; it is always red, may be dark or light, and occasionally assumes a very peculiar tint. I have, for instance, seen typical orange- or paprica-colored stools after intestinal hemorrhage. Many years ago I called attention to the fact that the erythrocytes may occasionally be completely destroyed even in those portions of the feces that are red and undoubtedly contain blood. In some instances, as I have shown, this destruction of red blood-corpuscles occurs with surprising rapidity. I remember a case of dysentery in which alkaline stools were evacuated several times a day that always consisted of red material; but did not contain red blood-corpuscles. If blood remains in the intestine for some time before it is evacuated, and particularly if it has to travel to the rectum from the upper portions of the small intestine, the red blood-corpuscles are, as a rule, completely destroyed by the action of the intestinal secretions, and the hemoglobin is converted into one of its decomposition-products. Under these circumstances the evacuations are usually very dark—almost black, like tar or pitch—so that I have frequently been in doubt in such cases whether the stools really contained blood-pigment or not. A number of drugs, particularly iron and bismuth preparations, and certain articles of diet (huckleberries) may lead to confusion, inasmuch as they stain the feces very dark. Sometimes bile in the stools may make them so dark in color as to raise the suspicion of hemorrhage. If sufficient care is exercised, the presence of bile and the coloration of the stools by bile-pigment should be recognized, for the shade is somewhat different from the tint imparted by blood-pigment; it is, as a rule, brown or greenish-black. Dejecta darkened by iron are usually gray-black in color. The presence of bismuth in colored stools is shown by microscopic examination, for the preparations of bismuth usually given (the subnitrate and salicylate of bismuth) appear in the stools in typical crystals that are shaped as in Figs. 1 (bismuth subnitrate) and 2 (salicylate of bismuth). Hematin crystals, on the other hand, assume the well-known shape delineated in Fig. 3.

Spectroscopic or chemic examination will frequently reveal the presence of blood-pigment, even when all the red blood-corpuscles are destroyed and no hematin crystals are found—*e. g.*, in cases in which the blood stagnates for some time in the intestine.

As soon as the fact is established that blood is being passed by the rectum, the most practical and important question to be answered at the bedside is, What is the cause in this particular case of hemorrhage from the intestine?

In a certain proportion of cases this question can easily be answered—for instance, if hemorrhage occurs in the course of typhoid fever, in phosphorus-poisoning, in scurvy, in some one of the other hemorrhagic diatheses, in severe pernicious anemia, or in severe icterus (without portal stasis), or if it follows some abdominal injury or an extensive cutaneous burn.

The diagnosis of hemorrhages occurring in the course of some well-characterized intestinal disease will be made with the diagnosis of the intestinal lesion. Hemorrhage due to the rupture of hemorrhoidal varices can readily be diagnosed by local inspection of the affected area
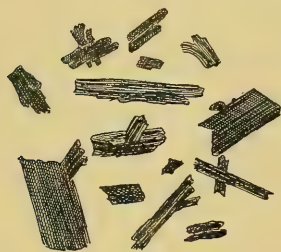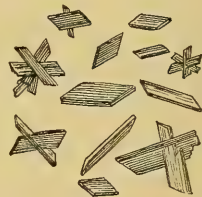
Fig. 1.   Fig. 2.   Fig. 3.

of the mucous membrane. The picture of acute dysentery is so distinct that the cause of a hemorrhage occurring in the course of this disease is never doubtful. Though rare, intestinal hemorrhage in the course of acute catarrhal enteritis is also easy to interpret, provided the typical symptom-complex of this disease is presented. Whether the cause of intestinal hemorrhage is catarrhal, tuberculous, or some less frequent form of intestinal ulceration, must be decided in the same way as the diagnosis of the character of the ulcer itself; this is done according to the different methods described in the section on Intestinal Ulceration. Mistakes in diagnosis can easily be made in these cases. Reference need only be made to the fact that in a tuberculous subject blood in the dejecta may depend either on tuberculous ulceration or on amyloid degeneration of the intestinal wall, while, lastly, hemorrhage in tuberculosis may occur even when the intestinal wall presents no structural changes recognizable macroscopically or microscopically (hemorrhages of hunger and inanition).

Hemorrhages occurring in carcinoma of the intestine are of great practical importance. The blood may either be passed as such or may

be mixed with fecal matter, mucus, or pus. Reference must be made to the section on Carcinoma of the Intestine for an account of the symptoms diagnostic of hemorrhage of the intestine in this disease.

Hemorrhage of the intestine due to venous stasis is relatively rare, and its possibility only comes under consideration when the clinical features, as a whole, show that there is some disease of the heart, liver, or the portal vein, capable of inducing venous stasis in the intestinal area. When intestinal hemorrhage occurs in a perfectly healthy person, or at least in one apparently healthy up to the time of the hemorrhage, the diagnosis as to the primary cause of the hemorrhage is frequently difficult. As a matter of fact, we must occasionally content ourselves with a tentative, probable diagnosis. Occasionally certain symptoms precede the hemorrhage that are in themselves insignificant and possibly escape detection ; for instance, pain or other abnormal sensations in the epigastrium. If, on inquiry, a history of such vague symptoms in the epigastric region can be elicited, a round ulcer of the stomach or intestine should always be thought of. Violent pain and enterorrhagia occurring suddenly in a patient with endocarditis or some valvular lesion should always suggest embolism of the mesenteric artery. Sudden intestinal hemorrhage, with symptoms of acute occlusion of the bowel should suggest axial rotation or intussusception of the bowel.

If no etiologic factor can be discovered, even after the most careful physical examination and consideration and analysis of all the features of the case, the possibility of hemorrhage from a benign tumor, an isolated varix, or an aneurysm should be thought of. Hemorrhage from a varix is more probable than hemorrhage from an aneurysm when bleeding occurs in an individual otherwise perfectly healthy and free from all intestinal disturbance ; this is particularly the case if hemorrhage from the bowel recurs at irregular intervals. When intestinal hemorrhage occurs in women without any other symptoms, so-called vicarious hemorrhage from the intestine may be assumed in exceptional instances. In the last few years I have made a discovery of great practical importance. It is that in many cases of enterorrhagia of obscure origin, in which the most careful examination was fruitless, and even a hypothetic diagnosis was impossible, in individuals otherwise healthy (apart from the enterorrhagia), the source of the hemorrhage may be distended hemorrhoidal veins high up in the rectum. It will be well to bear this possibility, which has been discussed above, in mind in all cases of doubtful etiology.

Whenever the nature of the hemorrhage is not clear, digital examination of the rectum should be made. This is hardly necessary in typhoid fever, as the source of the hemorrhage is so clear.

It is, in some cases, easy to determine the exact situation in the intestine from which the blood comes, but in some cases this is impossible. The most valuable assistance in this point of diagnosis is given by a knowledge of the etiology of the hemorrhage ; if the bleeding occurs in the course of typhoid fever, the blood is probably derived from the lowest portions of the ileum, while in dysentery the

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blood comes from the large intestine; after a burn, the hemorrhage is derived from the duodenum, etc. When no clues of this kind are available, the dejecta must be carefully examined, and it is frequently possible to arrive at a definite decision from careful analysis of the blood passed *per anum*. In some instances, of course, the conclusions to be drawn from an analysis of the feces are unreliable and of limited value. When small quantities of pure blood, that may be either light or dark, are evacuated, it is reasonably certain that the hemorrhage occurred in the lowest portions of the intestine, for in hemorrhage from the higher portions of the bowel small quantities of blood are not passed as such, but are always mixed with intestinal contents. When the blood is adherent to the external surfaces of a fecal mass, we are justified in concluding that the hemorrhage occurred in the rectum or the lower portions of the colon; when, on the other hand, the blood is intimately mixed with the feces, whether the latter are formed or unformed, it is reasonably certain that the source of the blood must be above the rectum, and probably above the sigmoid flexure. If pure blood is evacuated in large quantities, it may be derived from almost any portion of the intestine from the duodenum downward. The color of this blood in these cases occasionally allows us to draw some conclusions with regard to the exact localization of the hemorrhage, but great care should be exercised in doing this. It is true that extremely bright and arterial blood unquestionably comes from the rectum or colon in the great majority of cases; occasionally, however, if peristalsis is active and diarrhea supervenes, blood of a fairly fresh red color may be derived from hemorrhages in the ileum (typhoid ulcers) and even in higher portions of the intestine. Conversely, it is customary to conclude that hemorrhage occurred in the upper portions of the intestine when the blood evacuated is dark or tarry in color, and that the more tar-like the blood, the nearer to the stomach its source. This character of the blood, however, should be utilized with reserve, for if blood remains in the intestine for some time, the blood-pigment is changed to such a degree that the blood assumes a very dark color, so that stagnation of blood, even in the colon, may result in the feces being black and tarry. It is hardly necessary to mention, in passing, that hemorrhage *per se* does not stimulate peristalsis, since enterorrhagia may certainly be combined with constipation. When, as occasionally happens, diarrhea occurs in association with intestinal hemorrhage, the former is not due to the latter, but the diarrhea and hemorrhage are both due to the same primary cause.

Physical examination of the abdomen gives practically no help in localizing the source of the hemorrhage. It is true that some authors believe that an area of dulness or a visible and palpable accumulation of material in the intestine may be due to the presence of coagulated blood, but there are so many possible sources of error in diagnosing the presence of blood in the intestine from these physical signs that I consider it dangerous to rely on palpation and percussion; particularly as definite palpatory or percutory signs can be made out only when the

hemorrhage is very large. Further, percussion and palpation of the abdomen in intestinal hemorrhage may be harmful, and all physical examinations of the abdomen should be avoided as much as possible under these circumstances.

Occasionally fever follows severe hemorrhage, particularly if, either artificially or spontaneously, the hemorrhage is followed by a prolonged period of constipation. I have repeatedly observed a continuous rise of temperature of moderate degree under these circumstances. This fever is probably due to the absorption of septic material.

PROGNOSIS.

Loss of blood from the intestine is beneficial to the patient in certain hemorrhoidal states (see the section on this subject). The loss of small quantities of blood, for instance, in the course of acute enteritis or venous hyperemia is occasionally of no importance as regards the prognosis. Any severe loss of blood, however, must always be considered a serious event. It is true that enormous quantities of blood may occasionally be lost from the intestine without detriment to the patient, provided the general health is good. We know this to be the case from our experience in hemorrhage from ulcers of the duodenum. Naturally, severe hemorrhages of this kind may also terminate fatally. If an extensive hemorrhage occurs in the course of some disease like typhoid fever, in which the general health and the power of resistance are very much reduced, the prognosis of enterorrhagia is, for obvious reasons, very unfavorable. The same applies to small but frequently repeated hemorrhages from the intestine, even though the primary disease that causes these hemorrhages is in itself insignificant. When these small hemorrhages occur from hemorrhoidal varices, operative interference should be insisted upon.

The following observation that I have made possesses some prognostic value : When very small traces of blood are discovered in the stools of typhoid-fever patients, even if the quantities of blood are so small that they can be discovered only with the microscope (this, of course, would only rarely happen in daily practice), severe enterorrhagia should always be expected. I have often seen profuse intestinal hemorrhage occur from twelve to thirty-six hours after the discovery of these small microscopic foci of blood in the stools. When, therefore, traces of blood are discovered in the stools, the proper prophylactic measures should be taken without delay (give opium and stop baths).

When profuse hemorrhage occurs in cases where a repetition is improbable, the prognosis should still be guarded ; for even if the primary lesion responsible for the hemorrhage, as, for instance, embolism of the mesenteric artery, has exercised its effect and produced the hemorrhage, there is danger of a recurrence inasmuch as the curative thrombus may be detached or may disintegrate, and in this way a second hemorrhage may be produced in the course of the next few days.

TREATMENT.

The general principles to be adopted in the treatment of severe hemorrhage above the rectum are the following : In the first place, all factors capable of increasing the blood pressure, either by decomposing the intestinal contents or by increasing peristalsis, should be avoided. All bodily exercise should be avoided for the same reason, and absolute psychic and physical rest is an essential condition in the treatment. If the condition of the patient permits, all food should be withheld for at least twenty-four to forty-eight hours, and after this only fluid should be taken ; the diet should not be chemically or mechanically irritating in any way and should be given in small quantities. I consider the best remedy in the treatment of enterorrhagia—and I expressly include typhoid hemorrhages—to be opium, given either by the mouth or in the form of suppositories. This drug certainly fulfils the main indication in the treatment of intestinal hemorrhage—namely, arrest of peristalsis. I can only repeat and fully indorse the views expressed by Leube many years ago with regard to the treatment of intestinal hemorrhage : " The application of cold to the abdomen, either in the form of an ice-bag, ice-compresses, or a Leiter coil, is essentially valueless, for the cold cannot possibly exert any direct effect on the bleeding spot." Nothing definite is known with regard to the reflex action of cold, when applied to the abdomen, upon the blood-vessels of the intestine ; and further, this form of treatment has not stood the test of adverse criticism. As a matter of fact, I am inclined to believe that ice-compresses, when frequently applied, are apt to increase the peristaltic movements of the intestine. In addition to absolute quiet and opium, subcutaneous injections of ergot may be administered with benefit in severe intestinal hemorrhages.

The treatment of severe hemorrhage from the lowest portions of the rectum and from hemorrhoids will be discussed in the section on hemorrhoidal disease. It might be suggested that in hemorrhage from the upper part of the rectum, from the descending or the transverse colon, irrigations with ice-cold or hot water or with astringent solutions of tannin, alum, or silver nitrate would prove useful. The chief objection to this method of treatment is that these injections all tend to set up peristalsis and in this way counteract the formation of thrombi, and should therefore be applied only in desperate cases that obstinately resist all other methods of stopping the bleeding. Tampons made of iodoform gauze are occasionally inserted into the rectum for the purpose of stopping the hemorrhage, but the value of this means of treatment is very problematic, for in the first place troublesome tenesmus is produced and further flatus is retained and peristalsis excited.

Severe anemia or collapse must, of course, also be treated. In these cases warm bottles, counterirritants applied over the heart or injected subcutaneously, and transfusion with physiologic salt solution, should all be used in addition to measures which directly stop further hemorrhages.

The same general rules apply to slight, frequently repeated hemor-

rhages—namely, rest, opium, etc. In addition a number of other reme-
dies can be tried. When the hemorrhage occurs from the rectum, they
should be given by enema; when from other parts of the bowel,
by mouth. I consider subnitrate of bismuth in doses of one gram
three to ten times daily to be relatively the most efficient of all these
drugs. Tannin, alum, and acetate of lead are less effective than sub-
nitrate of bismuth, and, in addition, interfere with digestion when given
for a long time. [The administration, by the mouth, of calcium chlorid,
30 grains, in repeated doses, or an enema containing a dram of calcium
chlorid in a few ounces of water, should certainly be tried.—Ed.]
[Graeser [1] has given adrenalin hydrochlorate by the mouth with good
effect when all other means failed to stop repeated hemorrhages in
typhoid fever.—Ed.] Liquor ferri perchloridi is quite fallacious, for
it acts as a hemostatic only when it comes in direct contact with the
bleeding vessel; in the case of intestinal hemorrhages this is quite im-
possible, for the few drops of chlorid of iron that are given are too much
diluted by the time they reach the lowest portion of the ileum to do any
good. Chlorid of iron may occasionally exert an indirect hemostatic
action in intestinal hemorrhage by causing coagulation of masses of
liquid blood present in the intestine, the clot acting like a tampon. In
isolated cases ergot or hydrastis canadensis is useful. When hemorrhage
occurs from the rectum and the lowest portion of the colon, the remedies
described above in discussing the treatment of severe hemorrhages can
be applied by the rectum.

It need hardly be mentioned that every case of intestinal hemorrhage
should be kept under observation for a long time after the hemorrhage
stops, and that these patients should be extremely careful, especially as
regards their diet.

THE URINE IN DISEASES OF THE INTESTINE.

CHANGES in the constitution of the urine occur only in a very lim-
ited number of intestinal diseases and only under definite conditions.
Some of the changes observed, however, are so peculiar and so charac-
teristic that they deserve special notice here. It need hardly be men-
tioned that there is a certain interdependence between the functions of
the intestine and those of the kidneys as regards the excretion of water.
The quantity of urine, for instance, will be reduced in proportion to
the quantity of fluid excreted into the intestine (this is well illustrated
by the scanty excretion of urine in cholera Asiatica and nostras). The
chemic composition of the urine is naturally influenced by disorders of
digestion and absorption in a variety of intestinal diseases. These
details will not be exhaustively considered here because they refer more
or less to purely physiologic laws and events.

Sometimes albuminuria and casts in the urine, the latter more espe-
cially, arise from intestinal disease. Fischl first called attention to a
form of cylindruria arising in recent simple intestinal catarrh, even

[1] Graeser, *Münch. med. Wochenschr.*, July 28, 1903.

when uncomplicated by fever or by collapse. It occurs when copious evacuations follow each other in rapid succession. The casts can frequently be demonstrated a few hours after beginning of the diarrhea, and disappear again very rapidly. At times they are exceedingly numerous, at times scant. Hyalin appears almost without exception ; at times renal epithelial cells in process of degeneration, also white blood-cells, but red blood-cells very infrequently. The urine may contain albumin or be entirely free from it. Stiller described a form of mild albuminuria in addition to cylindruria as a frequent concomitant of simple acute gastro-intestinal catarrh. Fischl, Stiller, and Runeberg attributed these appearances to lowered arterial blood-pressure. Singer, on the contrary, considers the cause to be a disturbance of nutrition in the kidneys occasioned by the arterial anemia. In many cases the cause may be the toxin which produced the catarrh. Kobler has observed quite as grave renal symptoms in cholera nostras as in Asiatic cholera, and has also found that, in both acute and subacute intestinal affections combined with obstinate constipation, hyaline casts, cylindroids, and renal epithelium may appear in the urine independently of albuminuria ; red and white blood-corpuscles also occur at times. All disappear from the urine with the cessation of constipation. In all cases pain was present, and this Kobler regards as the chief factor in the renal symptoms (reflex contraction of the renal vessels, with transient nutritional disturbances in the renal epithelium). In addition damage done to the kidney by decomposition-products absorbed from the intestine must, as pointed out by Stiller, be taken into account. Investigations by Richter conducted in my clinic failed to establish any uniform relation between the pain and the presence of casts, which were absent in cases with extreme pain, such as malignant obstruction of the bowel.

Albuminuria and cylindruria in strangulated hernia (Englisch, Frank, Senator) must be mentioned here. It is true that albuminuria is not constant in these cases, but it is sufficiently frequent to justify the conclusion that a causal relation between them must exist. It is often, if not always, present in internal strangulation, and is accompanied by hyaline and granular casts. [In 231 cases of strangulated hernia albuminuria occurred in 6 per cent., and when death occurred, parenchymatous nephritis was found (Bundschuh[1]).—Ed.] Israel even observed typical hemorrhagic nephritis with numerous blood-casts in a case of sigmoid volvulus. As a rule, the more complete the obstruction, the greater the albuminuria, which, as it generally disappears rapidly after relief of the obstruction, must be explained in the same way as in acute constipation. Wallerstein produced cylindruria and albuminuria by occluding the anus of dogs and rabbits.

The excretion of indican and other conjugate ethyl-sulphuric acids is an important characteristic of the urine in intestinal diseases.

No detailed consideration of the physiologic-chemic aspects of indicanuria will be attempted ; the following brief statement will suffice : The indican in the urine (potassium indoxyl sulphate) is derived from

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[1] Bundschuh, *Beitrag z. klin. Chir.*, vol. **xxxi.**

indol. The latter substance is, under ordinary conditions, formed exclusively within the intestinal canal, chiefly in the small intestine. It is a product of the putrefaction of proteids. Kühne and Nencki have demonstrated—and I believe their view is now universally accepted— that indol is a product of *bacterial* putrefaction of albumin. Jaffe was the first to recognize that the quantity of indol formed in the intestine, and consequently the amount of indican excreted in the urine, depends on the amount of nitrogen ingested, so that the excretion of indican is approximately proportionate to the quantity of nitrogen contained in the bowel contents. Jaffe's views have been fully corroborated and are now universally accepted. During inanition a certain amount of indican is also found in the urine, which, Salkowski believed, was derived from the catabolism of the body proteids; but it has been shown by F. Müller that it is also derived from the putrefactive changes in the nitrogenous secretions and excretions of the "feces of hunger." Occasionally decomposition of pus leads to the excretion of indican in the urine; suppuration, therefore, must be considered another possible source of indican.

Ortweiler, relying on his own experiments and the investigations of a number of other authors, postulated three conditions under which the excretion of indigo is increased. In the first place, a sufficient quantity of albuminous material must be present in the intestinal canal; in the second place, the proteids in the intestinal canal must undergo more marked putrefaction than normal; and in the third place, the indol formed must be absorbed in sufficient quantity.

These facts lead us to assume that possibly diseases of the digestive canal, especially of the intestine, might lead to quantitative alterations in the formation of indol, and in this way cause an increased excretion of indican in the urine. As a matter of fact, this view is correct. Consideration will be limited to the rôle played by diseases of the intestine in the production of indicanuria, and the influence of gastric disorders will be omitted.

Carter was the first to call attention to the fact that intestinal obstruction may lead to indicanuria. Jaffe later studied this relationship carefully, both clinically and experimentally. DeVries, Henninge, Senator, Leube, von Jaksch, and more especially Ortweiler investigated this subject. I myself have paid particular attention for a number of years to the clinical aspects of indicanuria. The following is a summary of our knowledge of this subject.

All authors agree that occlusion of the small intestine, whatever its cause, produces a great, occasionally an enormous, increase in the urinary excretion of indican. Jaffe determined that 100, even 150, mg. were excreted in one day—that is from 10 to 15 times more than normal; the normal amount of indigo excreted in human urine fluctuates between 5 and 20 mg. a day. By experiments on animals he found that the amount of indican was increased within twenty-four hours after ligation, but did not reach its maximum until the second or third day. In examining the urine of human subjects with occlusion of the bowel

I also failed to find an increased excretion of indican during the first twenty-four hours; this observation corresponds with Jaffe's experimental results. If the obstruction is removed a few hours after the onset,—for instance, when a strangulated hernia is relieved without delay,—no increased excretion of indican occurs. This is due to the fact that in cases of this character where the obstruction of the bowel lasts for a very short time, one of the conditions for the formation of indican is not fulfilled—namely, advanced putrefaction of albumin does not occur in the intestinal canal.

Obstruction of the large intestine in animals (dogs) leads to no increase of the excretion of indican (Jaffe). Any increase at all is quite insignificant in comparison with that produced by obstruction of the small intestine. Jaffe, however, conservatively adds that an increased excretion of indican might possibly occur in long-standing obstruction of the large intestine, and that this may happen even though the small intestine is not manifestly involved, for under these conditions the prolonged obstruction in the large intestine would lead to stasis of bowel contents throughout the whole intestinal tract, and in this way lead to an increased formation of indol and to indicanuria. This view corresponds essentially with the actual facts observed. If the colon is occluded for a long time, marked indicanuria may be seen, especially when the contents of the colon stagnate above the ileocecal valve. (For the clinical details, the reader is referred to the section on Occlusion of the Bowel.)

In simple constipation, even when very obstinate, Jaffe failed to find large quantities of indican in the urine. De Vries, Ortweiler, and I have published similar negative observations bearing out this view. Brieger and Ortweiler produced constipation with opium and obtained the same results. Von Pfungen found that in simple constipation the amount of conjugate sulphates excreted in the urine rose or fell in proportion to a variety of factors that were operative in the intestine, but that the excretion of indoxyl-sulphuric acid did not run parallel to the excretion of the other ethereal sulphates.

In diffuse peritonitis indicanuria may be extremely marked, particularly in the acute purulent form and also in the chronic form. This fact was also discovered by Jaffe and was later corroborated by Senator, Brieger, Henninge, Ortweiler, and myself. In this disease the onward passage of the contents of the small intestine is interfered with in a variety of ways, and the same conditions are created for the formation of indol as in occlusion of the small intestine.

Indicanuria is not only found in diseases characterized by constipation or stasis of the bowel contents, but also in a variety of intestinal diseases complicated with diarrhea—for instance, typhoid fever, cholera Asiatica and nostras, tuberculous ulceration, and simple catarrh of the intestine. This applies, however, only to those diseases that are strictly localized in the small intestine, and not to dysentery and catarrh of the large intestine. This distinction, however, is only apparent, for, as Ortweiler has shown, the increased excretion of indican is not due to

increased peristalsis in these cases, but to alterations in the mucosa of the small intestine and the increased putrefaction of intestinal contents that results therefrom. Simple increase of the peristaltic movements of the intestine without lesion of the small intestine (such as is produced by laxatives) not only does not lead to an increase of indican in the urine, but may even cause a diminution of the urinary indigo or its complete disappearance. This is due to the fact that the normal amount of indol formed in the intestine is so rapidly expelled in the feces that a smaller quantity than normal is absorbed.

In discussing indicanuria, another urinary reaction must be mentioned, discovered by O. Rosenbach. It consists in boiling the urine after the addition of a few drops of nitric acid; if this is done, a Burgundy-red color occasionally appears (indigo red). Rosenbach is inclined to attach special prognostic importance to this sign, for he claims that it occurs only in serious intestinal diseases and in profound nutritional disturbances, and consequently always points to a serious prognosis. Rosenbach's statements and experiments have been repeated by a number of authors (Salkowski, Ewald, Abraham, Rumpel and Mester, von Jaksch), all of whom arrived at the decision that the clinical deductions drawn by Rosenbach from his urinary reaction are essentially invalid. They all found that a positive indigo-red reaction signifies the same as indicanuria, and merely demonstrates that the specimen of urine contains an abundant quantity of substances that can produce indigo.

The excretion of other conjugated ethereal sulphates (skatoxyl-, parakresol-, and phenol-ethereal sulphates) is not so important from a practical point of view as the excretion of indican. The same factors determine the increase and the decrease in the excretion of all these bodies, and the same tendencies must be at work to produce an increased excretion of ethereal sulphates as to produce an increased formation of indol in the intestine and indicanuria; consequently we are only justified in drawing the same clinical conclusions from the appearance of these bodies in the urine as from the appearance or the increase of indican. The methods of determining their presence in the urine qualitatively and quantitatively are so much more complicated than the methods of analysis for the indican in the urine that the practical significance of determinations of the conjugated ethereal sulphates is comparatively small.

[According to Herter,[1] the relations between the presence of indican and of the ethereal sulphates in the urine fall into three groups—(1) In which they are both largely increased: he produced this relationship by injecting large amounts of the common colon bacillus into the intestine of dogs. (2) In which the ethereal sulphates are greatly increased while indican is absent; experimentally this result followed the introduction of large quantities of Proteus vulgaris into the intestines of dogs. (3) Where indican is markedly increased, but the ethereal sulphates are normal. His observations show that the colon bacillus plays a very important part in the production of indol and indicanuria. It is

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[1] Herter, *Lectures on Chemical Pathology.*

well to remember that the use of some aromatic antiseptic drugs, such as salol, salophen, creasote, etc., give rise to a marked increase in the ethereal sulphates in the urine.

Pearce Gould [1] reported a case of acute intestinal obstruction which proved fatal from intestinal toxemia nine days after operation. The urine contained a great excess of indican, and, in addition, another chromogen yielding a brown pigment, which was decolorized by nitrous acid and ammonium sulphid.

Cystinuria, which is intimately related with the excretion of diamins, such as cadaverin and putrescin, has been thought to be the result of bacterial decomposition of proteids in the alimentary canal (Baumann); but Garrod [2, 3] believes that, like alkaptonuria, the presence of cystin in the urine may be due to an inherent peculiarity in the metabolism in the individual, or, in other words, is a chemic "sport," and not due to bacterial influence.

Ehrlich's diazo-reaction is rarely absent in tuberculosis, and so may be of use in the diagnosis of tuberculous ulceration of the intestines, since it is almost always absent in intestinal carcinoma (Krokiewicz [4]).—ED.]

Lorenz, a number of years ago, published a series of investigations from my clinic in which he showed that the urine frequently contains acetone and diacetic acid in certain disturbances of digestion, and that the former body appears more frequently in the urine than the latter.

Disturbances of intestinal function (functional disorders of the stomach are not referred to here), when severe, may be due to a variety of causes : they may either be primary or may be dependent on other diseases, and this fact alone demonstrates that the digestive disturbances *per se* must be considered the cause of acetonuria and diaceturia. Occasionally in these cases acetone has also been found in the feces, and in rare instances in the vomit. Lorenz found acetonuria in all cases of acute gastro-enteritis. In general, the excretion of acetone, as well as that of diacetic acid, was proportionate to the intensity of the other intestinal symptoms. He also found acetonuria and diaceturia in wide-spread intestinal ulcerations, in intestinal obstruction, and in intestinal disturbances due to the presence of teniæ; he also noted its presence in isolated cases of peritonitis complicated with vomiting and diarrhea. Lorenz, therefore, adds a special form of acetonuria to those already recognized—namely, " acetonuria in digestive disorders."

[The presence of acetone in the urine of patients with gastric ulcer while on rectal feeding was regarded by Payne [5] as due to virtual starvation, and not to any perversion of gastric digestion. Diacetic acid, as shown by Gerhardt's test with Fe_2Cl_6, is almost always present in the urine in cases of rectal feeding, and disappears when ordinary feeding by

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[1] A. Pearce Gould, *Trans. Clin. Soc.*, 1898, vol. xxxi., p. 47.
[2] Garrod, *Lancet*, December 13, 1902, vol. ii., p. 1616.
[3] Garrod and Cammidge, *Jour. Path. and Bacteriol.*, vol. vi., p. 327.
[4] Krokiewicz, *Wien. klin. Wochenschr.*, 1898.
[5] Payne, *Brit. Med. Jour.*, 1900, vol. ii., p. 885.

the mouth is resumed. It is interesting to note that although the urine contains a considerable amount of diacetic acid in these cases, there is an absence of the symptoms of acid intoxication. Gerhardt's test consists in adding to the urine a few drops of a solution of ferric chlorid; this precipitates the phosphates, etc., which are filtered off; a further addition of the solution of ferric chlorid to the filtered urine gives a Bordeaux-red color. This color-test must be distinguished from the dark purple color developed on adding a solution of ferric chlorid to the urine of patients taking salicylates and some other drugs. In both cases the color is prevented by citrate of potash. By heating the urine, diacetic acid is transformed into acetone, which does not give Gerhardt's reaction. As a result of excessive fermentation of carbohydrates the urine may become either more acid or less so. Increased acidity may depend on the formation of fatty acids, which, by attaching to themselves the available bases, lead to an increase in the acid phosphates and sulphates. Herter [1] has found acetates and formates in the urine of patients with excessive fermentative process; and oxalates have been shown by H. Baldwin [2] to appear in the urine as the result of carbohydrate fermentation.—ED.]

CATARRH OF THE INTESTINE (Catarrhus Intestinalis; Enteritis).

CATARRH is by far the commonest disease of the intestinal canal. This condition is very important not only because it is so frequent, but because it is often so obstinate that it seriously impairs the health of the patient, and, in certain forms and under certain conditions, particularly in early childhood, may directly endanger the life of the sufferer. In view of these facts I feel justified in describing this disease, which, apart from catarrh of the bronchi, may be regarded as the most frequent affection met with in practice, at great length.

From a pathologic point of view it is specially important to differentiate between independent and primary catarrhal states, on the one hand, and symptomatic secondary forms, on the other. I think, however, that the value of a description of these conditions will be greatly enhanced and will be more comprehensive and simple if the most important feature at the bedside—namely, the clinical course of the disease—is described in particular. The consideration of the symptomatology of catarrh of the intestine will be divided into two parts, dealing with the acute and the chronic form.

ETIOLOGY.

From a general survey of the almost unlimited number of factors which can produce catarrh of the intestine it appears that the great majority of cases of catarrh of the intestine are primarily due to chemic

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[1] Herter, *Brit. Med. Jour.*, 1897, vol. ii., p. 1847.
[2] Helen Baldwin, *Jour. Exp. Med.*, October, 1900.

irritants. The chemic substances in question are either poisons intro-
duced directly in food, or derived from the food in the gastro-intestinal
canal; or, lastly, poisons present in the blood and circulating through
the blood-vessels of the intestinal wall. These forms of catarrh
may be considered as due to intoxication. In another large number
of cases the catarrhal changes are due to the irritation of pathogenic
micro-organisms; the latter either act directly or form chemic poisons
that act as above. These forms will be spoken of as infective catarrh.
The great majority of cases of intestinal catarrh, both primary and
secondary, may be etiologically grouped under these two heads; very
few cases indeed are due to other causes.

TOXIC CATARRHAL ENTERITIS.

(*a*) To the group of chemic irritants introduced directly belong all
chemic bodies capable of damaging mucous surfaces, provided they
enter the intestine in a sufficiently concentrated form; for instance, cor-
rosive alkalis (even when in combination as salts, like sodium chlorid
or carbonate of sodium); acids (inorganic and organic); many of the
salts of metals (copper, lead, iron, silver, mercury); metalloids and
some of their compounds (arsenic, antimony, phosphorus); alcohol,
chloroform, ether; many drastic purgatives (senna, jalap, podophyllum,
colocynth, gamboge, croton oil); many derivatives of benzene (carbolic
acid, picric acid, chrysophanic acid, thymol, creasote); aromatic acids
(benzoic, salicylic, tannic acid); the volatile ethereal oils, camphor, tur-
pentine, and a large number of substances that act through the aromatic
compounds, chiefly terpenes, which they contain; among these may be
mentioned balsam of Peru, many spices, particularly the different varie-
ties of pepper, balsam of copaiba, etc., mustard, garlic, cantharides,
some of the alkaloids (particularly colchicin, emetin, veratrin, aconitin).
Many of these irritants directly damage the mucosa, and usually pro-
duce serious lesions; it is only when their effect is slight that the
morbid appearances of an uncomplicated superficial catarrh of the
intestine result.

(*b*) The chemic irritants present in the food. This group, like the
preceding one, is important in the etiology both of diseases of the stom-
ach and of diseases of the intestine. The irritants belonging to this
category produce the majority of intestinal catarrhs, so that it is of great
practical importance to study this question thoroughly, as every medical
man is in almost daily contact with these cases.

Food may produce catarrh of the intestine in a variety of ways.
The food itself may be perfectly good, but does harm merely from the
fact that excessive quantities are taken. When this occurs, the diges-
tive powers of the intestinal secretions become insufficient and a large
proportion of the excessive pabulum remains undigested. This factor
is important and should always be looked for, as it is a frequent cause
of trouble in adults and in half-grown children, and may also occur in
infants. In other cases the food in itself is good and the quantity taken

is moderate, but it acts injuriously on the intestine, owing to some un-explained individual idiosyncrasy of the patient. The most conspicuous example of this kind is milk, an article of diet which constantly pro-duces catarrh of the intestine in some persons, whereas in others it pro-duces the opposite effect—viz., constipation. The most remarkable results occasionally occur in some individuals who eat a variety of harmless articles of food. Again, some articles of food are perfectly harmless when they are eaten alone, but produce gastro-intestinal catarrh as soon as they are taken in combination with other articles of diet (for instance, beer and fruit). It appears that in this instance toxic combinations are formed. Lastly, from the presence of slight gastro-intestinal disturbances, articles of diet which are otherwise diges-tible cannot be disassimilated properly, and thus produce catarrh. This may be considered the most prolific cause of catarrh. The direct primary effect of all these factors is an attack of indigestion that does not necessarily lead to the development of a true catarrh in every case; at all events, it is impossible in many instances positively to diagnose catarrh from the clinical symptoms present. If the effect of the irri-tant is slight and not too intense, simple diarrhea supervenes. We can never determine, however, whether this is merely dyspeptic diarrhea or whether it is due to some structural changes of the nature of slight catarrhal lesions of the intestinal mucosa (compare the section on Diarrhea).

In all the cases that we have enumerated the substances introduced are harmless in themselves; at the same time the production of dys-peptic symptoms by these agencies may favor the development of nu-merous bacteria and fungi normally present in the gastro-intestinal tract. In this way the formation of abnormal fatty acids, gases, and other abnormal substances is favored; or some of the ordinary decom-position-products of bacteria are generated in excessive quantities, so that intestinal peristalsis is accelerated and inflammatory conditions develop.

Different from the group just considered are those catarrhs due to the ingestion of articles of food or of liquids that are injurious in them-selves. Cases of gastro-intestinal catarrh often develop after eating tainted meat, fish, fruit, vegetables, dishes made of flour, beer, wine, particularly milk and water. In a number of these cases ptomains, toxalbumins, organic acids, and other injurious substances are ingested directly with the food; in other cases again bacteria swallowed with the food or drink develop in the intestine and exert their pathogenic effects there.

(c) The chemic irritants exert their effect on the intestinal mucosa through the blood circulating in the intestinal wall. The best-known examples of this kind of catarrh are the so-called uremic catarrh of nephritis and the mercurial catarrh seen in certain cases of mercurial poisoning (poisoning with sublimate). J. Fisher has shown that the clinical picture of uremia is not uniformly present in nephritic catarrh. In 7 cases in which both macroscopic and microscopic examinations

showed a true catarrhal state in the large bowel, and to an extent also in the small bowel, uremia was established clinically in only 4.   I have had 307 cases of Bright's disease in my clinic in ten years.   Of these, 89 had diarrhea and 45 had constipation.   Of the 89 diarrheal cases, 43 were fatal.

It is possible that the hyperemia and the inflammatory changes seen in the intestine after extensive cutaneous burns belong to the same category.   Kijanitzin has described a poisonous ptomain that he succeeded in isolating from the blood and organs of animals with extensive cutaneous burns.   He claimed that this ptomain injected into other animals produced the well-known internal symptoms seen in burns —among others, diarrhea with the passage of blood.   Kijanitzin, it is true, did not observe any structural changes in the intestinal wall of the animals that he experimented on (compare also the section on Ulcers of the Intestine from Burns).

INFECTIVE CATARRHAL ENTERITIS.

A distinction must be drawn between the two groups of intestinal catarrh caused by micro-organisms or by the poisons they secrete.   In one group the affection of the intestine is only one of the symptoms of a general infection or one of a number of local effects of the same poison.   In the second group the pathologic action of the micro-organisms is limited to the intestine, or, more strictly, the stomach and intestine.

In some infectious diseases intestinal complications, either catarrh or ulceration, are the rule (typhoid fever, cholera).   In other diseases these complications are frequent (sepsis, influenza, pneumonia, recurrent fever), while in others they are very rare (scarlatina, measles, malaria, rheumatism).   Catarrh of the intestine may occasionally be seen in any acute infectious disease.   Among the chronic infections, tuberculosis is the most liable to become localized in the intestine, the local infection in this disease manifesting itself in the form either of a catarrh or more frequently of ulceration.

The second group in which the action of microbes is limited to the intestine can be subdivided into two classes.   A distinction can be made between micro-organisms that produce catarrh of the intestine indirectly and micro-organisms that produce it directly.   The bacteria and fungi that decompose the intestinal contents so as to produce chemically irritating products, organic acids, etc., produce catarrh indirectly.   In the section on the Bacteria of the Intestine we saw that in the majority of cases catarrhal diseases of the intestine are not produced by specific pathogenic micro-organisms.   In the great majority of cases, therefore, the catarrh is due to the indirect action of the ordinary intestinal bacteria ; at the same time there is no doubt that there are certain forms of microbes that are directly pathogenic for the intestine and are capable of producing enteritis (for particulars see p. 44).

So-called summer diarrhea belongs to the same category.   It is a

disease that occurs in the height of summer, attacks adults and children, and may assume an epidemic character. It would be quite unnecessary to enumerate all the older views in regard to the etiology of this *diarrhœa œstiva*—for instance, the theory that it is due to some " perversion of the liver function " (diarrhœa biliosa) or that it is due to eating too much fruit, etc. It is now recognized that certain microbes produce this disease, and it is probable that in the summer they develop more rapidly than at other seasons of the year, owing possibly to the higher temperature of the air.

[Booker [1] found that no single micro-organism is the specific cause of the summer diarrhea of infants, but that this affection is generally to be attributed to the result of the activity of a number of varieties of bacteria, some of which belong to well-known species and are of ordinary occurrence, the most important being Proteus vulgaris and streptococci. He found Proteus vulgaris in the feces of more than half the severe cases, and seldom in the mild cases. Cases of streptococcal enteritis have been often described (De Cerenville,[2] Beck, Tavel and Eguet,[3] Washbourn and Pakes [4]). De Cerenville's and Tonarelli's [5] cases resembled in general atypical typhoid fever. Tavel and Eguet distinguished two forms, one probably due to the absorption of toxins from the intestine and resembling cholera, the other form due to a general streptococcal infection. In a case described by Andrewes,[6] Klein's Bacillus enteritidis sporogenes was probably the cause of the original diarrhea, but a secondary streptococcal infection took place and was regarded as responsible for great congestion and some thickening of the walls of the small intestine. An epidemic of acute ileocolitis in children has been described by Cautley,[7] in which the only micro-organisms found by Andrewes were the colon Bacillus and the Bacillus enteritidis sporogenes.—ED.]

Microbes may enter the intestine in many ways. It is important, from a practical point of view, to determine how the microbes gained an entrance into the body, for the determination of this fact gives valuable information as to the etiology and the probable character of the catarrh of the intestine. The germs, for instance, may enter in the water that is drunk, in solid articles of food that are eaten, and occasionally in the air that is swallowed.

A form of intestinal catarrh related to the preceding class is that produced by entozoa. For a discussion of this form the reader is referred to the section on Diarrhea.

Cold—" catching cold "—plays an important rôle in the etiology of catarrh of the intestine (so-called *enteritis rheumatica*). This has been discussed in the section on Diarrhea, and it is only necessary here

[1] Booker, *Johns Hopkins Hosp. Repts.*, vol. vi.
[2] De Cerenville, *Ann. Suisse des Sciences Méd.*, 1895.
[3] Tavel and Eguet, *ibid.*, 1895.
[4] Washbourn and Pakes, *Brit. Med. Jour.*, 1898, vol. i., p. 1578.
[5] Tonarelli, *La Riforma Med.*, 1896.
[6] Andrewes, *Trans. Path. Soc.*, vol. l., p. 118.
[7] E. Cautley, *Trans. Med. Soc.*, vol. xxiv., p. 206.

to emphasize again the fact that true catarrh of the intestine can undoubtedly follow exposure to cold; as a rule, these attacks of diarrhea occur without any structural changes in the intestine that may be considered characteristic of catarrh. Intense cold applied directly to the gastro-intestinal canal may also produce catarrh of these parts; catarrhal inflammation, *e. g.*, has been known to follow the excessive use of ice or the ingestion of large quantities of ice-cold liquids.

Mechanical irritants may also produce catarrh of the intestine. The rôle of this class of irritants, however, is, etiologically speaking, insignificant and subordinate in importance to the others already enumerated. A few cases are on record in which it has been claimed that injury of the external abdominal walls produced catarrh of the intestine. The proofs adduced for this connection are, however, very incomplete.

When mechanical irritants, however, come in contact with the intestinal mucosa, catarrh may undoubtedly be produced; for instance, when stagnating hard fecal masses mechanically irritate the mucosa of the intestine and undoubtedly produce inflammation and even ulceration (compare the sections on Constipation and Ulceration). All other mechanical irritants, as foreign bodies that are swallowed, gall-stones, various solid articles that are introduced into the rectum, act so rarely on the intestine as compared with scybala that these forms of irritation must be considered exceptional. As a matter of fact, it is doubtful whether such foreign bodies can produce catarrh of the intestine, for it is a well-known and rather astonishing fact that the intestine seems to react only very slightly to foreign bodies. It is anatomically interesting to recall that this form of enteritis, moreover, usually remains purely local and circumscribed.

Occasionally catarrh complicates other diseases of the intestine. In this case, of course, the catarrh is merely a secondary symptom and is usually an insignificant complication of the primary disease. This form of catarrh is seen, for instance, in carcinoma, in stenosis of the intestine, and in intestinal obstruction. Many forms of peritonitis are also complicated by catarrh. Occasionally intestinal catarrh complicates inflammatory processes that are localized in the vicinity of the intestine.

Venous engorgement is usually considered an important factor in the etiology of intestinal catarrh. It is almost universally believed that diseases of the heart and the liver which produce venous hyperemia of the intestine can also produce catarrh of the intestine. This view, however, is not correct. My reasons for opposing this view are given in the section on Venous Hyperemia of the Intestine, to which the reader should refer. The only point that I am willing to concede is that venous stasis may be a predisposing factor for the development of catarrh, provided other tendencies are operative that can produce such a change; venous stasis, alone, however, can never produce catarrh.

The intestine of children seems to be particularly vulnerable to the various irritants enumerated. It is a well-known fact that catarrh of the intestine is enormously frequent in the earliest years of life, and particularly in infancy. There is a popular prejudice to the effect that

dentition produces a certain predisposition to catarrh of the intestine; some authors even go so far as to speak of a diarrhea from dentition. The laity believes that the occurrence of diarrhea acts beneficially in dentition and that it is the best prophylactic against the occurrence of cerebral symptoms during that process. Kassowitz has recently investigated this question and is fully in accord with some of the older authors, who positively deny any connection between dentition and diarrhea. Kassowitz calls attention to the fact that the statistics in regard to the age of children in which catarrh of the intestine is most frequent fail to reveal any connection between the occurrence of diarrhea and of dentition. The figures obtained, as a matter of fact, directly negative the dentition theory. Another fact that militates against this view is the relative infrequency of so-called diarrhea from dentition during the winter months. Kassowitz is strongly inclined to consider this form of diarrhea to be alimentary in character.

The significance of warm seasons in the development of catarrh of the intestine has already been briefly referred to. Warm weather is a predisposing factor, for the increased temperature of the air favors the development of certain microbes and creates ideal conditions for their growth. It is possible that the greater frequency of diarrhea in warm climates (tropic diarrhea) can be explained in this way.

[In the recent South African war diarrhea was extremely frequent and attacked nearly every fresh arrival in the country; besides improper food and drink, infection of food by flies, rapid variation in the day and night temperatures, the irritating effects of sand and dust in the food, were probable factors.—Ed.]

Some time ago I called attention to the fact that chronic catarrh of the intestine seems to be hereditary. Whether the cases I have observed were merely coincidences, or whether a greater vulnerability of the intestinal mucosa can really be transmitted by heredity, must for the present remain undecided, as the number of observations is much too small to enable me to draw any definite conclusions.

PATHOLOGIC ANATOMY.

Catarrh of the intestine may involve long or short portions of the bowel; in some instances a single distinctly circumscribed portion of the intestine is involved; in other cases the whole intestine throughout its length and in addition the stomach are affected. These differences in the extent of the catarrhal process are directly dependent on the intensity of the inflammation and its etiology. The most interesting forms of catarrh are the primary independent ones. It is generally known that the colon is most frequently affected primarily, and in many instances is the only portion of the intestine that becomes inflamed. Catarrhal inflammation confined to the small intestine does not seem to occur. Such experienced observers as Woodward, for instance, deny the existence of such a condition. I have, however, repeatedly seen at necropsy catarrh of the jejunum and ileum. In some of the cases that

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have come under my observation the process seemed to stop suddenly and abruptly at the ileocecal valve. Generally, however, the large and the small intestine are involved together, either in small portions of the intestine or in considerable areas. The nomenclature of catarrh of the intestine has been arranged according to the seat of the inflammation ; thus we speak of duodenitis, jejuno-ileitis, typhlitis, appendicitis or scolecoiditis,[1] colitis, and proctitis.

In acute catarrh the mucosa of the intestine is reddened either uniformly or in spots. The color varies from light rose to dark purple. Around the follicles and plaques, on the summit of the valvulæ conniventes and on the villi, we usually find the same severe degrees of discoloration. When the inflammatory process is very severe, extravasations of blood are occasionally seen. At the same time the mucous membrane is swollen and sometimes edematous. The mucosa is very frequently covered with mucus that is glassy in character, stained by bile or blood, or is more or less opaque. In the latter instance the cloudiness is due to formed elements that are mixed with the mucus. On microscopic examination a large number of desquamated epithelial cells, and occasionally a small number of pus-cells, will be found. Sometimes considerable areas of the epithelium desquamate and form gray shreds lying in close proximity to the intestinal wall. The fecal contents of the intestine are usually liquid ; this is due in part to the exudation of large quantities of fluid from the inflamed intestinal wall, in part to the great excess of digestive secretions (bile, pancreatic and intestinal juice) that are rapidly propelled onward through the intestine by the increased peristaltic action of the bowel. The villi and solitary follicles are usually swollen. The latter are prominent and distinctly visible as whitish nodules surrounded by an injected area (enteritis follicularis seu nodularis). Peyer's patches are rarely involved to any great extent in idiopathic acute catarrh of the intestine. As in acute catarrh of other mucous membranes, there may be no signs of inflammatory hyperemia postmortem, even though the degree of hyperemia and inflammation was very severe during the life of the patient. The peritoneum and the mesenteric glands usually remain intact in idiopathic acute catarrh of the intestine ; they only become involved in certain forms of specific or secondary catarrh (compare typhoid, tuberculosis, etc.).

Microscopic Appearances.—The blood-vessels of the mucosa and usually of the submucosa are more or less distended with blood. Occasionally small extravasations can be seen, chiefly between the crypts of Lieberkühn. The interstices between these glands are frequently wider than normal and contain more or less abundant masses of round-cells ; the latter appearance may be considered characteristic of true inflammatory catarrh. Round-cells are also abundant in the most superficial part of the submucosa, immediately under the muscularis

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[1] Appendicitis will not be dealt with here. This form of catarrh of the intestine will be described in the section on Diseases of the Peritoneum, inasmuch as its clinical significance is due to the peritonitis that complicates this disease.

mucosæ, and in the deeper layers of the submucosa, chiefly around the blood-vessels. The swelling of the solitary follicles—if they are swollen at all—and of Peyer's patches is probably due to proliferation of the essential cells of these glandular structures and in part to immigration of round-cells. The epithelium of the mucosa in almost all cases is detached, particularly in the large intestine. This I regard as merely a postmortem phenomenon, and I have performed a number of control experiments in animals that strengthen this view. This separation of the epithelium is found in every dead body if the examination is made later than six hours after death. The character of the disease from which the patient dies is immaterial. Whether or not this desquamation of epithelium is favored during life by the existence of a catarrh cannot be positively determined, but it seems probable that this is the case. The epithelium of the mucous membrane of the intestine is undoubtedly involved in the catarrhal process; this is shown by examination of the epithelial cells in the stools in catarrh, for they are always more or less degenerated (see below). The epithelial cells of the glands may either be intact or be cloudy and swollen. Lieberkühn's crypts often present an interesting picture (compare Atrophy of Intestine). Even in cases of very acute catarrh the position and the outline of these structures are frequently changed; they are always either uniformly enlarged or the fundus alone is wider than normal. The opening is usually narrowed, so that the whole crypt becomes bottle-shaped. In some cases they become detached from their base and raised; sometimes they protrude into the lumen of the intestine or become completely detached and desquamate. I have seen conditions of this kind in children a few weeks old, even when the catarrh had existed only four days. The deeper layers of the submucosa are markedly hyperemic only in severe acute catarrh; the same applies to the accumulation of round-cells around the blood-vessels. Even in this condition the muscular coat remains unchanged.

It is clear that numerous parasitic and chemic irritants that are always present in the intestine can exercise a very deleterious effect on the mucous lining of the intestine if it is changed by catarrh in the way just described. The immediate result of these irritations is the development of areas of moderately deep destructions of the intestinal mucous membrane; in other words, catarrhal and follicular ulcers.

In chronic catarrh, that either develops gradually after the acute form or has an insidious onset from the beginning, the color of the mucosa varies from a pale gray-red to a dark venous red. The latter color is seen when catarrh supervenes on the basis of chronic venous engorgement. In other cases the appearance of the catarrhal mucosa is gray and slate colored; this is due to the extravasation of pigment between the glands (hemochromatosis of the intestine). This coloration can occasionally be seen in a perfectly healthy intestine, and must, under these circumstances, be considered as the relics of a past attack of acute catarrh. Rosenfeld states that the pigment so frequently found in the mucosa and muscularis is not derived from the blood-pigment, but

directly from albumin. He bases his statement on both its morphologic and chemic properties. The mucosa and submucosa are sometimes very greatly swollen and infiltrated with serous fluid ; and, as a result, the whole intestine is often thickened.

Chronic catarrh of the intestine is histologically identical with acute catarrh, so that for a long time the chronic process presents the same microscopic picture as the acute one. I have on various occasions examined cases of catarrh of the intestine that were characterized by a particularly chronic course, some idiopathic, some secondary (following valvular lesions) ; in many of these cases the glands of the intestine were elongated and well developed, as in a normal intestine ; the only abnormality by which the presence of catarrh could be determined was the accumulation of pigment either in the interstices between the glands or in the muscularis mucosæ, and in addition a slight widening of the interstices. In the majority of cases of chronic catarrh accumulations of round-cells, which are so characteristic of the acute form of catarrh, are not seen ; on the other hand, there is always connective-tissue proliferation in the chronic form and not in the acute one.

As a result of chronic catarrh, atrophy or hypertrophy of the intestinal mucosa may develop. A special section will be devoted to a discussion of the former condition ; a short account of the hypertrophied condition will be given here. In this condition the glandular tubules are elongated, run a serpentine course, and form diverticula. Occasionally their orifices become occluded, so that the accumulation of secretion within the glands leads to the formation of small cystic sacs (*enteritis chronica cystica*). The extensive proliferation of connective tissue leads to the formation of polypoid excrescences in various portions of the bowel (enteritis polyposa). The latter form is comparatively rare, and is most frequently seen in the large intestine. In still other cases the walls of the intestine become thickened throughout. If this occurs, the muscular coat shares in the change. I have repeatedly measured the thickness of the intestinal wall in health and in disease, and have found that the wall of the normal small intestine is from 0.3 to 0.7 mm. thick, the large intestine, without the tenia, 0.6 to 1 mm., whereas in chronic catarrh the wall of the small intestine may be 1.6 mm. in thickness, the wall of the large intestine, 1.875 mm. Some authors claim that this thickening of the intestinal wall may progress so far as to lead to narrowing of the intestinal lumen. I have been unable to confirm this statement from my own experience.

SYMPTOMATOLOGY OF ACUTE CATARRH.

The clinical picture of acute catarrh of the intestine is determined by the primary cause of the disease, the seat of the inflammation, its extent, severity, and many other factors, so that it is quite impossible to give an account which will accurately describe all cases. In the following remarks an attempt will be made to describe in the main the characteristic symptoms of acute " catarrh from indigestion," or so-

called idiopathic rheumatic catarrh.   All the so-called specific forms of intestinal catarrh will be discussed in the sections on the different diseases that produce these inflammations.   In the following account it is assumed that the colon and the lower part of the jejunum and the ileum are both involved, for, as a matter of fact, this is usually the case.

The condition of the stool is one of the most important clinical features of the disease and one of the most significant factors in the diagnosis.   The other local and remote symptoms that accompany catarrh are all of subordinate importance.   The description, therefore, begins with an account of the fecal evacuations.

The dejecta—and this is characteristic of catarrh of the intestine— are diarrheic in character—that is, their consistence is less solid than normal; they are usually liquid and increased in number.   In the course of twenty-four hours 2, 5, 10, 15 stools, or even more, may be passed.   At the beginning of the attack the first and second stools are usually pultaceous, whereas later evacuations are quite thin, watery, and frequently foamy from the admixture of gas, or again gelatinous or slimy.   Even after a few days, however, a solid evacuation may occasionally be passed between a number of liquid stools, or isolated hard scybala may be found in a watery mass of dejecta.   The more extensive the area of the colon involved, the more predominant the diarrhea. When the small intestine alone is affected, diarrhea does not necessarily develop (compare below, paragraph on the localization of intestinal catarrhs).   The total quantity of material passed with the feces frequently considerably exceeds the quantity of food ingested.   This is due to the fact that the digestive secretions (the bile, the pancreatic juice, and the intestinal secretion) that are poured into the small intestine are evacuated with the stools or that the altered mucosa or its blood-vessels produce an enormous amount of secretion when catarrh supervenes.

The odor of the feces in acute catarrh of the intestine may be either fecal, exceedingly offensive, or odorless, and occasionally different stools vary in this respect.   If much fermentation occurs in the bowel contents,—as, for instance, after plentiful ingestion of foods rich in starch and cellulose,—the products of these fermentative processes will be expelled with the stools.   This is due to the fact that peristalsis is so much accelerated that these gaseous products cannot be absorbed as rapidly as in health.   When the stools consist chiefly of exudates and mucus, the fetid odor is less intense.   Sometimes, and particularly if certain articles of food (chiefly milk) are taken in abundance, the odor of the stools is markedly acid, but the reaction of the stools is almost always neutral or alkaline, rarely acid.   This feature is not determined by the catarrh *per se*, but more by the character of the food and the species of ferment organisms that happen to be present in the intestine. The reaction may, therefore, change in the same individual.   The color of the stools is usually dark brown and occasionally light yellow; in adults the stools are rarely green in color, whereas in small children this is frequently the case.   These differences in color are primarily dependent on the involvement of the colon alone, or of the colon and the

small intestine, particularly the uppermost portions of the small intestine, together. The diet, of course, and certain medicines also influence the color of the stool. If the feces are light yellow in color, a positive Gmelin's reaction for bile-pigment is often obtainable; when they are green, this reaction is usually very distinct. When diarrhea is extremely profuse, the dejecta may be perfectly colorless, for in these instances they often consist exclusively of serous exudate. Colorless stools are very rarely evacuated in ordinary catarrh of the intestine, whereas in some of the specific forms of intestinal catarrh, as in cholera nostras, this peculiarity is frequently seen.

The stools nearly always contain an admixture of mucus: it either floats on top of the watery dejections in the form of large or small shreds, is light and glassy in appearance, or stained various colors by the different pigments present in the intestinal contents, or it is intimately mixed with the solid and serous constituents of the bowel contents and transforms the dejecta into a jelly-like mass. In very rare cases of acute catarrh of the intestine the dejecta consist exclusively of mucus. For some of the details of this subject and for the significance of pus and epithelial cells in the feces the reader should refer to what has been said in regard to mucus in the general section on the Feces. Large numbers of pus-cells are rarely seen in acute catarrh, and the clouding and opacity of the mucus are not due to pus-cells, but to the presence of desquamated well-preserved or partly changed intestinal epithelium. Blood is very rarely seen in simple catarrh of the intestine; and, if present at all, it is found only in extremely small quantities and always shows that the inflammatory hyperemia is very intense.

Various food remnants may frequently be seen in the feces with the naked eye. This, of course, is not surprising. Occasionally the undigested or indigestible substances that originally caused the catarrh (pieces of cabbage, etc.) appear in the stools as late as the third, fourth, or fifth day. As soon as this material is evacuated, improvement begins. (For the examination for meat and connective-tissue remnants see pp. 83 and 84.)

Chemic analysis of the stools for the different digestive ferments and the various products of digestion is of subordinate importance in clinical work.

Bacteriologic examination of the dejecta may occasionally yield some information in regard to the etiology of the attack. The reader should refer for the details of the chemic, particularly the Schmidt-Strasburger refermentation, test and bacteriologic examination to the two sections that deal exclusively with these subjects.

The other symptoms are insignificant in comparison to diarrhea, at least so far as the diagnosis is concerned. When diarrhea is absent,—and this occasionally happens—*i. e.*, in those rare cases of isolated catarrh of the small intestine that we have mentioned,—it is generally impossible to recognize the existence of a catarrh. The most important subjective symptom, apart from diarrhea, is the pain. The pain is either

colicky or inflammatory in character, pinching and biting, frequently very severe, and accompanied by sensations of general malaise. The pain usually begins suddenly, but occasionally has a more gradual onset. Even in isolated colitis the pain is so severe as to be felt over the whole abdomen; when the rectum is also involved, tenesmus develops which forces the patient to evacuate the bowels as rapidly as possible. This colicky form of pain is more paroxysmal in character, and usually ceases as soon as the stools are passed. In intense catarrh there is a certain amount of tenderness on pressure that is due to the inflammation of the bowel; this tenderness frequently persists in subacute cases after the violent spontaneous and colicky pains have subsided. In some instances it is possible to trace the course of the colon on the abdomen from the distribution of the area of tenderness on pressure. This can occasionally be done in cases in which the patients are otherwise completely free from pain.

The physical signs of catarrh are very uncertain, very variable, and are consequently of still less importance in the diagnosis than the pain. Loud, rolling noises (borborygmi) are often audible. If the abdominal walls are thin, violent peristaltic movements can be seen in exceptional cases. Short taps over the belly will elicit distinct splashing; occasionally distinct fluctuation of the liquid bowel contents can be felt through the abdominal walls. The configuration of the abdomen varies and will depend on the presence or absence of abundant quantities of gas in the bowel. If much gas is present, the abdomen may protrude, but as soon as the gas is expelled, the outline of the abdomen changes. The note elicited by percussion is as variable as the configuration of the abdomen. If much fluid is present in the intestine, the percussion-sound over the bowel may be high, dull, and not tympanitic, whereas in other cases it may be deep and almost tympanitic over the same area. Large accumulations of gas, however, are very rare in idiopathic catarrh of the intestine. If much gas accumulates, as it does in exceptional cases of idiopathic intestinal catarrh, all the other symptoms and consequences of this event will appear (compare the section on Meteorism).

Fever is a very uncertain complication of acute catarrh of the intestine. In many instances it is completely absent; in others there are several mild chills, general febrile discomfort, with a slight rise of temperature; again, in other cases, there are a single chill and a rise of temperature up to 102° F. (39° C.) or 104° F. (40° C.). In simple catarrh, however, the temperature drops back to normal within a few days. These variations of the temperature are probably due to differences in the etiology of simple acute catarrh of the intestine. For the present, however, our knowledge of this subject is very incomplete, for nearly all the observations on simple acute catarrh of the intestine have been made in private practice, where careful clinical study is impossible.

The urine presents no peculiarities in mild cases of acute catarrh, but as soon as the evacuations of the bowels become very profuse and watery, the urine usually becomes scanty and concentrated. In some

cases of acute enteritis it contains casts and albumin. This has been discussed above (p. 165).

[From a study of the leukocyte-count in the summer diarrhea of children Knox and Warfield [1] find that, though the number of leukocytes is usually increased, the counts vary so much that high or low leukocytosis cannot be regarded as of diagnostic value. From intoxication with decomposition products or bacterial toxins the polymorphonuclear leukocytes may be increased.—ED.]

An acute splenic enlargement, at times of considerable size, is described by Fischl as occurring in many cases. The splenic enlargement rapidly decreases after disappearance of the catarrh, which he regards as a true acute febrile intestinal catarrh, and not as a mild form of typhoid. Ewald has never observed this splenic enlargement.

Gastric symptoms frequently complicate intestinal catarrh. It is natural that the stomach should in many cases be affected by the same irritants that produce acute catarrh of the intestine; in this way the symptoms of gastro-intestinal catarrh are frequently produced. For the stomach symptoms the reader should refer to the section on Acute Catarrh of the Stomach. Even though no structural lesions of the stomach are produced, certain gastric symptoms nevertheless frequently accompany acute catarrh of the intestine; these are loss of appetite, nausea, and in exceptional cases so-called sympathetic vomiting.

The general health of the patients is usually considerably impaired, even though no fever is present. There are commonly general debility and weakness, both in the mild cases of diarrhea and in the more severe attacks. In very serious cases, even though they do not present the symptom-complex of cholera nostras, pronounced symptoms of collapse may develop; these are due not so much to the small amount of nourishment that is taken, as to the rapid and very considerable loss of water by the bowel. Another factor producing these serious symptoms is impairment of the action of the heart, due probably to reflex irritation from the intestine. This is seen in all diseases of the stomach, intestine, and peritoneum, and is probably analogous in its pathogenesis to the tapping experiment of Goltz (Klopfversuch). A number of authors have recently supported the theory of absorption of toxic substances from the intestine to account for these general symptoms, but the subject is not sufficiently advanced to require discussion here at present. The weaker the individual, the more severe these symptoms of collapse; they are more pronounced and develop more rapidly in young children and in very old persons. In children the syndrome of acute hydrocephalus occasionally develops, presenting all the well-known features as originally pointed out by Marshall Hall and described in the section on Anemia of the Brain in another volume of this series.

The course of acute intestinal catarrh varies greatly. In mild cases restitution to normal occurs within a few days; in more severe cases several weeks may elapse before the catarrh can be considered cured, and in another group of cases acute catarrh never gets well, but gradu-

[1] Knox and Warfield, *Bull. Johns Hopkins Hosp.*, vol. xiii., p. 107.

ally develops into chronic catarrh which may be very obstinate and persistent and may even become an incurable condition. The exact course and termination of acute catarrh of the intestine are, therefore, dependent, in the first place, on the etiology; in the second place, on the treatment, chiefly on the dietetic regulations that are imposed during the disease and during convalescence. Even after an attack of acute diarrhea is over and the patient seems to be perfectly normal, the intestine remains somewhat susceptible, so that slight causes frequently produce symptoms of intestinal disease. A remarkable sequel of acute catarrhal diarrhea is a mild attack of constipation, which usually follows immediately after the diarrhea and lasts for a short time. I wish to lay great stress on the importance of this post-diarrheal constipation, for I have known chronic habitual constipation to develop from it. Death rarely occurs in simple acute catarrh of the intestine. A fatal issue is seen in exceptional cases, accompanied by grave symptoms in very debilitated individuals.

SYMPTOMATOLOGY OF CHRONIC CATARRH.

The older clinical descriptions of chronic catarrh of the intestine are very unsatisfactory, chiefly because the diagnosis of "chronic catarrh of the intestine" was frequently made in ordinary daily practice when this disease was not really present; thus, many cases of chronic diarrhea that were due to some nervous disorder, or still more frequently cases of simple habitual chronic constipation, were all called chronic catarrh. Many years ago I began to study the most characteristic symptom of this class of diseases, namely, the changes in feces, in a great number of cases, not only in those that were carefully observed during life, but especially in cases that died from some intercurrent disease and in which an anatomic examination was rendered possible postmortem. In this way I was able to throw much light on the nature of chronic catarrh, and to establish its diagnosis of this condition on a reliable footing. The following description is based on my early examinations, and I still adhere to the views enunciated many years ago.

The chief symptom of chronic catarrh of the intestine, as I have said, is the abnormal character of the feces. The motions are irregular, and the consistence and constitution of the stools are also abnormal.

Diarrhea which may be considered characteristic of acute catarrh of the intestine is not constant in the chronic form, and the frequency of the motions varies greatly. The following modification of the evacuation of the bowels may occur in this condition:

In one group of cases there is marked constipation, and a solid motion occurs only every second or fourth day, occasionally only after the administration of a laxative. This I consider to be the essential feature and the most characteristic point in catarrh of the large intestine. The primary cause of this constipation is probably a reduction in the automatic powers of the nervous mechanism of the intestine (possibly due to nutritional disturbances), and this reduction is produced

directly by the chronic catarrh itself. Even though this automatic function is reduced, there may still be normal or increased irritability to stimuli and irritants. This is quite analogous to similar conditions known to exist in other nervous mechanisms. The correctness of this view is manifested by the fact that very frequently constipation and diarrhea alternate in cases of chronic catarrh.

These cases constitute a second group ; the irregularity of the actions of the bowel is seen in so many instances that it is considered one of the most characteristic features of the disease. If we are justified at all in drawing conclusions from any one symptom, I am inclined to say that the alternate occurrence of constipation and diarrhea almost invariably indicates chronic catarrh of the intestine. The two conditions alternate with astonishing regularity : for two or three days a solid motion will be passed every day ; on the following day from four to six very thin or soft stools, or stools mixed with mucus, are passed, and at the same time the patients suffer from very violent abdominal pain ; on the following day there will be constipation, and solid stools will be passed for two or three days, etc. In other instances a fairly normal evacuation of the bowels will occur for several days in succession, then from four to seven liquid evacuations accompanied by colicky pain will occur on one day, then constipation again, etc. The most characteristic feature of these cases is the constipation ; at the same time the irritability of the nervous apparatus remains fairly good, consequently increased peristalsis supervenes as soon as the stagnating bowel-contents begin to decompose. Another type of periodicity is occasionally seen in these cases, namely, the constipated or the diarrheic periods may persist for a much longer time—for days, weeks, or months, or may alternate at irregular intervals and without any definite type of periodicity. I am inclined to the belief that the periodic attacks of diarrhea that occur at such irregular intervals are caused by some accidental intercurrent agency that produces an acute exacerbation of the chronic process.

In a third group of cases, rather limited in number, there is a daily evacuation of unformed and pultaceous feces.

In a fourth class of cases the patients pass several diarrheic stools daily. This is exactly the opposite condition to that met with in the first class of cases. The diarrhea may persist for several months ; in these instances the small intestine is involved together with the large intestine. The character of the dejecta at once shows the existence of catarrh of the small intestine, for it gives a diffuse bile-pigment reaction or contains epithelial and round-cells that are impregnated with bile. I have been able to verify all this on several occasions by anatomic postmortem examination. The explanation of this phenomenon is as follows : The ingesta, in the first place, are not completely digested in the small intestine ; if there is catarrh, certain abnormal products (acid, etc.) are produced in addition, so that the residues of incomplete digestion, together with the abnormal products of fermentation, act as an irritant to the catarrhal mucosa of the large intestine, in this way stimulating its muscular coat to increased peristalsis. In the second

class of cases this seems to occur more or less periodically, hence the periodic character of the diarrhea.

In addition to these forms of dejections that may be called typical there are occasionally a great variety of bizarre perversions of defecation that can only be interpreted to signify some direct disorder of the nervous influences governing the character and the evacuation of the stools; in other words, there must be a combination of catarrh and a more or less serious disturbance of the nervous mechanism of the intestine or of the central nervous system. In some of these cases, for instance, the motions occur only during the night or only in the early hours of the morning; while during all the rest of the day the patients are perfectly comfortable.

Ewald observes that in these cases the intestinal symptoms may exist independently of organic change or of intestinal catarrh, being merely a secondary symptom of a general neurosis. This is certainly the case. It is possible too that in many of my cases of this kind there was a purely accidental coexistence of intestinal catarrh and general neurosis. Boas further remarks that it is not improbable that there is a primary and purely functional nervous diarrhea, and that its continued chemic and mechanical irritation of the intestinal wall sets up a genuine catarrh.

I repeat, that the foregoing description of evacuations in chronic catarrh is based not only on clinical findings, but also upon long-continued examinations of feces, the anatomic results of which were substantiated postmortem, both macroscopically and microscopically.

The quality of the dejecta is apparently normal if they are formed, or if there is constipation, as in catarrh of the large intestine, with the exception, however, that mucus is always present in the stools. The apparently normal character of the stools has been explained in the preceding paragraphs. The admixture of mucus, however, is of paramount importance. It has already been explained in the general part of this volume why any admixture of mucus to the stools, if it is at all considerable, must be considered pathologic, and must always denote some morbid increase of the activity of the intestinal mucosa. In very rare cases the excretion of mucus is due to an entirely different cause (compare the section on Enteritis Membranacea). In the overwhelming majority of cases the passage of mucus is due to that disease of the mucosa which is, anatomically speaking, called catarrh. Ulceration of the intestine, carcinoma, etc., *per se*, never lead to the evacuation of mucus; if mucus is passed in the stools in any of these conditions, it must always be assumed that some catarrhal changes have occurred that are secondary to these diseases. Mucus in the dejections enables us to formulate the diagnosis "catarrh" in general, it being immaterial what the pathogenesis of this catarrh may be. Conversely, the absence of mucus in the dejecta, it being immaterial whether the stools are liquid or solid, usually militates against the diagnosis of catarrh; in habitual constipation, for instance, mucus is never present in the stools; occasionally, of course, very hard masses of fecal material are covered in

spots by a peculiar layer of shellac-like mucous material which has been described elsewhere. This feature is very important, for it frequently enables us to decide whether infrequent defecation is due to habitual constipation or to a chronic catarrh of the large intestine.

In very rare cases the feces are not constantly covered with mucus, even though there may be a severe catarrh of the rectum and the descending colon, with the production of a large amount of mucus (as shown by postmortem examinations). This may be due to the fact that the mucus is very tough and adheres too tenaciously to the wall of the intestine, or, on the other hand, that the fecal masses that pass through the catarrhal area are too small to scrape off any of the mucus. This finding, however, is exceptional, and careful observation will show that on many occasions the feces are covered with mucus, and so enable a positive diagnosis to be made.

The quantity of mucus evacuated may vary greatly : occasionally such large quantities are passed that the motion consists exclusively of large masses of mucus (some of the cases of so-called enteritis membranacea are of this nature). All the various possible combinations and mixtures of mucus with solid or liquid feces described on pages 87 to 89 may be encountered (refer to page 192 for the details).

The color, the odor, and the reaction of the stools are the same in chronic as in acute catarrh. Blood is never present in the stools of chronic catarrh—at least it is never there owing to the presence of catarrh *per se ;* if it is found at all, it comes from some complicating lesion, as an ulcer, hemorrhoids, etc. Pus is also rarely found in appreciable quantities ; at all events it is never visible to the naked eye ; occasionally a few isolated pus-cells or a small group of such cells are discovered under the microscope. There is a disease called blennorrhœa intestinalis, in which the dejecta resemble pus diluted with water ; feces of this kind are not a symptom of catarrhal enteritis, but of a croupous diphtheric form of catarrh (compare pp. 215, 216). Large-masses of epithelial cells, however, are very frequently found ; these elements are, as a rule, in various stages of degeneration. They must also be considered responsible for the cloudiness of the mucous secretion that is normally glassy and transparent. An inexperienced observer is liable to assume that the opacity of the mucus is due to the presence of pus-cells, an error which only microscopic examination will correct.

The character of the food, on the one hand, and the intensity of peristalsis, on the other (diarrhea or constipation), will determine the consistence and the reaction of the feces and will also determine the amount of fermentation that occurs in the bowel contents. It is unnecessary to go into the details of these questions again.

Physical examination of the abdomen reveals the same variations as in acute catarrh of the intestine. The results of this examination are usually quite unreliable, so far as making a diagnosis is concerned. Sometimes the conditions are perfectly normal ; in other cases, particularly during the period of diarrhea, the bowels are distended with gas, so that the percussion-note and the resistance over certain areas of the

abdomen are correspondingly changed. When the bowel-contents are liquid, fluctuation can usually be felt ; in constipation the presence of hard scybala can, as a rule, be determined by palpation. The abdomen is very frequently tender on pressure, occasionally particularly so in definite areas.

Many of the patients never have any subjective symptoms, but the majority of sufferers from chronic catarrh, however, complain bitterly of a general feeling of discomfort and a feeling of diffuse soreness in the abdomen ; this sensation appears spontaneously either after eating or after defecation. These patients rarely complain of violent pain ; the latter is experienced only in acute exacerbations of the catarrh. The general feeling of discomfort that I have described is in part due to the inflammatory process, in part to the abnormal distention of the bowels by gas. Occasionally flatulence may become so severe that it leads to very distressing consequences, and this is more liable to occur in the chronic than in the acute form of intestinal catarrh. A variety of other symptoms are occasionally seen in chronic catarrh of the intestine, but they are no more constant nor characteristic than the ones we have already enumerated. The general health of the patient may remain unchanged in some instances ; in others it may become seriously undermined. The general nutrition may become impaired, although this rarely happens if the lesion is strictly limited to the large intestine ; it does occur, however (for reasons that can be readily understood), if the small intestine is involved at the same time. In children particularly, extremely serious disturbances of nutrition may depend on chronic catarrh of the small intestine (compare Intestinal Atrophy).

The appetite and the functions of the stomach are rarely disturbed in chronic catarrh of the large intestine, but are usually seriously affected in catarrh of the small intestine.

For many years different authors have regarded a variety of nervous disorders as the direct consequence of enteritis chronica—for instance, general lassitude, distaste for work, psychic depression leading to hypochondriasis, and general irritability. It can readily be understood why a patient afflicted with so disagreeable, persistent, and tedious a disease should become depressed. I have already discussed this subject in the section on the Nervous Sequelæ of Habitual Constipation, and have shown that this is a disease that, as I have repeatedly emphasized, is very frequently confounded with chronic enteritis by the general practitioner. The same conditions obtain in chronic catarrh of the intestine as in habitual constipation, and all the statements made as to the interdependence of constipation and certain nervous (cerebral) symptoms apply with equal force to chronic intestinal catarrh. Psychic depression is a natural consequence in an individual with a neuropathic predisposition who is afflicted for years with chronic catarrh of the intestine.

The course of chronic catarrh of the intestine is usually very tedious. Chronic enteritis may persist for many years or even for life. The intensity of the symptoms varies, and the patients may enjoy long

periods of almost complete health; at the same time the intestine remains permanently vulnerable, so that a patient who has once suffered from chronic catarrh of the intestine easily develops symptoms after some slight injury of the intestine that a healthy person could bear with impunity. Death, however, rarely results directly from simple chronic catarrh of the intestine, but the disease may ultimately lead to death from exhaustion or from impairment of the general nutrition. This accident occurs only in very weak subjects and in children.

Whereas, therefore, the prognosis as to life is apparently favorable, the prognosis as to a cure is not so hopeful. A complete return to the normal anatomic condition is probably out of the question in very chronic cases of catarrh, for examination of the intestine in these cases always reveals some traces of the process. The functional disorders may disappear, provided the disease does not persist for too long a time; whether or not this can also occur after the catarrh has lasted for several years seems to me more than doubtful; and in my judgment a catarrh of this kind is incurable.

DIAGNOSIS.

The diagnosis of chronic catarrh of the intestine can be discussed in a few words. Such symptoms as constipation or diarrhea, pain or borborygmi, or any of the other symptoms that are encountered with more or less frequency never positively demonstrate the existence of a catarrh. These signs can all be interpreted in many different ways and may be due to many different causes. The only diagnostic symptom of value is the appearance of mucus in the dejecta; for it practically occurs only in chronic catarrh of the large intestine. The other conditions in which it is seen are colica mucosa and those rare cases in which the jelly-like contents of the uppermost portion of the small intestine appear in the feces.

Localization of the Catarrh.—In the preceding paragraphs a general account of catarrh of the intestine has been given. The clinical picture described is essentially that of a catarrh of the large intestine, and, as a matter of fact, the disease is most frequently localized in this portion of the bowel. In the paragraphs on the pathologic anatomy of catarrh I have called attention to the fact that the disease may, however, be localized in many different portions of the intestine. The whole small and large intestine may be involved at once, or the large intestine may be involved alone,—this is a frequent occurrence,—or again the small intestine may be involved alone—this is rare, but does occur, as I can testify from personal observation; again, the beginning of the small intestine, particularly the duodenum and the upper jejunum, may be involved alone (this usually occurs in combination with catarrh of the stomach), while the lowest portion of the large intestine may be involved alone (this condition is called proctitis, and will be discussed in a special section). The vermiform appendix may be the only portion of the intestine that is diseased (this

condition is called appendicitis [scolecoiditis] and will be described separately in the section on Diseases of the Peritoneum). Again, the cecum and the ascending and the transverse colon may be affected separately or together, or these portions of the bowels may be involved together with the ileum. When these combinations occur, the process may be more intense in the one portion or in the other, may involve chiefly the small intestine, or those portions of the large intestine that we have enumerated ; occasionally these differences can be recognized clinically.

The localization of intestinal catarrh is not only theoretically interesting, but is of great practical importance, for the possibilities and methods of treatment to be employed vary according to the situation of the catarrh. It is, therefore, far from unnecessary or useless to look out for clinical signs indicating the position of catarrh in the bowel. Some of these symptoms have long been known and utilized. Some twenty-five years ago I was the first to call attention to the importance of the stools in this respect, and to formulate rules which made it possible to draw certain conclusions in regard to the localization of the catarrh from the composition and consistence of the feces. These rules were based on extended clinical and pathologic examinations.

Icterus can appear only when the inflammatory process involves the duodenum ; it appears as an expression of intestinal disease in catarrh of the duodenum, a disease that usually occurs together with acute catarrh of the stomach and rarely involves the duodenum alone. Inflammation limited to the duodenum probably occurs only in duodenitis due to extensive cutaneous burns (compare the section on Ulceration of the Intestine Following Cutaneous Burns).

As a localizing symptom pain is of use only when it is strictly circumscribed and limited to some distinct area. The pain that is elicited on pressure is more important than spontaneous pain, for the reason chiefly that in acute processes violent spontaneous pain frequently radiates over wide areas. The pain of typhlitis and appendicitis is limited to the right iliac fossa and is particularly important. Another form of pain that is important is tenderness to pressure along the whole course of the colon or along certain portions of the large intestine in colitis. The form of pain called tenesmus may be considered characteristic of inflammation of the lower part of the rectum. Pain on pressure in the right hypochondriac region is a very unreliable index of duodenitis unless there is icterus at the same time. When icterus and pain in this region are both present, however, the diagnosis of duodenitis is very probable. We may be reasonably certain that when pain is elicited by pressure in the mid-abdominal region, and not in the sides and iliac fossa, the affection is of the small intestine alone, without involvement of the colon. In diffuse acute enteritis the whole abdomen may, of course, be tender on pressure.

Auscultation of the gurgling sounds that are occasionally heard in active peristalsis is of very little value in localizing intestinal catarrh. This is due to the fact that sounds of considerable intensity—and only such sounds can be considered in forming a diagnosis of catarrh—can

usually be heard in other portions of the abdomen than those in which they originate. A certain sound, for instance, may be heard with the same intensity on the two sides of the abdomen by two different observers who are auscultating at the same time. From repeated investigation of this matter I feel justified in making this statement.

Percussion gives very little information of value, and its results are exceedingly unreliable. In general it may be said that the percussion-note is normally duller and higher in the left iliac region than in the right. Traube taught that in typhoid fever this proportion is frequently reversed; I find, however, that this alteration is not constant in typhoid fever nor peculiar to this disease. It may occur in any other form of increased peristalsis, even in simple catarrh. Occasionally in the latter condition the percussion-note in the two iliac fossæ becomes equal. Any variation in the degree of distention or filling of the cecum and the sigmoid flexure, either by gas or solid material, exercises an influence on the percussion-note in that region, so that the so-called "normal" percussion-note in this area is in itself very variable. Some authors claim that a dull note in the center of the abdomen indicates an accumulation of a large quantity of catarrhal fluid in the small intestine, but this conclusion seems to me very uncertain, for the same percussion-note can be heard when the bowel contents are normal and pultaceous, or even when the small intestine is quite empty.

Palpation may either reveal pain on pressure or may produce gurgling sounds. The significance of the latter symptom in the diagnosis of catarrh and of the localization of the process in the large intestine is small, for the occurrence of gurgling merely indicates that the proportion of liquid and gas present in the intestine is abnormal, for normally gurgling sounds cannot be produced by simple palpation.

In a previous section (p. 165) the conclusions to be drawn from the examination of the urine in regard to the localization of intestinal catarrh have been considered.

While these different signs are all of subordinate importance because they vary so much, the examination of the dejecta provides most valuable information. As a matter of fact, the localization of intestinal catarrh depends almost exclusively on the examination of the feces. An analysis of the feces in many instances furnishes so many reliable facts that an experienced observer is frequently able to draw definite conclusions from the macroscopic examination of the stools alone. As the most important points that can be elicited from an examination of the character of defecation in general (constipation, diarrhea, alternating constipation and diarrhea) have been described, they will not be repeated, and the following remarks will be limited to the interpretation of the significance of the different fecal constituents in localizing catarrh of the intestine.

Mucus.—The evacuation of pure mucus without any admixture of fecal matter indicates catarrh of the rectum, of the sigmoid flexure, and of the descending colon (see also the section on Enteritis Membranacea). When solid balls of fecal matter, particularly small masses, covered with

a layer of mucus, are passed, catarrh of the same portions of the large intestine is indicated (very large columns of fecal matter may occasionally be covered by a very thin layer of mucus that is attached in streaks and gives the feces a varnished appearance; this may occur without the existence of catarrh).

Catarrh of the upper part of the colon causes different modifications of the stool. When hyaline, microscopic lumps of mucus are found to be intimately mixed with solid or pultaceous-solid stools, and when no mucus can be seen with the naked eye, catarrh of the upper portion of the large intestine without involvement of the lower portion is indicated; it may also indicate catarrh of the small intestine, as I have found from a careful study of a number of cases of this disease.

When there is catarrh of the whole large intestine as high as the cecum so that the dejections are very thin, small shreds of mucus are still intimately mixed with semiliquid fecal matter, but there is this difference, however, that under these conditions the mucus can be recognized with the naked eye. These cases, moreover, are, in addition, distinguishable from catarrh limited to the rectum and the descending colon by the fact that the admixture of mucus and fecal matter is much more intimate. Occasionally a case of catarrh of the whole colon is met with in which the mucus and the fecal matter are not so intimately mixed. I refer, for instance, to cases of ulceration in which large portions of the mucosa are destroyed. Here a comparatively small quantity of mucus is excreted and the contents of the lowest portion of the small intestine are propelled rapidly through the large intestine.

When, therefore, mucus and fecal matter are intimately mixed, it being immaterial whether the feces are solid, pultaceous, or liquid, it may always be safely concluded that the upper portions of the colon or the small intestine are involved in the catarrhal process.

Boas recommends lavage in the diagnosis of catarrh of the large intestine. For this a tube some $\frac{3}{4}$ meter long is introduced as high as possible into the rectum, and $\frac{1}{2}$ to 1 liter of lukewarm water is poured in through a funnel, precisely as is done in lavage of the stomach. The tube is then lowered, and the injecta siphoned off. If catarrh of the large intestine exists, the recovered fluid will contain mucus that is readily demonstrable. Although this proceeding gives no new diagnostic sign, it is a convenient method, and positive results are useful in localizing catarrh in the large intestine.

I have considered *yellow-colored mucous granules* in the evacuations as diagnostic of affections of the small intestine, but the mucous nature of these granules has recently been disputed. [A. Schmidt, Boas.—ED.]

Bile-pigment.—If a typical bile-pigment reaction can be obtained in the dejecta, or at least in some of the constituents of the stool, this positively indicates an affection of the small intestine. The higher up the intestine is involved, the more pronounced the reaction; either the whole stool gives a positive reaction, or the bile-pigment is found attached to certain constituents of the feces, in particular to the mucous constituents. In this case the mucus is stained dark orange, ochre,

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greenish-yellow, or green. Occasionally cylindric epithelial or round-cells are found impregnated with bile, or in other cases droplets of fat (rare) are seen to be stained intensely yellow. The coloration of these elements indicates that they are derived from the small intestine.

An acid reaction of the dejecta also invariably shows involvement of the small intestine. I can recall no conflicting case.

The following observation of Boas is of interest. He took the filtrate of feces, and without any addition whatever subjected it to a digestion test by means of an albumin plate. His result was positive, and he is doubtless correct in his conclusion that the diarrhea originated in the small intestine.

The appearance of large quantities of undigested food-particles in the feces has also been utilized in deciding upon the situation of the catarrh, in this way, namely, that the appearance of this material indicates involvement of the small intestine. A careful analysis of all the recorded observations both at the bedside and on the postmortem table in regard to this matter shows the following : The appearance of large quantities of fragments of muscle tissue indicates catarrhal involvement of the small intestine. Quantities of connective tissue (Schmidt) with few muscle-fibers indicate a disturbance of gastric digestion. The appearance of large quantities of starchy material in the stools also indicates involvement of the small intestine. Neither meat-fibers nor particles of starch *per se*, however, indicate catarrh, for they may appear in the stools in different forms of intestinal dyspepsia, and, in particular, in simple increased peristalsis (without catarrh and due to other causes—as, for instance, diarrhea). The appearance of fat in the stools (compare fatty stools) is still less important in localizing affections of the intestine.

Mayor has described a case of inflammation confined to the sigmoid flexure as *sigmoiditis*. In his four cases with an acute course, the region of the sigmoid flexure was swollen and very painful on pressure. Two of the cases had fever. All went on to recovery, one with abscess-formation and perforation into the intestine. Mayor regards these cases as analogous with typhlitis, and attributes them to fecal retention, probably combined with specific infection the result of erosions of the intestinal wall. These cases, without biopsy or necropsy, are ambiguous, and cannot possibly prove the existence of an affection confined to, or emanating from, the sigmoid flexure. Such a conclusion cannot be accepted without further confirmatory observations. The extreme infrequency of cases such as Mayor's *sigmoiditis* is alone sufficient to justify the belief that his cases must have some affection other than sigmoiditis from fecal retention, for the sigmoid flexures of innumerable individuals are constantly filled with putrefying feces. Boas reports two cases as *chronic sigmoiditis*. In one case the symptoms were : Spontaneous pain, particularly intense on pressure over the sigmoid flexure, diarrhea with mucus, tenesmus. This doubtful symptomatology became evident only after operation for a rectovaginal fistula of several years' duration. The other case occurred in direct connection with a grave acute dysentery. It does not appear that these cases prove that the

catarrhal processes was primary and confined to the sigmoid flexure. Leube mentions as not infrequent *circumscribed, chronic inflammatory infiltration of the intestinal wall, particularly of the sigmoid flexure.* The infiltrated parts present a smooth, uniform, and extended surface of increased resistance, and he terms it chronic inflammatory infiltration of the walls. Personally I have never encountered a clinical picture that I could interpret as a localized catarrhal affection of the sigmoid flexure. In one case which I believed to be a perisigmoiditis as the result of primary sigmoiditis, there was a swelling, apparently inflammatory, strictly localized in the left iliac fossa, about the sigmoid flexure. Operative measures showed the case to be actinomycosis, but as recovery followed, its point of origin was not clear. The truth as to the existence of these cases can be decided only by postmortem observations.

TREATMENT OF ACUTE CATARRH.

It is impossible to exercise any influence on the structural changes that are the basis of catarrh of the intestine; in other words, direct therapeutic procedures along such lines are impossible. The return to the normal must proceed according to the mysterious rules that govern these processes; the cure, in other words, must be performed by the organism itself. The more recent the process, the less stable are the pathologic alterations present, and the greater the prospect of a complete *restitutio ad integrum.* In chronic inveterate catarrh of the intestine the tissue changes are irreparable; these tissue changes, however, in all cases do not necessarily produce functional disturbances of sufficient severity to cause distress or discomfort to the individual affected; they all, however, create a *locus minoris resistentiæ* in the intestine, so that the slightest damage immediately produces an exacerbation of the slumbering process.

From a consideration of these facts a rule may be formulated which should always be borne in mind in the therapy of catarrh of the intestine, but is very frequently neglected. This rule is that the only kind of intestinal catarrh that can really be cured is a recent acute catarrh, since then only can a complete restitution to the normal be produced. For this reason recent acute catarrh calls for a most careful supervision of the diet, which should be very strict, and should be carried out consistently until all the symptoms disappear, and even for some time after this.

Occasionally it is possible to remove the cause (the *indicatio causalis*); if the acute catarrh is due to the ingestion of some chemically irritating substance, the appropriate antidote should be administered (acids if alkalis have been swallowed, etc.). Another way of counteracting the effect of chemic irritants is to attempt to remove them by the administration of purgatives. Purgative treatment is also called for, in the first place, when tainted, decomposed, or poisonous food has been eaten; and in the

second place, when excessive amounts of food have been taken. Occasionally a case of acute catarrh will baffle all treatment for a number of days ; the patient will have from five to fifteen motions a day, and the case may resist all astringent treatment and all other therapeutic procedures. Frequently in these cases on carefully questioning the patient it will appear that he has eaten something that is responsible for the acute catarrh ; for instance, before the onset of the diarrhea he may have eaten peas with sauer-kraut and fat meat. When facts of this character appear in the history of the patient's illness, laxatives should be given, even though there are frequent diarrheic motions. Very often a copious solid motion of an extremely penetrating odor will be evacuated and the patients rapidly recover. Laxative treatment should also be instituted when the diarrhea is due to coprostasis ; in these cases constipating remedies should certainly not be given. The diarrhea in these cases is an attempt on the part of the organism to get rid of the irritating material that is causing the catarrh. The best remedy to administer for producing evacuation of the bowels in these instances is calomel (in doses of 0.2 to 0.5 gm.) ; in addition, castor oil and irrigation of the intestine may be employed. The latter method of treatment is particularly useful if the stomach is at the same time considerably affected.

If a certain individual knows that definite articles of food and drink (as beer, milk, water, fruit, etc.) habitually produce a catarrh of the intestine, the ingestion of such articles of diet should be avoided from a prophylactic point of view. The same applies to the ingestion of very cold drinks and of ice-cream and ices in all those patients who know that swallowing cold food causes their intestinal mucosa to react in an abnormal manner. Persons who know that they develop catarrh of the intestine when they become drenched, or when they " catch cold " either from exposure of the whole or of certain parts of the body, should avoid these accidents.

Catarrh of the intestine due to malaria calls for specific treatment with quinin ; all other forms of intestinal catarrh that are due to specific infections are so far not amenable to specific treatment.

Another factor in treating the primary cause of the disease is to consider the action of the bacteria or fungi that are present in the intestine. Very frequently the general symptoms of a case of catarrh of the intestine will point to the action of these micro-organisms as the primary cause of the disease. When this is the case, it should be counteracted, and with this object a large number of so-called antifermentative and other drugs may be given. The chief difficulty from a practical point of view is to determine when this action of the micro-organisms should be considered an important factor in the pathogenesis of the catarrh. The following signs point in this direction : Abundant formation of gas, manifested by loud rolling and gurgling and the expulsion of much flatus, acid reaction of the feces, increased excretion of indican and ethereal sulphates in the urine, and a number of symptoms indicating that the catarrh is localized in the small intes-

tine. The latter point is important inasmuch as it is known that the action of bacteria and fungi is chiefly carried on in the small intestine. Under these conditions dilute hydrochloric acid or calomel (0.05 to 0.1 as a dose two or three times daily) may be given. In such small doses calomel acts more as an antiseptic and aseptic than as a laxative, and inhibits the activity of the lower organisms present in the bowel (possibly by the formation of chlorid of mercury). Resorcin in 5 per cent. solution, given either in water or in brandy in doses of three to six teaspoonfuls daily ; menthol, 0.1 to be given two or three times daily ; salol, 0.5 to be given from three to six times daily ; naphthalin, in the dose of 0.1 to 0.5, is not so useful a remedy, because, in the first place, it has a very disagreeable odor, and, in the second place, it is capable of producing symptoms of irritation in the urinary passages ; creasote, in 0.05 to 0.1 doses, given several times a day. To disinfect the large intestine by irrigation of the colon, boric acid in 0.5 per cent. solution, creolin in 0.1 to 0.2 per cent. solution, or salicylic acid in 0.2 per cent. solution should be employed.

When it is impossible to attack the cause directly, or when the primary cause of the catarrh has been removed, the second indication for treatment would be to attempt a cure of the structural changes (*indicatio morbi*). It has already been mentioned that this is beyond our powers ; but an attempt can be made to avoid all those factors that would interfere with the natural curative processes that are carried on by the organism wherever catarrhal lesions are present. In the first place, the diet should be carefully regulated. The necessity and the importance of rigid supervision in this respect cannot be sufficiently emphasized. The ideal condition would be to place the intestine at rest and to keep it empty for some time ; the next best thing is to administer food that is non-irritating in character and occupies the smallest possible bulk. On the first and second days of an attack of acute enteritis we should give warm or lukewarm tea (Chinese tea, caraway-seed, fennel, peppermint, anise-seed tea) and gruels ; later, bouillon with yolk of egg, softened stale white bread or biscuit, red wine, and water that has either been allowed to stand or has been boiled (fresh cold water and carbonated waters are best avoided). As soon as the diarrhea is over, the patient should be given boiled calves' brains or thymus, aspic, chicken hash, scraped beef, mashed potatoes, and, as beverages, cocoa, chocolate, coffee, or milk ; a gradual return to the ordinary articles of diet should be permitted, but for a considerable time fruit, vegetables, flour dishes prepared with yeast, fat and acid articles of food should be avoided.

Treatment for the relief of the symptoms (*indicatio symptomatica*) begins with the administration of remedies intended to place the intestine at rest ; in part these remedies still fulfil the *indicatio morbi*. They act indirectly by enabling the organism to carry on the curative process under ideal conditions. The two chief remedies for this purpose are heat and opium. When there are fever, frequent calls to stool, and pain and great lassitude, the patients should be ordered to

go to bed, for they are much better when kept perfectly quiet and warm. Dry or moist hot compresses should be applied over the abdomen. In catarrh of the intestine due to catching cold, diaphoresis (a hot bath) may be valuable. In some cases a so-called Priessnitz compress exercises a very beneficial effect.

If there is no reason for thoroughly emptying the bowel, opium is by far the best means of stopping the diarrhea, as it inhibits the peristaltic movements of the bowels and relieves the pain. It is well not to give doses that are too small, though one ought, at the same time, to consider the idiosyncrasies of the individual. I wish particularly to emphasize the fact that the constipation which usually follows the administration of opium in these cases should not be treated with laxatives even if it lasts for two or three days, but should be allowed to disappear naturally. The same rule applies to the treatment of that form of constipation which frequently follows catarrh of the intestine when it ceases spontaneously without the administration of opium. Opium may be given in its solid form, as the extract, the tincture, or in the form of Dover's powder (pulvis ipecacuanhæ et opii); it may also be given in the form of suppositories or in a starch enema. When, in exceptional cases,—for instance, in very profuse diarrhea accompanied by violent vomiting,—opium cannot be given *per os* or *per anum*, subcutaneous injections of morphin should be given instead.

In mild simple catarrh of the intestine due to catching cold no drug at all is needed. Rest, possibly in bed, careful regulation of the diet, a hot foot-bath, a Priessnitz compress, or, if the pain is violent, a poultice over the abdomen, or, finally, if there is much flatulence, some warm carminative tea, are sufficient.

If symptoms of collapse appear, the extremities should be rubbed and wrapped in warm cloths; at the same time spiced wine or hot tea with brandy should be given, and subcutaneous injections of camphor administered. In very exceptional cases, obstinate acute catarrh of the intestine will require treatment with so-called styptics; as the indications for the administration of this class of drugs are the same as in chronic catarrh, and as these remedies will be dealt with in the treatment of the latter condition, reference should be made to that section for the details (p. 202).

TREATMENT OF CHRONIC CATARRH.

In chronic catarrh the cause of the disease is even more difficult to remove; in other words, the *indicatio causalis* can rarely be fulfiled. This is clear from a study of the etiology of chronic catarrh. In treating this condition a careful clinical history should be taken and every detail of each individual case carefully studied. Occasionally certain changes in the mode of life and in the diet of the patient exercise a beneficial effect on the course of the disease; for instance, by changes in the proportion of water taken in the usual articles of food, and by obviating repeated exposure to cold under certain conditions, etc.

Particular attention must be given to the avoidance of constipation in chronic catarrh of the intestine. A description has been given of a form of this disease in which constipation and diarrhea alternate. In this condition the periods of constipation should be curtailed as much as possible, and fecal accumulation prevented by the application of the proper methods (preferably by enemas). The rationale of this treatment, of course, is that the retention of fecal matter, even for a short time, constitutes an irritant to the intestine when it is in a state of increased irritability, from chronic catarrh.

The more chronic the catarrh, the more difficult is it to obtain a cure. As a matter of fact, a complete *restitutio in integrum* is impossible from a structural point of view when the changes are of long standing. At the same time, treatment need not be purely symptomatic, and a rational application of therapeutics can do more than remove certain symptoms : it can, under certain circumstances, influence the primary process that produced these symptoms. The most important therapeutic measure is the regulation of the patient's general régime and diet.

Occasionally a patient with chronic catarrh complicated by violent diarrhea should be instructed to remain in bed ; but generally a moderate amount of exercise may be permitted. Physical fatigue, however, should be avoided, as it seems to act unfavorably when there is a tendency to diarrhea. The same applies to violent emotions, for an exacerbation of the symptoms frequently follows some emotional disturbance. It is also important to avoid wetting of the surface of the body and exposure to cold, so that an abdominal ["cholera"] belt is a useful prophylactic.

If the catarrh is accompanied by diarrhea or by alternating attacks of diarrhea and constipation, the following diet should be avoided :

Beverages.—Beer ; mild, slightly acid, white table wines ; all sweet wines ; champagne, lemonade, and, as a rule, the stronger carbonic waters. Particular attention must be given to the drinking-water. If infected in any way or degree, it must positively not be drunk. Yet the best of water is not tolerated by some patients, as, for instance, some cannot bear the excellent Vienna spring-water. Some patients can drink the waters at one place but not at another. When traveling, such individuals should drink only boiled water or some deoxidized natural mineral water, such as that of Bilin, Krondorf, Selters, Ems, Borschom, Vichy, Nocera, and others of a similar nature.

The following articles of diet are permissible : Weak tea (without sugar), coffee (when severe gastric symptoms are present coffee had better be replaced by tea), cocoa, all red wines with the exception of the very sweet varieties, as certain Dalmatian, Greek, and other wines, heavy white wines (Rhine wines, Palatine wines, and dry Tokay, etc., only, however, in very small quantities—*i. e.*, a wineglassful at a time) ; brandy, rum, arac, Sliwowitz, and other forms of liquors with the exception of sweet wines ; water, provided it is very pure and good.

Milk occupies a peculiar place in the dietary of these cases. At times it is tolerated very well, at others not at all, so that we must con-

sider the idiosyncracy of each case. Again, an exclusively milk diet is at times successful. Schmidt has tried to explain the bad effect in some cases of milk, by far the mildest and least irritating of all foods, on the hypothesis that it is due not to the materials already in the milk itself, but to their decomposition-products. To retard this decomposition he advises boiling the milk with salicylic acid 0.25–0.5 to the daily quantity of $1\frac{1}{2}$ to 2 quarts. The mucilaginous foods (oat-, rice-, and barley-gruel) are useful in copious diarrheas, as is also whortleberry wine. Liquids should never to be taken ice-cold, and ices of all kinds should be avoided.

Solid Food.—The following articles of diet are to be strictly interdicted : All varieties of fruit (raw or cooked), the great majority of vegetables, salads, fat and sweet flour dishes and baked foods, fat meat, rye bread, dishes containing much sugar, acids, and acid articles of food. The following articles are permissible : Bouillon, soups mixed with gruel or flour (but without the addition of vegetables), soft-boiled and raw eggs and ordinary egg dishes that are not too fat, good fresh butter, calves' brain and thymus, tender meat so prepared that it is soft, preferably hashed, a little fish and oysters, but not lobster. The following vegetables may be given : Mashed potatoes, rice, sago, groats boiled soft in milk, occasionally macaroni. Simple sufflés made of maize, but not too sweet or too fat, and prepared without yeast ; rolls and stale white bread.

It is, of course, impossible to enumerate here all the different articles of diet that are permissible or non-permissible. I wish to give merely a few general directions. The preparation of the food is of great importance ; the simpler the preparation of all the permissible articles of diet enumerated, the better for the patient. Complicated products of the culinary art are usually harmful. It is not to be expected that every physician shall be an expert cook, but it is reasonable that he should know and be able to indicate the general principles of cookery to be applied in particular cases. For comprehensive information on diet and preparation of foods we refer to special works on the subject (Wiel, Wegele, Penzoldt, Ewald, Boas, Hedwig, Heyl ; see also Literature). The choice of foods will frequently occasion most bizarre effects, about which the patient's personal experience is the only guide.

In some instances it is necessary to begin with a very strict dietetic régime, or even to have recourse to artificial foods, such as the so-called prepared infants' foods. Schmidt emphasizes the fact that in all irritable conditions of the intestine it is not sufficient merely to divide the food finely, but that " all foods must be administered in a state of finest division." That the smallest cellulose particles may at times prove harmful will show the necessity for care. Schmidt is right again when he insists on the same precautions with medicinal remedies, particularly subnitrate of bismuth.

Another important point for consideration is the number of the meals and the quantity to be eaten at each. In general it may be said that the frequent and small meals are best.

I need hardly emphasize the fact that in catarrh with pronounced constipation the dietary restrictions formulated above may be somewhat relaxed and the diet somewhat extended. A careful regulation of the diet and rigid persistence in the restrictions indicated are undoubtedly the most important part of the treatment of chronic catarrh of the intestine. As soon as the patient deviates from these rules and commits some error in diet, he is invariably punished by the exacerbation of the process.

A course of mineral waters (either alkaline or saline) is a very popular method of treatment in chronic catarrh of the intestine. Experience has shown that certain mineral waters do, in fact, exercise a very favorable influence on the course of chronic enteritis. In simple diarrhea without structural changes in the intestine such a course of waters is usually without any effect; in simple habitual constipation it acts merely by promoting evacuation of the bowels for the time being. In true catarrh of the intestine, however, certain courses of water, as we know from experience, exert direct beneficial effect not only on some of the symptoms of the disease, but also upon the underlying morbid process itself. The good effects are much more conspicuous when the patient takes the course of water at the spa. Here many other factors assist in the cure: first and foremost the patients are obliged to adhere to certain dietary regulations; in addition, they seem to find it easier to restrict their diet at a spa than at home. At the same time the waters themselves certainly exercise a beneficial effect, for we know that waters of this kind taken at home are very effective and possess a direct curative value. It is not definitely known how this good effect is brought about—whether the structural changes are really favorably influenced by the waters or whether their alkalinity acts symptomatically by altering the chemic constituents of the intestinal contents, by influencing bacterial decomposition, or by stimulating peristalsis.

Carlsbad water is the best. It is specially indicated when a tendency to diarrhea is the most prominent symptom. It acts beneficially both on account of its chemic constituents and the high temperature of the Carlsbad spring. Vichy comes next to Carlsbad. The least beneficial of all is Marienbad, which is chiefly of use in cases of pronounced constipation. Between these two extremes come Kissingen, Tarasp, Homburg, and a number of other less popular waters. Patients with catarrh of the intestine complicated by constipation and anemia frequently go to Franzensbad and Elster.

In using Carlsbad water particular attention should be paid to the method of administration. Small quantities should be taken twice a day, or very small quantities (25 to 50 gr.) three to five times a day. Another factor of importance worthy of special note is that Carlsbad water can benefit patients with some chronic disease of the intestine only when several courses of water are taken during the year, and not merely a single course of four weeks once a year. The latter method is usually adopted, but it is much better for these patients to drink

Carlsbad water for one month at least four times a year; this, of course, can be done only at home. Since the disease is chronic, the treatment must be prolonged.

[Hemmeter [1] speaks highly of the beneficial effect of the waters at Saratoga Springs, which contain calcium and magnesium carbonate as well as sodium chlorid, and points out that if more scientific attention was paid to dietetic treatment there, Saratoga would prove a dangerous rival to Carlsbad and Vichy. He also recommends Hot Springs, Va., the water of which is hot, contains magnesia, and Bedford Springs, Pa.—ED.]

The value of various kinds of baths is small in the treatment of diarrhea; warm mineral baths, or baths to which pine-needle extract, mud, bran, or infusions of different herbs have been added may be given. Cold baths, however, are harmful in some respects, and should be avoided unless special precautions are taken. This, of course, applies only to baths taken by the patients themselves; methodic cold-water treatment, on the other hand, carried out in a well-conducted institution, very frequently exercises a most beneficial effect on the course of the disease. Among the available hydrotherapeutic measures mention may be made of the so-called Priessnitz compresses applied to the abdomen, and wet abdominal binders (" girdle of Neptune "). Applications of this character can easily be made at home, but it is always better and more advisable for the patients to enter a sanitarium in which facilities can be found for carrying out a hydropathic cure. This is particularly advisable since the various necessary manipulations and procedures, such as wrapping and packing, cold rubs, douches, baths, etc., are rarely carried out in a correct manner in general hospitals and clinics (at any rate at the present time, although it is to be hoped that the future will work changes in this respect), or at home.

A visit to the mountains frequently exerts a favorable influence on the course of chronic catarrh of the intestine; some good effect may also be obtained from a trip to the seashore, or, in fact, from any change of scene.

Direct medical treatment has two objects in view: In the first place, to counteract certain symptoms; in the second place, to exert a direct effect on the anatomic process. It is very doubtful whether drugs can do anything toward promoting a cure of the primary lesions that cause the catarrh. It is usually believed that the remedies used with this object—namely, the so-called astringents—act on the catarrhal mucosa and at the same time relieve the symptomatic diarrhea. Relying on this idea, it is customary to give certain metallic salts, among them lead acetate and sulphate of zinc (although these two remedies are not so popular now as they were formerly) or the nitrate of silver (0.2–0.3 : 200) in the dose of three to five dessertspoonfuls a day. The last remedy is the most popular one at the present time, and its administration is frequently followed by some improvement. The astringent drugs containing tannin belong to the following group.

[1] Hemmeter, *Diseases of the Intestines*, 1901, vol. i., p. 474.

This second group of preparations probably exercises no direct effect upon the diseased mucous membrane of the intestine, but diminishes the diarrhea through the inhibitory action exerted by the drugs on abnormal fermentation processes going on in the intestine. In this sense they indirectly aid in the cure of the primary process. The most important drugs of this class are the subnitrate and the salicylate of bismuth ; these preparations can be used in chronic catarrh, as well as in intestinal ulcerations, but only when larger doses of 1.6 are given from three to five times a day. Bismuth has a very powerful constipating effect, both when given alone and in combination with opium. Care should be exercised, however, not to continue its administration too long in cases of prolonged constipation. Tannic acid itself, and the large number of drugs containing tannic acid (calumba, cascarilla, rhatany, catechu, kino, lignum campechianum, fructus myrtilli), all exert a constipating effect, but only in this sense that the tannic acid contained in all these preparations possesses distinct antiputrefactive properties, and so inhibits the formation of decomposition-products in the intestine that would ordinarily irritate the mucosa and set up diarrhea. It is chiefly useful, therefore, in diarrhea due to the abnormal decomposition of the ingesta in the intestine. Tannic acid itself should always be given in small doses (0.05 to 0.2) and never for too long a time. Recently tannigen (acetyl tannin) has been recommended in chronic diarrhea, particularly in cases that are the result of catarrh of the intestine or of ulcerative processes in the bowel, as well as the really effective tannalbin and tannocol (each in dose of 0.5 to 1). The dose of tannigen is 0.2 to 0.5, to be given three times a day. To the same group of remedies belong the preparations of coto. Cotoin should be given in doses of 0.05 to 0.1, either as a powder or as a gum-arabic emulsion. A dose of paracotoin should be twice as large, and of the tincture of coto 20 drops should be given (all these preparations should be given once or twice a day). In addition all the remedies mentioned on page 199 may be used, where the reader will find the description of these remedies and the indications formulated for their use. Lastly, calcium carbonate, phosphate, and salicylate, in $\frac{1}{4}$ to $\frac{1}{2}$ per cent. solutions in carbonized water, must be mentioned.

When the large intestine is chiefly or exclusively involved, irrigation of the bowel is extremely useful. This method of treatment I can recommend very strongly, for I have found that the application of the various drugs mentioned to the bowel direct by irrigation is most useful. The method was evolved by Hegar, who described the well-known technic employed for the injection of medicated water into the bowel. The following drugs may be employed for irrigation (the choice of the drug, of course, will vary according to the indications in any given case) : Silver nitrate should be given in the strength of 0.2–0.5 : 1000 ; tannin in the strength of 2–5 : 1000 ; boric acid in the strength of 5 : 1000 ; salicylic acid in the strength of 1 : 1000. Strauss recommends injections of the extract of whortleberries, which he claims should take precedence over the decoction of whortleberries taken *per os*, as introduced by Winternitz. In some instances irrigation of the intestine with plain water,

or, better still, with a 0.6 per cent. salt solution, is a useful procedure and mechanically aids the evacuation of the bowel contents. If there is much pain, the temperature of the irrigating fluid should be high (105° to 110° F.).

Let me again emphasize the caution that prolonged obstinate constipation should never be allowed in chronic catarrh of the intestine. It is hardly necessary to throw out a warning against the use of energetic, so-called drastic, purgatives in the treatment of this condition. The most appropriate methods of combating a tendency to constipation are simple irrigations of the intestine or enemas ; if, for some reason or other, irrigation of the lower bowel cannot be performed, one of the bitter waters that usually act in small quantities, calomel, or castor oil should be administered.

Under certain circumstances it may be necessary to give opium in cases of chronic catarrh—for instance, when the diarrhea is so profuse that it must be arrested, or when the pain is so severe that it has to be stopped. The administration of this wonderful remedy, of course, should be carefully supervised and limited to the treatment of very definite and urgent conditions, otherwise the patient may acquire the opium habit. Occasionally a congeries of symptoms develops in morphomaniacs that points to involvement of the intestinal tract, but that is not due to the original catarrh, but to the abuse of opium. If necessary, the pain of catarrh of the intestine may be symptomatically treated with preparations of belladonna, but the latter can hardly replace opium.

[Hemmeter has often obtained toxic effects from the use of belladonna in chronic intestinal catarrh, but never any good results. He prefers the denarcotized extract of opium.—ED.]

If there is severe meteorism, it should be treated according to the principles enunciated on page 145.

INFLAMMATION OF THE RECTUM (Proctitis).

A few paragraphs dealing with inflammation of the rectum are necessary, as this affection in some respects assumes an independent character and differs in some particulars from inflammation of other portions of the intestinal tract ; certain differences can be observed as regards the etiology, the anatomic conditions, and the effects of treatment. It is true, of course, that catarrhal and inflammatory affections of one part of the bowel correspond in all essential features with those of other parts ; nevertheless there are certain differences, and I will devote this section merely to calling attention to those points which give inflammation of the rectum a certain independence ; all the statements in the section on Catarrh of the Intestine apply with equal force to catarrh of the rectum, and form the basis of the following description.

ETIOLOGY.

The factors which produce inflammatory and catarrhal lesions of the upper portions of the intestinal tract, such as chemic irritants formed from the ingesta or developed from the decomposition of food, as well as bacteria and bacterial poisons in certain infectious diseases, do not exert so deleterious an effect on the rectum. On the other hand, there is a group of factors which, though not prone to affect other parts of the alimentary canal, is especially apt to cause damage in the rectum. These factors act locally and do harm mechanically or chemically. Scybala are the most prolific cause of inflammatory lesions of the rectum, and when for any reason they are retained in the rectum or its ampulla, may bring this about in two ways : by producing catarrh of the rectum or by leading to the formation of piles (for the relationship between these two lesions see also the section on Hemorrhoids). Other local causes that can do damage are irritating enemas, injuries produced by the insertion of the syringe-nozzle, and, finally, the insertion into the rectum of foreign bodies of any kind or description.

In the case of the rectum gonorrheal infection may play an important rôle in producing inflammation in male subjects who indulge in unnatural coitus. [Gonorrheal proctitis in the male has been set up by using an enema syringe previously employed for douching in gonorrheal vaginitis (Martin [1]).—Ed.] In women gonorrheal infection may spread from the vagina, from the perineum, or when the glands of Bartholin are infected by a fistulous connection between them and the rectum. [Baer [2] found that gonococcal infection of the rectum occurred in 38 per cent. of women with gonorrhea, though symptoms were often absent.—Ed.]

Occasionally an obstinate form of proctitis may be due to the local irritation of oxyuris, a condition which may remain unrecognized ; the females of this species of parasite find a frequent habitat in the rectum, and, it is believed, produce pathologic irritation of the mucous lining of this part by performing boring movements with their cephalic extremity. [Bilharzia may set up irritation in the rectum ; Bowlby [3] has recorded a case in which papillomatous growths composed of loose fibrous tissue rich in cells and evidently of inflammatory origin containing ova of the parasite were removed from the rectum of an Arab. —Ed.]

It is generally believed that the direct application of cold to the rectum or anus (sitting on cold damp ground, etc.) may cause proctitis. Boas doubts this ; I have no personal experience bearing on the subject. Inflammation of the rectum is often secondary and develops in the train of other diseases, such as some forms of intestinal ulceration, carcinoma, hemorrhoids ; while occasionally morbid conditions starting in neighboring organs spread by extension to the rectum, especially some diseases of the bladder and sexual organs.

[1] T. C. Martin, in Hemmeter's *Diseases of Intestines*, vol. ii., p. 615.
[2] Baer, *Deutsch. med. Wochenschr.*, 1897.
[3] Bowlby, *Trans. Path. Soc.*, vol. xlii., p. 136.

[Proctitis may also be due to dysentery, syphilis, septic infection after operation, damage from impaction of the child's head during child-birth, and may be associated with retroversion of the uterus. Actino-mycosis of the anus is a very rare condition, but it has been observed and may be due to infection from above—viz., from food taken by the mouth, spread of infection from adjacent organs, or in most exceptional cases to an ascending infection, the fungus being introduced directly into the rectum from without (Poncet [1]).—ED.]

ANATOMY.

The changes of the mucous membrane of the rectum in proctitis are in all essential features the same as those seen in catarrh of other portions of the intestinal tract; this applies even to the formation of catarrhal ulcers. One peculiarity of proctitis, however, is that the inflammatory process shows a tendency to involve deeper tissues and to extend down into the submucosa, the muscular coats, and even into the periproctal connective tissues; in this way there is a tendency to the development of periproctitis. This is due to the fact that the rectum is more accessible to pyogenic bacteria, which enter the bowel from without and infect the inflamed membrane. It will readily be understood, therefore, why in inflammation of the rectum pus is often formed in addition to mucus, and that the production of pus is more common in rectal catarrh than in catarrh of other portions of the bowel. I have already mentioned in a previous section that in chronic proctitis hemorrhoids quite frequently develop secondarily.

CLINICAL FEATURES.

The course of proctitis may be acute or chronic. In the acute form all the symptoms characteristic of acute catarrh of the intestine in general are observed, namely, increased secretion of mucus, changes in the character of the stools, pain, and often fever. The following factors determine the local diagnosis of acute proctitis :

The pain is experienced deep down in the pelvis and in the perineal region; it is often exceedingly severe, and always well pronounced. If the inflammatory process involves the anus, this part of the rectum as well as the perineum may become very tender on pressure; if the inflammation extends in an upward direction, there may be tenderness in the left iliac fossa.

The most characteristic symptom, however, is tenesmus, that may occasionally become so severe as to be almost unbearable. The patient, while suffering from burning, stabbing, and pressing sensations, has a strong desire to defecate, but when an attempt is made, does not succeed in passing any of the contents of the bowel except occasionally a little blood-stained mucus or pus. Vigorous straining usually causes protrusion of some of the mucous membrane or of piles from the anus ; which, it is hardly necessary to point out, is accompanied by the most excruciat-

[1] Poncet, *Lyon Médical*, Oct. 2, 1898, vol. lxxxix., p. 137.

ing pain. In very aggravated cases tenesmus may be practically continuous and be absolute torture. In addition there may be spasm of the sphincter, a condition that in itself is extremely painful, and further makes any manipulation of the anal region so difficult and painful that digital examination of the diseased parts is practically impossible. Violent strangury, retention of urine, erections, and pain radiating into the abdomen are more remote symptoms of proctitis.

Proctitis has no direct effect on the formation of the feces; occasionally, however, there is so copious a secretion of fluid into this portion of the bowel that the fecal material in the rectum is very greatly diluted and the motions are thin and watery. Mucus is always present and mixed with the stools, whether they are solid, pultaceous, or liquid. The mixture of mucus and fecal matter is, however, never very intimate in proctitis; on the contrary, the mucus is separable from the fecal material proper, or may even be passed separately. Blood is often found in the dejecta—*i. e.*, in the mucus or the pus passed in the motions. It must also be remembered that the amount of pus passed is more considerable in some forms of proctitis than in others; in the gonorrheal form of proctitis, for instance, there is a much greater amount of pus than in any of the other forms. The frequency of defecation is not, as a rule, increased, notwithstanding the existence of tenesmus; on the contrary, we frequently find that these patients, although suffering from aggravated forms of tenesmus and though feeling a very urgent and almost continuous desire for stool, become constipated.

Digital examination is rendered impossible or at least very difficult by the spasmodic contraction of the sphincter muscle; moreover, this examination with the finger or the proctoscope is extremely painful. On digital examination the rectum will be found to be very hot; when the affection has extended deep into the tissues, the mucous lining of the rectum is less movable than normally, and the rectum will, moreover, be felt to be hard and rigid to a certain extent throughout, the wall of the bowel in this portion feeling tough and solid. In cases in which phlegmonous inflammation is beginning to develop in the periproctal tissues (periproctitis), the swollen condition of the circumrectal parts and the infiltration of the affected area can usually be felt on digital examination of the rectum. With the speculum it will be seen that the inner surface of the rectum is deep red in color and is covered with mucus; occasionally small hemorrhages and superficial erosions of the mucosa may also be seen. In view of the fact that acute cases of inflammation of the rectum are so easy to diagnose, digital exploration and an examination with the speculum seem quite unnecessary. It may possibly be necessary in certain cases to perform these methods of examination in order to gain information as to the etiology of the inflammatory process.

The symptoms of the chronic form of proctitis are the same in kind as in the acute form, but differ in degree; the pain and the tenesmus are not of the agonizing character, and, as a rule, blood is also absent from the stools, excepting in cases complicated with hemorrhoids.

In cases running either an acute or a chronic course, paresis of the sphincter may develop. This may be due to fatigue of the sphincter muscle as a result of long-continued spasmodic contraction, or it may be due to true inflammatory changes in the substance of the muscle. In cases in which the closure of the sphincter becomes incomplete or even in cases in which the muscle is still capable of performing its function in a normal manner, there may be exudation of secretion from the anus whenever there is violent tenesmus or desire for stool; the material oozing out under these conditions may, according to its quality, produce burning, itching, or even excoriation of the circumanal region.

The diagnosis of acute proctitis can usually be made without difficulty; the diagnosis of the chronic form, on the other hand, often requires much care and study. The data obtained by inspection of the anal region, examination of the rectum by the finger and with the rectal speculum, the history of the case, and all the other available symptoms must be studied with great care in order to exclude other conditions which might possibly lead to confusion in the diagnosis, such as hemorrhoids, fissure, carcinoma, syphilitic ulcers, diphtheria of the rectum, dysenteric or tuberculous ulceration of the rectum. It need hardly be mentioned that under certain circumstances any of these conditions may coexist with a catarrh of the rectum.

The rectal examinations with the finger and with the rectoscope are often indispensable in order to clear up the etiology of the case; for instance, to detect the presence of foreign bodies in the rectum or of ingesta, etc.

The prognosis of acute proctitis, especially when the disease is the result of "catching cold" or of some mechanical injury, is in general favorable; if the case is properly treated and if the patients live in a correct manner, the inflammation usually subsides within a short time. It is only in those cases which have a very rapid onset or are neglected that there is a tendency to develop the phlegmonous form of proctitis and periproctitis; on the other hand, certain forms of proctitis, such as the gonorrheic form, show a great tendency to run a chronic course. When the disease has become chronic or when the onset of the disease is slow and insidious, the prognosis as regards complete recovery should be guarded—chronic catarrh of the rectum, like chronic catarrh of any other mucous membrane, reacts badly to treatment, not to speak of the obstinate character of the primary disease that may possibly be the cause of the catarrh. In every case of chronic catarrh the condition must be considered an obstinate one. In addition, the possibility of secondary complications must never be lost sight of, such as periproctitic processes, cicatricial strictures, development of hemorrhoids, prolapse of the rectum, and paresis of the sphincter.

TREATMENT.

In the acute forms rest in bed is, above all, essential; as a rule, the pain is so severe that the patients take to bed of their own accord. The best position for the patient to occupy is on the side, or, if possible, on

the belly. At the very beginning of the disease, especially when the initial symptoms are severe, retention of feces should be endured for from two to four days by the administration of opium ; later the accumulated fecal matter must be rendered soft or liquid by the administration of lukewarm irrigations of water or of oil, or by the administration by mouth of castor oil or of salines. In addition the quantity of the food should be reduced, and should be of such a kind as to leave the smallest possible residue and to form excreta that are as non-irritating as possible. Locally, warm applications may be made to the perineum, and warm sitz- or tub-baths aid in relieving the pain. When inflammation is very severe, the application of leeches to the rectum is often soothing and useful. If in spite of all these measures the tenesmus is not relieved, it becomes necessary to administer opium or morphin, preferably as suppositories, or, if this method cannot be employed, internally or by hypodermic injection. In addition, the patient should take warm sitz-baths or whole tub-baths warm. A very efficient means of treatment are irrigations of the mucous lining of the rectal mucosa, for in this way the accumulating masses of mucus and of fecal material can be washed away ; the best irrigating fluids are lukewarm physiologic salt solution, with perhaps the addition of a little tincture of opium.

In chronic proctitis the same treatment and principles should be followed *mutatis mutandis* as in the acute form ; what variations are made should be according to the intensity of the symptoms. In addition, certain astringent irrigations with zinc or copper sulphate, silver nitrate, alum, tannic acid, or boric acid may be given. All these irrigations should, however, be employed only after the rectal mucosa has first been cleansed thoroughly by simple washing. Any ulcerated areas within reach of the speculum may be treated directly by the application of concentrated solutions of silver nitrate.

If local treatment alone is not successful in chronic proctitis, the general measures recommended in the treatment of chronic catarrh of the intestine should be employed (see pp. 198–204), especially systematic cold-water and mineral-water cures (Carlsbad, etc.). Stress need hardly be laid on the self-evident fact that care must be taken to remove any factors which can possibly be the cause of the inflammation of the rectum.

I will not enter into the discussion of anal fistula, of prolapse of the rectum, nor of periproctitis, as these affections properly belong to the sphere of surgery.

PHLEGMONOUS INFLAMMATION OF THE INTESTINE
(Enteritis Phlegmonosa sive Purulenta).

ALL observers agree that this disease is exceedingly rare, and that purulent inflammation of the submucous tissue of the intestine is rarely a primary and an independent process. Personally I have never seen

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a case of this character.   The following case, reported by Bellfrage and Hedenius, is the one most frequently quoted from the literature of the last twenty years.   This patient was a man of fifty-two years who suddenly developed symptoms of peritonitis.   On the third day of his disease physical examination revealed what was apparently an irreducible femoral hernia.   A herniotomy was performed, and the swelling was found to be a fatty tumor.   The patient died on the evening of the same day.   At the autopsy there were peritonitis and a peculiar morbid condition involving 18 cm. of the jejunum, which was very much thickened in the affected area ; the mucous membrane was grayish yellow and covered with a number of light-yellow spots, some of which were superficially ulcerated ; the base of these ulcers consisted of very loose friable tissue infiltrated with pus.   The thickening of the intestinal wall was chiefly due to thickening of the submucous and the muscular coats, both of which were infiltrated with pus.   The submucosa in particular was purulent and in some places was 8 cm. thick.   Moisejew has recently described an apparently similar case : a circumscribed purulent infiltration of the submucous and muscular coats of the jejunum —purulent peritonitis.

Cases of this kind are very rare.   The disease is probably of infective origin ; at any rate, in Moisejew's case streptococcic infection is described.   Nothing definite, however, is so far known with regard to the etiology of the phlegmonous inflammation—in fact, it is impossible to give a correct clinical picture of the disease.   As far as the diagnosis is concerned, I hardly believe that it can be made ; consequently it is impossible to describe any particular form of treatment.

Phlegmonous enteritis, however, is not infrequently encountered as a concomitant or secondary symptom of other diseases of the intestine. It is particularly liable to follow certain forms of intestinal ulceration (compare the section on these lesions), and may be a sequel, for instance, of embolic, dysenteric, tuberculous, and carcinomatous ulcers, or occasionally of intussusception of the bowel and of strangulated hernia.   All these forms of suppurative inflammation of the intestinal wall may independently lead to the development of certain symptoms and may produce certain definite consequences ; nevertheless I believe that during life the clinical picture will be dominated exclusively by the primary disease.

I wish to mention briefly here a remarkable form of inflammation of the intestine which Maragliano recently had an opportunity of investigating.   He saw several cases of this peculiar disease within a few days.   His cases were affected with a form of very acute enteritis entirely confined to the ileum.   It was found that a species of Bacterium coli was probably the primary cause of this inflammation.   The process begins with the formation of hemorrhagic foci between the serosa and the mucosa ; later, ulcerative destruction of the serosa, mucosa, or the whole intestinal wall occurs.   The disease is ushered in by a prodromal stage without fever, but with confined bowels and abdominal pain ; then marked signs of enteritis, with violent abdominal

pain, meteorism, frequently with constipation and vomiting, and occasionally fever, develop. Finally peritonitis from perforation or symptoms of ileus develop. No exact statements can be made in regard to the character of this disease.

[A case recorded by H. F. Harris [1] of necrosis of the mucous membrane of the small and large intestines due to streptococci appears to belong to this group. There were necrosis and hemorrhage in the mucous membrane and extensive infiltration of the submucous coat, with polymorphonuclear leukocytes and red blood-corpuscles.—Ed.]

DIPHTHERIC AND CROUPOUS INFLAMMATION OF THE INTESTINE (Enteritis Diphtherica et Crouposa).

According to modern views, a condition is not spoken of as diphtheria unless the presence of Löffler's bacillus can be demonstrated. If this definition of diphtheria be accepted, the intestine is very rarely the seat of genuine diphtheria. It has long, however, been customary to apply the term "diphtheric" to those forms of intestinal disease considered in this section. This term has been selected to describe the peculiar appearances of the morbid lesions, and I shall adhere to the old view and employ the old term in this section.

Involvement of the intestine in true diphtheria, from the bacteriologic point of view, is exceedingly rare, and will be described in the volume treating of diphtheria. In this section this form will not be dealt with, but the following description will include a number of morbid changes in the intestine that may be due to a variety of other etiologic factors, but present approximately the same pathologic-anatomic aspects. They are all characterized by croupous and diphtheric lesions of the intestinal mucosa, and occasionally of the deeper layers of the intestinal wall. It is convenient to consider all these different forms together, inasmuch as their structural lesions are so much alike.

I wish, however, to lay special stress on the fact that the disease called dysentery ("Ruhr") will be dealt with in another portion of this series and is excluded from consideration in this section.

ETIOLOGY.

It is probable that in all cases of croupous diphtheric disease of the intestine the characteristic anatomic changes seen are produced by bacteria. From the absence of careful bacteriologic examinations this assertion cannot be regarded as proved on experimental or scientific grounds. Cornil, who is also inclined to this view, maintains that the fibrinous membranes formed in the intestine in the severe diarrhea, dysentery and enterocolitis of tropical countries, etc., are due to specific micro-organisms. Cornil based his conclusions on histologic methods of investigation.

Bacteria alone, however, cannot possibly damage the tissues in this

[1] H. F. Harris. *New York Med. Jour.*, Nov. 1, 1902, vol. lxxvi., p. 749, No. 18.

way without some special predisposition on the part of the individual. For the production of such a predisposition the presence of certain factors that possess the power of preparing the soil for the action of bacteria is necessary. Such influences have been called " the primary cause of diphtheric inflammation of the intestine." They vary greatly in character. In the first place, certain chemic poisons belong to this group : Mercury, for instance, prepares the soil for certain intestinal affections that occur in poisoning with this drug ; in uremia the resistance of the intestine is also weakened in such a way that croupous diphtheric lesions or changes may occur.

It has long been known that serious intestinal symptoms occasionally follow the administration of calomel, particularly if it is given for the purpose of producing looseness of the bowels, but does not exercise the expected effect ; while the same symptoms may appear after the inunction of gray ointment and the subcutaneous injection of preparations of mercury. In recent years, since the method of treating wounds antiseptically with corrosive sublimate has been in vogue, intestinal symptoms of this character are observed with relative frequency and seem particularly liable to occur after irrigation of the uterus. I recently had occasion to see a few examples of this kind myself. The name *dysenteria mercurialis* has been introduced into our nomenclature to designate this group of symptoms. It appears that large quantities of mercury are by no means necessary to produce this symptom-complex. Jacusiel, for instance, observed serious intestinal symptoms develop after a single inunction of half a dram of gray ointment that he made himself. Certain circumstances, particularly anemia, general weakness, and possibly an old catarrh of the intestine, seem to favor the development of intestinal trouble. Ziemssen throws out a caution against the administration of opium together with mercury, for he claims that the development of intestinal lesions is promoted by this practice. The most dangerous method of applying mercury, so far as the deleterious effect on the intestine is concerned, is washing out serous cavities and wounds with corrosive sublimate solutions ; second in importance is the administration of mercury subcutaneously.

Two possible explanations for the peculiar effect of mercury preparations on the intestine, even when the drug is applied externally, have been advanced : one theory is that the effect is exercised through the blood ; but to my mind this view is absolutely untenable, inasmuch as the mercury when present in the blood must be in the form of mercuric albuminate, a substance which is incapable of producing inflammation (Rossbach) ; the other theory is that the metal is excreted in large quantities by the liver (Overbeck), and in this way enters the intestine in the bile.

In the course of nephritis the development of intestinal symptoms is frequently seen. The morbid changes in cases of this kind when examined postmortem will be found to vary greatly ; these are either cases of diarrhea in which the intestine is found normal (the mucosa pale), or of simple catarrh that lead to *enteritis hæmorrhagica.*

Both these possibilities have been discussed in the section on Diarrhea and on Enteritis. Characteristic cases of uremia frequently present certain definite lesions of the intestine that correspond to the picture of enteritis diphtherica, and also frequently show extensive ulceration. (For the details of this type of enteritis the reader should refer to the section on Uremic Ulceration of the Intestine.)

Occasionally other factors disposing to enteritis diphtherica are met with : one of the most frequent of these causes is is so-called sporadic dysentery. It has already been pointed out that dysentery is described in another volume of this series, and I shall consequently merely mention the following points in regard to this disease : At the present day three distinct varieties of dysentery are recognized : (1) The endemic or tropical form, which is unquestionably due to the presence of protists (certain species of amebæ) ; (2) the epidemic form of dysentery, which is very probably due to specific bacteria ; (3) the sporadic form. We are concerned only with the latter here.

Virchow [1] was the first to throw some light upon the true pathogenesis of dysentery. With his customary clearness he disentangled the maze of confusing opinions that existed with regard to this disease up to that time. Even now the following statement, made some fifty years ago, holds good. " There can be no doubt that dysentery is due to the action of certain substances that are contained in the bowel contents upon the catarrhal surface of the intestine ; or, to express this more clearly, a simple catarrhal affection of the intestine (which may be called catarrhal dysentery) is exacerbated and becomes dysentery proper (diphtheric dysentery) as the result of the action of certain intestinal decomposition-products." In other words, he lays special emphasis on the significance of stagnation and decomposition of the intestinal contents in the genesis of dysenteric changes in the large intestine. An accurate decision as to the responsible factors has not yet been arrived at. Two possibilities are conceivable : On the one hand, chemic substances that are formed in the decomposing contents of the bowel may produce an inflammation of the intestinal mucosa of a diphtheric character, or certain bacteria may exert the same effect. So far this matter has not been decided.

The above explanation, however, makes it easier to understand the pathogenesis of certain other forms of diphtheric affections of the intestine, particularly the form of enteritis diphtherica that occurs in stenosis of the intestine. At the same time it must be remembered that special conditions must exist in order that diphtheric enteritis should develop in fecal accumulation ; for, as every one knows, there are innumerable cases of very obstinate fecal accumulation in which all evidence of diphtheric inflammation of the bowel is absent. Whether in these cases the drying of the feces prevents the formation of the chemic substances that are necessary, or whether the specific bacteria responsible for diphtheric lesions of the intestine are absent, remains to be proved.

We know from clinical experience that diphtheric diseases of the

[1] *Virchow's Archiv,* vols. ii. and v.

intestine also occasionally occur in cachectic individuals, in subjects with carcinoma, and in cases of phthisis that are very much reduced. It is probable that all these diseases and conditions constitute predisposing factors that favor the development of diphtheric inflammation of the intestine.

Enteritis diphtherica is also occasionally seen as a sequel of a number of acute infectious diseases—for instance, septic conditions, scarlatina, typhoid, cholera, and small-pox. In this form, which constitutes a special etiologic group, the question arises whether the specific microbes or other etiologic factors of these diseases are directly responsible for the diphtheria of the intestine.

[Pneumococcal inflammation of the intestinal tract may give rise to the formation of membrane. Cary and Lyon [1] give the details of a boy who had double pneumonia and wide-spread fibrinous exudation upon the mucous membranes, from which the Diplococcus pneumoniæ was isolated. The mucous shreds were passed by the bowel, and there was tympanites. The boy recovered. Another case of membranous enteritis from which the Diplococcus pneumoniæ was isolated has been recorded by Weichselbaum. [2] In 100 autopsies in pneumonia Osler [3] found that 5 cases showed a thin, flaky exudation on the surface of the mucous membrane.—ED.]

PATHOLOGIC ANATOMY.

The different forms of enteritis diphtherica enumerated above, while varying greatly in their direct or indirect etiology, correspond essentially in regard to their morbid anatomy. The main traits of the pathologic anatomy are more or less identical both in regard to the macroscopic and the microscopic findings. As these pathologic appearances are exactly the same as those of dysentery (both the endemic and the epidemic form), the reader should refer to the description of the pathologic anatomy of dysenteric lesions. In order to avoid unnecessary repetition, therefore, a brief descriptive summary only will be given here.

Diphtheric enteritis in the great majority of cases involves the large intestine alone, either in its entirety or in its lower portions. In rare cases, particularly in the mercurial and uremic form of diphtheric inflammation of the bowels, the small intestine is also involved. The intensity of the process, provided the disease has not developed too far, is, as a rule, greatest in those portions of the bowel in which the bowel contents remain in contact with the intestinal mucosa for the longest time; these situations are: the upper part of the rectum, the sigmoid flexure, the splenic flexure, the hepatic flexure, and the cecum. The process always begins on the summit of the ridges of mucous membrane; in the large intestine it begins on top of the longitudinal ridges

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[1] Cary and Lyon, *Trans. Assoc. Amer. Phys.*, 1901, vol. xvi., p. 379.
[2] Weichselbaum, *Wien. klin. Wochenschr.*, 1890, vol. iii., p. 187.
[3] Osler, *ibid.*, Philada., vol. xii., p. 188.

of the teniæ and the transverse plicæ sigmoideæ. This localization of the earliest changes is absolutely identical with the conditions seen in dysentery. In the small intestine the process begins on the summit of the valvulæ conniventes (valves of Kerkring) and on the tips of the villi.

In the early stage the process presents the appearance of a simple catarrh of the intestine, especially on microscopic examination ; in some cases, however, the diphtheric changes are not ushered in by the catarrhal stage, but the appearances of enteritis diphtherica of the mucosa are presented at once. The mucous lining in these cases is covered with a grayish-white membrane ; later there is coagulation necrosis, and the process extends downward into the tissues of the intestinal wall, causing necrosis and ulcerative disintegration of the parts ; finally, large, frequently enormous, loss of substance occurs, leading to the late consequences of enteritis diphtherica—namely, perforation, stenosis, or thickening of the intestine. All these processes, together with the histologic details presented, will be considered at length in the section on Dysentery. The changes seen in mercurial poisoning are so similar to these appearances that a separate description is hardly required ; the only difference between the enteritis seen in mercurial poisoning and in other forms is that in the former case the small intestine is more frequently involved than in the latter.

CLINICAL FEATURES.

When secondary croupous or diphtheric affections of the intestine arising in the course of some severe infectious disease remain moderate in severity, they may remain entirely latent and undiscovered, for the symptoms of the primary disease distract attention from the symptoms produced by the secondary complication. When, however, the enteritis assumes more intense degrees of severity, it produces certain distinctive and prominent symptoms. No detailed account of these symptoms will be given here, since they will be described at length in the section on Dysentery, as the conditions now under consideration are identical with those of dysentery. The most characteristic feature of this complication is the change in the stools. The evacuations are frequent, there is excessive tenesmus, and the stools are very thin. The feces are mixed with mucus, blood, and pus, or may consist of the latter materials alone ; quite commonly they contain shreds of tissue. In many cases of chronic diphtheric inflammation of the intestine, particularly in carcinomatous, tuberculous, and other cachectic individuals, the evacuations are quite liquid, gray, or almost white, so that the feces give the impression that they consist of pus mixed with water. As a matter of fact, examination of these dejecta shows the presence of enormous numbers of pus-cells. Cases of this kind have also been called *blennorrhœa intestinalis.* I wish to point out again, as already stated on page 188, that purulent feces are never passed in catarrhal enteritis, but are seen only in the diphtheric form of inflammation of the bowel.

The course of the disease varies greatly, as everything depends on the etiologic factors producing it. In the secondary form of diphtheritis of the intestine that is seen in cachectic subjects, the course of the disease is slow and insidious. In mercurial dysentery following irrigation of the uterus with corrosive sublimate, very violent symptoms may appear within twenty-four hours. The prognosis is serious under all circumstances; it is only when the process is circumscribed and limited to a small portion of the bowel that there is any prospect of a complete restitution to normal and freedom from incurable sequelæ. Very frequently, however, death is caused by general exhaustion, peritonitis, perforation of the intestine, or hemorrhage. Finally, there are a number of cases that recover from the diphtheric process itself, but suffer for the rest of their life from the formation of cicatricial tissue and the resulting strictures of the intestine.

Reference should be made to the sections on Dysentery, Ulceration of the Intestine, and Proctitis for the therapeutic measures to be employed in these cases.

ATROPHY OF THE INTESTINE (Atrophia Intestinalis).

ATROPHIC conditions of the intestine have received incidental description and passing notice from many observers, among them Klebs, Leube, Werber, Kussmaul and Maier, Kundrat, Woodward, Habershon, Damaschino, Fenwick. In 1882 I published a systematic description of it, the material part of which—that is, the simple description of the condition—is still admitted to be correct; but since Gerlach's work on the subject in 1896, a complete reversal of opinion has taken place regarding the interpretation of the appearances described. They are no longer regarded as the results of pathologic processes, but merely as a *pseudo-atrophy*, due mainly to intestinal flatulence, either during life or postmortem.

Despite this altered view, I shall give a short description of the anatomic changes I have described as *atrophic conditions of the intestinal canal*. In conclusion I shall then detail the views recently entertained by other investigators.

The subject of atrophy of the intestine will be more readily and clearly understood if atrophy of the different anatomic layers of the intestinal wall is considered separately.

1. **The Epithelial Layer.**—Atrophy of the epithelial layer of the intestine as an isolated change does not occur. The changes in the epithelium considered in the sections on Acute and Chronic Catarrh of the Intestine are quite within the limits of these processes. It is true that epithelial cells are never seen on the free surface of the mucous membrane of the large intestine when this portion of the tissue is microscopically examined. The same applies to the duodenum and the ileum, for a continuous layer of epithelium is found only in exceptional cases, and then only in small areas of the bowel, especially on the villi

or on the mucous membrane lying between the villi. This absence of epithelial cells, however, is without doubt a simple postmortem phenomenon. The epithelial layer of the intestine is found to be absent in the bodies of individuals who die at all ages and of all diseases, and even though the intestine itself is otherwise perfectly normal. Desquamation of intestinal epithelium occurs very rapidly after death. I have been able to verify the fact that it occurs within six hours after death; control experiments performed on dogs lead to the same result.

2. The Mucosa Proper.—What has been said concerning the epithelial layer also applies quite generally here (Klebs, Leube, Kundrat, Werber, and others), but more particularly to the changes occurring during childhood, and affecting the villi, glands, and follicles. I have also described the atrophic state of the mucosa to be seen in adults.

Atrophy of the mucous membrane of the intestine is by no means easy to detect by simple inspection. In very advanced degrees of atrophy the inner surface of the bowel looks perfectly smooth, and when carefully scrutinized, the fine reticulated appearance presented by the normal mucosa, due to the presence of the glands of Lieberkühn, is not visible. In older cases of atrophy the intestine looks pale and is occasionally distinctly thinner than normal, especially when the other layers of the bowel are also involved in the atrophic process.

In the most advanced degrees the glands of Lieberkühn are entirely absent. All that the mucosa consists of is a matrix of connective tissue that is darkened here and there with blood-pigment and contains isolated round-cells. In very old and very advanced cases the latter may also be absent. At the same time the thickness of the mucosa is found to be greatly diminished: instead of being 0.375 to 0.5 mm. (normal) in thickness, it may only be 0.25 to 0.1 thick. In one case I found it to be only 0.012 to 0.024 mm. thick. Here the tissue that corresponded to the mucosa consisted merely of a cicatricial tissue. When atrophy is not very far advanced, the crypts of Lieberkühn are absent only in certain portions of the mucosa. On careful examination a number of glandular tubules in process of disintegration and desquamation are seen. Microscopic examination of the mucosa will show the presence of numerous round-cells; the mucosa may be of nearly normal height. The villi of the small intestine degenerate *pari passu* with the atrophy of the glands; they shrink and finally become very small. In advanced cases they will be found entirely absent in a number of fields or even in whole sections of the intestine, so that occasionally a piece of atrophic duodenum may in all respects resemble a piece of atrophic colon.

Atrophy of the mucosa is more frequently seen in the colon than in the small intestine. The cecum is the portion of the bowel most often affected, and then the first portion of the ascending colon. Another portion of the intestine that is probably affected in as many, if not in more, cases than the ascending colon, is the lowest portion of the ileum —close to the ileocecal valve; then, in a descending scale, the other portions of the large intestine, and lastly, the upper portions of the

ileum. The jejunum is very rarely involved. In rare and isolated instances the atrophic process extends without interruption from the rectum upward to the lower portions of the jejunum. Occasionally portions of the intestine are involved that are not continuous; in the great majority of cases, however, as already stated, the cecum alone is involved. I examined a great many bodies in Jena, and found that the mucous lining of the cecum was atrophic in 80 per cent. of the adults. According to Scheimpflug, an examination of the autopsy material in Vienna shows that atrophy of the cecum occurs in 96 per cent. of the cases.

3. The Follicles.—It is a well-known fact that the follicular structures of the intestine are frequently destroyed by ulceration—for instance, in catarrh, in typhoid, and in tuberculosis. It is questionable, however, whether simple atrophic loss of follicles can ever occur without the preliminary necrosis. The statements of the various authors who have studied the question of atrophy of the intestine in children (athrepsia, tabes mesenterica), and who have paid particular attention to the follicles in these conditions, differ greatly (Lambl, Hervieux, Werber, Kundrat, and others). Some writers claim that the follicles are hypertrophied, others that they are not changed at all, while others believe that they are atrophied, and a few claim that they are "burst" and atrophied at the same time. Baginsky describes a peculiar appearance. He claims that in some cases a few of Peyer's patches protrude above the level of the mucosa; that the follicular structures present the microscopic appearances of partial atrophy, and that they contain very few cells. He expressly states that there is no necrosis to be seen.

I have so far never seen primary isolated atrophy of the follicular apparatus of the intestine in adults. Even if large portions of the mucosa are atrophic, the solitary and agminate follicles are usually well preserved both in number and size.

4. The Submucosa.—The diameter of this coat of the intestine fluctuates within wide limits even in subjects whose intestine is perfectly normal. The diameter of the submucous layer of the small intestine varies from 0.25 to 0.6 mm.; of the large intestine, from 0.35 to 0.75 mm.

Pronounced atrophy of the submucosa may be considered a very rare occurrence. Even when the mucosa is atrophic over large areas— say from the rectum to the upper jejunum—I have still found the submucosa completely normal. I have never, in particular, seen anything in the submucosa (following catarrh of the intestine) that resembled "cirrhosis"; in exceptional cases the submucous layer is somewhat narrower than normal. In these instances it may contain a certain number of round-cells, especially around the blood-vessels and along the muscularis mucosæ.

5. The Muscular Layer.—Atrophy of the muscular coat is next in importance to atrophy of the mucosa. I have published a large number of measurements of the normal intestinal musculature in a special monograph on atrophy of the intestine, but my data appar-

ently differ from those usually accepted as correct. I will, however, limit my remarks to a brief discussion of the subject.

Klebs summarizes the various opinions that have been expressed by different writers on this subject as follows : "Atrophy of the intestinal musculature is found in general atrophy of the wall of the intestine. Under these conditions the process of atrophy is so far advanced that the walls of the intestine become thin and translucent ; the different strands of muscle-fiber become distinctly visible, owing to the fact that they are separated by larger interspaces. This condition is specially noticeable in the chronic catarrh of the intestine that is so frequent in children ; it is also seen in cases of general marasmus depending on phthisis, carcinoma, typhoid, or exhausting suppuration. The walls of the intestine may become so thin that rupture of the bowel occurs from very slight injury."

These statements, repeated and indorsed by many writers with slight modifications, are not correct, at least not in the general form in which they are expressed. My investigations have shown the following to be the case :

In general cachexia (carcinoma, phthisis, typhoid) advanced emaciation, complete loss of the subcutaneous fat, and in cases in which the skeletal muscles are very much atrophied, in which, further, the cecum is the only portion of the intestine where there is atrophy of the mucosa and in which there is no catarrh of the intestine (with the exception of cases of typhoid), atrophy of the intestinal musculature never occurs. We see, therefore, that the conclusions usually drawn from the supposed existence of atrophy of the intestinal musculature in regard to the loss of functional powers of the intestine cannot be maintained.

In chronic catarrh of the intestine without atrophy of the mucosa the measurements of the intestine rarely show much variation from the normal. Occasionally there is slight hypertrophy of the muscular coat.

In atrophy of the mucous lining of the intestine there are no regular and constant conditions in this respect ; here, however, the measurements of the muscular coat are, as a rule, smaller than normal, although no definite proportion exists between the degree of atrophy of the mucosa and of the muscular coat. It is difficult to give a valid and conclusive explanation for the occurrence of atrophy of the intestinal muscularis. The fact that the submucosa remains intact in so many cases of muscle atrophy seriously invalidates the correctness of some of the explanations that have been put forward.

A very important form of atrophy is the independent variety that I have described. This is probably a form of congenital atrophy (or more correctly hypoplasia) of the muscular coat that occurs when the intestine is otherwise normal. I have already spoken of this condition in another place.

In connection with the consideration on atrophy of the intestinal muscularis a few remarks on fatty degeneration of the musculature of the intestine may be added. E. Wagner in particular has studied this lesion carefully. He describes it ten times in 400 autopsies (mostly

in phthisical subjects or habitual drunkards). I carefully examined 50 intestines microscopically in this connection, but found fatty degeneration in only 3 old subjects, two of whom were without doubt drunkards. In the most advanced case of the three the fatty degeneration extended from the jejunum to the descending colon ; in the latter portion of the bowel there were only very insignificant areas of catarrh that were widely disseminated throughout this portion of the intestine. Fatty degeneration of the muscular coat seems to exist independently of other alterations of the intestine—in other words, it seems to be a primary change. Histologically, the ordinary picture of fatty degeneration of unstriped muscle-fibers is presented. The layer of longitudinal fibers seems to be somewhat more involved than the circular. The muscularis mucosæ may also be affected.

6. **The Nerves of the Intestinal Wall.**—Jürgens has described a form of fatty degeneration of the nerves and the muscular apparatus of the intestine that he considers to be an independent affection. He calls the disease "atrophia gastro-intestinalis progressiva." Blaschko, in two cases, found the ganglia and the nerves of Meissner's and Auerbach's plexus and their connecting fibers in a condition of fatty degeneration. In one of the two cases, occurring in a female drunkard, the muscular tissue was at the same time in a state of fatty degeneration. Sasaki has also described two cases of diffuse sclerotic and fatty degeneration of the nervous apparatus of the intestine, together with similar degenerative changes of the muscular layer in one of these cases. Both of his patients died from a disease presenting the clinical picture of progressive pernicious anemia. In addition he found local degeneration of the ganglion-cells of Auerbach's plexus in the immediate vicinity of typhoid follicular and tuberculous ulcers, and near several other local lesions of the intestine.

According to the investigations of Scheimpflug, the histopathologic changes in the nervous apparatus of the intestine (consisting in cloudy swelling, fatty degeneration, necrosis, contraction of tissue, and sclerosis) are not particularly rare. He found this condition in tuberculous meningitis, in croupous pneumonia, in acute arsenical poisoning, in acute yellow atrophy of the liver, and in a variety of chronic diseases that led to general marasmus.

At the beginning of this section I mentioned that a great part of the conditions I described as intestinal atrophy resulting from intestinal catarrh is considered by Gerlach a postmortem appearance. The microscopic aspect of the intestinal mucous membrane is dependent on the contractility of the muscular coat, so that when from any cause this is drawn out thin, the mucous membrane must assume a pseudo-atrophic appearance. The prolapse of Lieberkühn's crypts is also regarded as postmortem by Gerlach, and he remarks that I did not take into consideration sufficiently the postmortem distention and meteorism. This criticism, which, applies also to all earlier writers on intestinal atrophy, I must concede to be correct. Similar views are expressed by Huebner ; also by Habel, except that he believes the loosening and projection of

the Lieberkühn's crypts to be not purely postmortem, but probably favored at least by a previous catarrh. Faber and Bloch and Ernst Meyer have also arrived at the conclusion that the atrophic state of the intestinal canal is a postmortem change due to distention. They worked from the basis of the relation existing between the clinical picture of pernicious anemia and gastro-intestinal atrophy. Even after the appearance of Gerlach's work, Martius and Lubarsch, and also Ewald, confirmed these relations, thus still holding to the belief in the occurrence of a pathologic intestinal atrophy. Faber and Bloch, however, deny it. Finally, at my request, Ponfick very recently had other experiments conducted by Strassman, while Ponfick himself conducted controls. With his permission I reproduce his results in brief. He also confirmed my observations when he examined the viscera more than eight hours postmortem, but, conversely, his findings were the reverse when examination was made from forty-five minutes to seven hours postmortem. Portions of intestine that showed no changes in normal or decomposed state without meteorism (except here and there desquamation of the superficial epithelial layer) showed pseudo-atrophy when artificially distended. The symptoms of pseudo-atrophy are directly proportional to two factors : first, the length of time that has elapsed between death and examination ; second, meteorism, which in the corpse varies with different loops of the intestine. The apparent hypertrophy and atrophy of the glands, as also the varying thickness of the muscular coat, are caused respectively by distention and by contraction of the intestine.

In view of these uniform results it remains my simple duty to acknowledge that the question of atrophic conditions generally needs a modern revision. As explained before, in my previous work I did not take into account the distended state of the intestine. Of course, any criticism of this applies equally to all writers on the subject before me, for they also took no account of it to my knowledge. It remains to be decided, then, whether there exists at all a pathologic atrophy of the intestine. *A priori* it appears possible, when we consider the established occurrence of pathologic gastric atrophy. Personally, I am convinced of it, but the proofs still require further methodic examinations. Naturally, these investigations, to be of real clinical value, must not be based on one accidental finding postmortem, but must be paralleled by careful examinations *intra vitam*. Individual cases I have followed carefully for months, and constructed a clinical picture that seemed to justify atrophy of the large intestine particularly. But as it is impossible to state, after a lapse of twenty years, whether the intestine was distended and what length of time after death the section was made, and, further, the manuscripts being no longer in my possession, I believe it to be for the best to forego at present any further consideration of a clinical nature of intestinal atrophy.

[SPRUE, OR PSILOSIS.

Reference should be made to the tropical disease sprue, or psilosis, which forms a very distinct clinical entity, but is not very easy to localize from a pathologic point of view, as it is somewhat difficult to distinguish between the specific primary lesions of the alimentary canal and the secondary changes which become implanted in the course of this chronic affection. Manson [1] regards the primary change as probably catarrhal, with premature shedding of the epithelium, but extensive superficial ulceration follows and subsequently atrophic changes due to starvation. Definite ulceration of the colon may occur, so that it has been spoken of as chronic dysentery.

Synonyms and History.—The clinical aspect of the disease being striking, a number of names have been applied to it, such as chronic diarrhea, diarrhœa alba, white flux, chronic enteritis, chronic diarrhea of warm countries, chronic or endemic diarrhea of Cochin China, tropical diarrhea, chronic dysentery, cachexia aphthosa, and sprue.

The term psilosis (φιλός bare) was suggested by Thin to describe the bareness or rawness of the mucous membrane. The conception of psilosis or sprue as a distinct disease is largely due to Thin [2] and Manson.

Short Description.—"An insidious chronic remitting inflammation of the whole or part of the mucous membrane of the alimentary canal, occurring principally in Europeans who are residing or have resided in tropical or subtropical climates. It is characterized by irregularly alternating periods of exacerbation and comparative quiescence; a peculiar, inflamed, superficially ulcerated, exceedingly sensitive condition of the mucous membrane of the tongue and mouth; great wasting and anemia; pale, copious, and often loose, frequent, and frothy fomenting stools; very generally by more or less diarrhea, and also by a marked tendency to relapse. Sprue may either be primary or it may supervene on or complicate other affections of the alimentary canal. Unless properly treated, it is usually fatal" (Manson).

The cause of the disease is not known. It has been thought to be due to an intestinal worm and to a specific bacterium, but at present its actual cause has not been established. It chiefly occurs in China, Java, Ceylon, and India.

Morbid Anatomy.—As already pointed out, the changes found in the alimentary canal at the termination of a case are complicated by secondary lesions and do not represent the specific primary condition. Thus at the end of a long case the alimentary canal presents a threefold pathologic condition: (1) The specific and primary lesion; (2) the specific starvation lesion; (3) the secondary irritative lesion (Manson). The primary and specific condition is probably catarrhal and confined to premature shedding of the epithelium and folliculitis, while secondary

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[1] Manson, *Allbutt's System of Medicine*, vol. iii., p. 774.
[2] Thin, *Med.-Chir. Trans.*, 1892, vol. lxxv., p. 285.

inflammatory changes leading to the formation of granulation and scar tissues result from persistent irritation ; the intestine thus becomes useless and starvation results, which gives rise to specific changes in the wall of the bowel. It may be primary and come on insidiously, or secondary and supervene after dysentery. The disease may come on long after the patient has left the tropics.

The intestine becomes thinned and is translucent, and looks as if it had been shaved with a razor ; there may be eroded or ulcerated areas, and in places cystic dilatations of Lieberkühn's crypts. Changes are sometimes found in the pancreas,[1] but are not absolutely constant (Drysdale).[2]

Symptoms.—The bowels are irregular and the motions copious, pale, drab-colored, yeasty, and sickly smelling ; the tongue, mouth, and throat are tender and sore from loss of epithelium and the formation of small herpetic vesicles, hence the term " Ceylon sore mouth." There is dyspepsia, with much flatulence. There are wasting, anemia, and great debility. There is an extremely severe secondary anemia, in which the red blood-corpuscles may fall below two millions per cubic millimeter, and show a low hemoglobin value (Bassett Smith[3]). The temperature is below normal. The disease is extremely chronic and very prone to relapse. If the disease can be thoroughly treated in an early stage before the intestine has become so damaged as to be practically functionless, the outlook is not so gloomy as in cases allowed to run their course.

Treatment.—Manson insists on the importance of physiologic rest as far as possible for the intestine and a strict milk diet at first, on which marked improvement occurs. Subsequently fruit, especially strawberries, has been found to have a good effect. Cantlie[4] recommends a purely meat diet of five ounces of minced beef three times a day, with plenty of jelly between the meals and toast-water to drink.—Ed.]

MUCOUS COLIC AND MEMBRANOUS CATARRH OF THE INTESTINE (Colica Mucosa et Enteritis Membranacea).

Synonyms, Diarrhœa tubularis s. Colitis pseudomembranacea s. Pellicularis s. Tubulosa s. Fibrinosa, s. Catarrhus Desquamativus s. Myxoneurosis intestinalis membranacea, etc. [Mucous colitis.]

Differences still exist as to the interpretation and acceptance of the clinical conditions described in this section, and the characteristic symptom of which is a copious excretion of mucous masses. The history of these affections dates from 1825, when Mason Good first described diarrhœa tubularis. No new data were advanced until Siredey, in 1869, and DaCosta, in 1871, called attention to the nervous element in the condi-

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[1] Bertrand and Fontan, *De l'enterocolite chronique endemique des pays Chauds*, 1887.
[2] Drysdale, *Trans. Path. Soc.*, vol. l., p. 114.
[3] Bassett Smith, *Brit. Med. Jour.*, 1903, vol. ii., p. 641.
[4] J. Cantlie, *ibid.*, 1899, vol. ii., p. 640.

tion. Leyden's work, in 1882, on enteritis membranacea, gave the stimulus to renewed investigation, which was at first confined chiefly to the membranous dejecta and was subsequently directed to the actual nature of the processes.

[Dr. Richard Powell's [1] account (in a paper read in 1818) of certain painful affections of the intestinal canal contained cases of mucous colitis and was the first published account in the English language. For a number of isolated references to the disease before this time, beginning with Bauer in 1747, the reader may refer to F. H. Hawkins' [2] paper. Jonathan Hutchinson [3] showed specimens of casts before the Pathological Society in 1857.—Ed.]

The nature of the membranous and tubular casts found in the dejecta may be considered as definitely settled—they consist of mucus. But concerning the nature of the processes there is still some difference of opinion. The question is, are those cases described in the literature and practice as enteritis membranacea always due to anatomic change of catarrhal inflammatory nature, even though characterized clinically by some features not met with in the ordinary catarrhs; or must we eliminate the catarrhal origin entirely and accept, for all the cases in this category, a nervous hypersecretion of mucus.

In my estimation these questions cannot be decided on clinical grounds alone, but only by the aid of pathologic anatomy.

Working on the clinical aspect, I announced (1884) the belief that a number of conditions termed in practice enteritis membranacea were most likely not of inflammatory catarrhal origin, and suggested the name *colica mucosa* for these conditions. In a later article (1895) in this work I treated them as a distinct process, in which an abnormally large production of mucus takes place from an anatomically sound mucous membrane. I accepted the version of Leube that it might be a neurosis of secretion. But I also emphasized the fact that in other cases also called enteritis membranacea there is found an anatomic, catarrhal, inflammatory change.

This division into two groups—the one of purely nervous origin, the other of anatomic origin, as suggested by me—has been attacked in the spirited discussions of recent years. At present each of three views is maintained as to the origin of the various conditions called in practice enteritis membranacea. They are: (1) Partly of nervous, partly of anatomic origin (Nothnagel, Ewald, Fleischer, Mannaberg, Rosenheim, Schuetz, Hemmeter, and others); (2) nervous hypersecretion of mucus in the intestine (DaCosta, Siredey, Vanni, Potain, Glénard, Einhorn, Westphalen, and others); (3) all of anatomic origin (Boas, Åkerlund, Mathieu, and others). [Von Noorden [4] lays special stress on long-continued constipation as the exciting cause of mucous colic, but insists that it produces the disease only in neurasthenic or hysteric subjects.—Ed.]

[1] Richard Powell, *Med. Trans. Roy. Coll. Phys.*, London, 1820, vol. vi., p. 106.
[2] F. H. Hawkins, *Edinburgh Med. Jour.*, November, 1901.
[3] Hutchinson, *Trans. Path. Soc.*, vol. ix., p. 188.
[4] Von Noorden, *Colica Mucosa* (American translation), 1903.

After this brief explanatory introduction we shall proceed first to the material parts of the subject. To proceed objectively, we must include everything in the literature described under the head of enteritis membranacea, and also its analogues. In conclusion we shall return to the pathologic considerations.

Pathologic Anatomy.—Discussions of this subject would doubtless soon cease and unanimity of opinion be attained if there were more reliable postmortem observations. Unfortunately, the necropsies at present available are very few. They fall into two groups, but are detailed under the general term enteritis membranacea.

One group gave entirely negative results as regards the intestinal wall. O. Rothman for years observed a patient who had always suffered from constipation, and who in the last nine years of his life had characteristic attacks of pain and passage of " fibrinous " masses. Exclusive of a lacerated perforation in the duodenum which led to fatal peritonitis, C. Ruge found " nothing abnormal despite careful examination of the intestinal tract." Edwards' case may be identical with one referred to in the literature under Osler's name, under whose care the patient was.

Male, seventy-one years old, under observation six weeks, died of chronic nephritic and purulent basilar meningitis. No history obtainable on account of unconsciousness. Section of intestine revealed in ascending colon, membranous, restiform mucous masses in abundance. Otherwise absolutely nothing abnormal in intestine. No signs of past or present colitis discoverable. The commentary reads verbatim : " We were unable in any way to connect the existence of the membrane with the patient's condition just preceding death, and can but conclude that it was simply without clinical manifestation, except the passage of membrane in the stools."

Franke's observation in a case, though no necropsy was obtained, is important on account of the conditions found and the clinical history :

Hysterical woman had passed for a long time masses of coagulated mucus with most violent pain. As all other methods had been fruitless, Franke determined on colotomy. The mucous membrane at place of incision "appeared normal. . . I found absolutely no signs of a catarrhal inflammation of the mucous membrane." Improvement from day of operation. Patient passed firm feces, to which only occasionally a little gelatinous mucus was adherent. The patient later refused closure of the artificial anus for fear of a return of her former trouble.

Marchand describes " branching whitish coagula imbedded in viscous mucus in the contracted, almost empty, large intestine exsected from a subject who died of metastatic sarcoma after many months' decubitus " ; of appearances *intra vitam* nothing was known. It is worthy of note that such manifestations are occasionally seen at autopsy in cases in which symptoms of enteritis membranacea are entirely absent.

So far as can be learned, no microscopic examination was made in the foregoing cases. However, there are available a few very accurate ones. One is by Jagic, working under direction of Weigert, from whom also the postmortem findings are taken :

Female, age forty-eight; diabetes with atrophy of pancreas, atrophy of left and hypertrophy of right kidney. Stomach and small intestine normal. Large

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intestine normal size, contains brown fecal masses, mucous membrane striated with whitish, viscous masses adherent to the mucous membrane, proving on careful staining to be mucus. From a subsequent advice from Weigert I gather that the mucous membrane offered no macroscopic signs of inflammation. In the mucus were found cells that still in part showed the form of crypt epithelium. In comparison with the quantity of mucous substance surrounding them the cells are scant. The lumens of Lieberkühn's crypts are distended, the goblet-cells swollen and in part broken off. Lumen of the crypts densely filled with homogeneous masses. The contents of the crypts show a close relationship with the membrane covering the mucosa. We can observe in parts that the contents of the crypts are poured into the membrane. The submucosa and deeper layers of the intestinal wall normal.

Supplementary letter: In the interstices of the crypts the round-cells increased but little over the normal, and then particularly in such places at which production of mucus and formation of membrane is greatest.

Weigert considers these mild inflammatory manifestations as secondary. For comparison, Jagic gives a careful histologic description of two cases of true chronic catarrh of the large intestine : macroscopically, mucosa injected, swollen, succulent, thickly covered with mucus ; microscopically, in parts with lesser mucous formation, incomparably more cells, also leukocytes and desquamated epithelium ; interglandular tissue generally more plentiful. Unfortunately, I have been unable to secure further clinical details of the first case, but it is important from the fact that a pathologist of Weigert's ability failed to find both macroscopic and microscopic signs of inflammation.

[In a woman aged fifty years, who passed cylindric mucous casts of the intestine, sometimes $1\frac{1}{2}$ to 2 feet long, for about two years before her death from carcinoma of the sigmoid flexure, Dr. Pye Smith[1] found casts *in situ* below the stricture at the autopsy. When these were washed off, the mucous membrane was found to be unaffected.—Ed.]

Similar to the Jagic-Weigert case, yet in my estimation different, is the following case of M. Rothmann :

Female, admitted June 14, 1892, for carcinoma of base of skull ; died November 25. From day of admission suffered from obstinate constipation. On October 25, after an irrigation, patient for first time passed several meters white, slimy, restiform masses. No difficulty experienced therein. Subsequently, each time after irrigation similar masses were excreted on two or three occasions, always without feces and without pain. On section transverse colon, where not filled with feces, and in the markedly contracted parts of the descending colon in particular, the mucosa was injected, greatly plicate. Between the plications were whitish, partly membranous, partly restiform effusions from the free space that is maintained by the lumen of the contracted abdomen. Portions of large intestine covered with membrane were free of feces. In ascending colon, on the contrary, there was a great deal of feces, no membrane, mucosa reddened. In lower large intestine and rectum, where there was also membrane, this could be detached, without loss of substance, from the reddened mucosa. In the small intestine feces, mucosa slightly reddened. Microscopic examination carried out with aid of various staining methods gave mucin coloration everywhere, but no fibrin coloration, and showed that the mucous masses had entirely crowded out the epithelial layer of the mucous membrane. They penetrated the lumens of the glands and extended down to the fundi, and ramifications could be followed into the goblet-cells of the glands. Between the gland-tubules there were decided cell proliferation and widening of the mucosa.

Rothmann himself says in summing up : " There exists (besides the

[1] Pye Smith, *Trans. Path. Soc.*, vol. xlvii., p. 52.

increased secretion of mucus) a slightly inflammatory affection of the mucosa, which must, however, be regarded as secondary." Doubtless objections can be made to his conclusion. Especially it might be objected that the small intestine is also reddened—*i. e.*, affected with catarrh—although in it there is no membrane formation. And the supposition that the reddening in the small intestine is of a different nature than that in the large is entirely without foundation. It would have to be proved in the first that the catarrh was secondary. But for this case, the catarrh, as shown by excellent staining results, particularly when compared with Weigert's case, appears to be entirely too marked. In all probability it was idiopathic, and the view that it is primary is justified.

From the older literature we cite the following case from Abercrombie (Case 125 in his work):

Female, eighteen years, with incurable tympanites; "many hysterical affections"; constipation. Improvement followed sojourn in country, summer of 1826. In November obstinate constipation recurred; troubled with acidity. In December the evacuations began to contain a viscous mucus, and later large quantities of a white substance were passed. Fever at night, pains in region of ascending colon. In course of six or seven months practically bedridden. "At this time the most prominent symptom was the passage of extraordinary masses of a substance that at times appeared as a clear, transparent gelatin. Frequently it appeared as long, thready, fibrous masses; again there would appear large pieces of firm, uniform, tough membrane. Occasionally the membrane formed unmistakable tubes; sometimes it resembled hydatids; again it formed cuticular sacs that inclosed apparently normal feces. The tubes were frequently 4 to 5 inches long. At times there appeared in the evacuations quantities of a white matter resembling cream. These varied stools often ceased for days at a time, and in the interim were entirely normal. Recurrence was usually preceded by constipation, a sensation of heat in the intestine, a gnawing sensation in the stomach, thirst, and headache. Feces passed with the diseased evacuations appeared normal, but were hard and lumpy. Improvement end of April, 1828 (1827?), and patient got up. In July complete disappearance of pathologic stools. Evacuations remained perfectly normal, and only at times a mild aperient was required. Patient had begun coughing middle of June, and took to her bed in July; in August high fever set in; pains in left breast; early in September copious evacuation; September 9, death. On section, left-sided pyopneumothorax; complete tuberculous infiltration of left lung, with enlarged cavity; perforation. Right lung sound. Abdominal viscera, save mucous membrane of colon, normal. Colon over entire extent dotted with small spots of clear white color, sharply differentiated from the surrounding mucosa. A few were larger than a good-sized pin-head; careful examination developed the fact that they were slightly elevated vesicles, which, when opened, discharged a small quantity of a clear fluid. As a consequence of the great number of the vesicles the mucosa assumed an extraordinary appearance; other membranes normal. Mucosa of cecum plainly showed two points of suppuration. Small intestine normal."

Apparently Abercrombie describes in this case a past chronic cystic colitis. This manifestly had no bearing on the acute tuberculous infiltration of the left lung that took place in the last three months of life. But dispassionate observation must connect it with the earlier intestinal manifestations.

Hemmeter has recently reported that in two cases of enteritis membranacea under his observation he had found the indubitable histologic

picture of catarrhal inflammation. In one case it was found only in the colon ; in the other, in the colon and ileum.

Etiology.—Under the head of enteritis membranacea the literature gives various symptom-complexes, and in our opinion, indeed, different diseases. Thus we have a divers array of etiologic relations.

Most authors attribute the majority of cases to women (80 per cent., Litten ; 90 per cent., Kitagawa and Einhorn ; 75 per cent., Hemmeter and Langenhagen). [According to Bottentriet,[1] half the cases are in men and children and the other half in women, children being affected in 7 to 8 per cent. In von Noorden's 76 cases 48 were women, 24 men, and 4 children.—Ed.] Most cases occur in middle life. Cases have been observed in late life, in childhood (Boas, two-year-old nervous girl), and even in the new-born. [R. Giffard[2] says infantile muco-membranous enterocolitis occurs especially among the neuro-arthritic children of the rich.—Ed.]

Most cases occur in nervous, neurasthenic, hypochondriac, hysteric individuals. Writers are so well agreed upon this point that there is no need to mention any names. Still a few observers report enteritis membranacea in persons whom they distinctly class as not nervous. Enteroptosis and achylia gastrica (Einhorn) have been regarded as of pathogenetic importance. [Mathieu[3] regards it as a hypersecretion of mucus in patients of a neuro-arthritic type who suffer from enteroptosis. Glènard[4] considers it related to enteroptosis, inasmuch as hepatopsis leads to altered vascular tension in the liver, which, by diminishing the secretion from the mucous membrane of the intestine, favors the precipitation of mucin by acids in the intestine.—Ed.] This is apparently accounted for by the fact that both states are not infrequently encountered in the nervous and neurasthenic. A general nervousness may have been responsible for a case attributed to disease of the generative organs (Letcheff, Akerlund). Mathieu, Chevalier, and Langenhagen assume a connection with lithiasis intestinalis ; Langenhagen and Hemmeter, with arthritis. We do not believe that conclusive evidence of these has yet been adduced. Various observers have regarded constipation as significant for the mucus formation. As a matter of fact, constipation is not too infrequent. Geoffroy, indeed, goes so far as to declare that spasm in the musculature of the large intestine causes the increased secretion of mucus. Others, among them Vanni, Mannaberg, dispute the assumption that coprostasis is the direct cause of the hypersecretion of mucus. It has even been asserted that pathogenetic relations exist between appendicitis and enteritis membranacea, but this does not brook criticism at all. [Gilbert and Lereboullet[5] believe that it may be one of the many secondary symptoms which are prone to occur in simple family cholemia, a condition in which there is slight jaundice, usually without bile-pigment, in the urine.—Ed.]

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[1] Bottentriet, *Brit. Med. Jour.*, 1903, vol. i., p. 1488.
[2] René Giffard, *Thèse*, Paris, 1903.   [3] Mathieu, *La Semaine Méd.*, 1897, p. 226.
[4] Glènard, *Acad. de Méd.*, April 20, 1897.
[5] Gilbert and Lereboullet, *Gaz. Heb. de Méd. et de Chir.*, September 21, 1902.

The etiologic significance of the foregoing will be discussed further on under Pathogenesis. We shall only mention here artificial enteritis membranacea (Boas), which consists in the plentiful production of mucus by rectal irrigations of tannin, alum, and silver nitrate solutions.

Symptoms.—The lack of uniformity described under Etiology holds true here also. Only one point in common is met with in all descriptions by various writers, and that is the occurrence of large quantities of mucus in the dejecta. As the constitution and quantity of this mucus have served to distinguish this disease from ordinary intestinal catarrh, it will be proper to give first the description of the dejecta.

The description of the macroscopic appearance of the stools is the same in all the published accounts. The dejecta, according to all authors, consist exclusively or in great part of membranous shreds or tubular masses resembling the membranes of croup and the coagulate membranes found in the air-passages. The color of these membranous tubules is ordinarily gray, but they may be transparent, like mucus, or grayish white and not translucent, like fibrin; occasionally they are colored somewhat brown by the admixture of feces. In a few cases I have seen the tubes and shreds colored reddish by blood. The amount of membrane evacuated is frequently very large. In almost all cases the feces consist exclusively of this material, and it is only rarely that the evacuation of membranous tubules or shreds is preceded by the passage of fecal matter. The mucoid material appears either as ribbon-shaped shreds or membranous masses which are rolled into large lumps, and look like swollen potato-peelings. In other instances the material is passed in the form of tubular casts that vary greatly in size; they may be a meter long, or may form reticulated or arborescent structures.

[Hawkins describes three forms or stages in the disease; the mucus may be found: (1) In the form of cylinders or long thin bands which frequently resemble ascaris lumbricoides or segments of a tape-worm; these cylinders are passed after the feces and lie on the surface of the feces; (2) in the forms of tubes which may form a cast of the intestine and be of very considerable length: they are mixed with the feces and may be found in them; (3) the pellicular or hemorrhagic form; the mucus occurs in shreds or pellicles from 1 to 3 inches in length. Hemorrhage, which occurs in the previous forms, may be profuse.—Ed.]

If the dejecta are carefully suspended in water, the recently evacuated masses can, as a rule, be unfolded so as to form flat membranes. Occasionally very fantastic shapes are seen. Rosenheim, for instance, saw shreds of membrane that resembled a bladder. Other kinds of material are often passed with the feces and may lead to error and confusion with the membranes just described unless the material is carefully examined microscopically. Fascia and tendons are comparatively frequently passed in the stools, and occasionally a piece of an artery or a vein taken in the food or a membrane from an orange may be passed with the feces and lead to error. In one case peculiar ribbon-shaped masses that were evacuated consisted exclusively of oscillatoria; in

another case, of leptothrix threads and threads of thrush. I remember a case in which the diagnosis of membranous enteritis seemed probable until a microscopic and chemic examination of the material evacuated showed that it was curdled milk.

Microscopic examination of masses when recently passed shows that the material of which the membranous structures consist is in no way different from ordinary intestinal mucus. The microscopic examination must, of course, be made in perfectly fresh specimens and not in specimens that have been preserved in alcohol or in some other preserving fluid. Under the microscope a glassy, structureless matrix will be seen, which, on addition of acetic acid, usually shows some clouding and then striation. Columnar epithelium can nearly always be found in the membranes; occasionally these epithelial cells are present in almost incredible numbers. In many instances the structure of the cylindric cells is still preserved; in others, the cells are seen in various stages of degeneration; in the latter case they are shrunken and contain large vacuoles. Round-cells, on the other hand, are seen in astonishingly small numbers and are frequently entirely absent. Åkerlund observed " in a couple of evacuations a not inconsiderable number " of leukocytes, but never particularly numerous, and never grouped in the membranes. Triple phosphates, cholesterin plates, and other fecal constituents are found more often. He demonstrated regularly, but in insignificant numbers, numerous varieties of cocci and bacilli, but does not attribute any real significance to them. Krysinski calls particular attention to the fact that all the lower micro-organisms which are normally found in the feces seem to be greatly increased in this disease. This statement is not corroborated by other observers, including Brunner; personally, I was unable to find that the micro-organisms are present in larger numbers in the feces in this disease than in other conditions.

Several authors have attempted to gain an insight into the nature of the process we are discussing by chemic examination of the mucous masses that are evacuated. The earlier observers all determined the presence of mucin or of some substance resembling mucin (Clark, Thomson, Perroud, DaCosta, Hare—all quoted by Woodward); my own observations and those of the following authors corroborate this— Fürbringer, Krysinski, Walter, von Jaksch, Kitagawa, Rothmann, Litten, Hirsch, Vanni, Leube, Pariser, Åkerlund, Ewald, Schmidt, and others. [J. B. Leathes,[1] by chemic examination, found that the casts were not mucin, and when epithelial cells entangled in the casts were removed by washing, did not give proteid reactions. He was inclined to consider the material to be like chitin derived from carbohydrates. Eastes[2] thought that it was allied to keratin.—Ed.] Modern staining method of mucin and fibrin entirely corroborate chemic results. It must be accepted as absolute to-day that mucin is the chief constituent of the membranous masses. In addition there are other mucoid substances regarded as globulins or nucleo-albumins; the latter, however, are present in such small quantities that their significance

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[1] Leathes, *Lancet*, 1901, vol. ii. [2] Eastes, *ibid.*, 1901, vol. ii.

is greatly subordinate to that of mucin. Leube, it is true, reports that " in some cases " the membrane undoubtedly consists in great part of mucin, but that " in other cases " only traces of mucin can be found. In the latter instances he believes that the albuminoid substances take the place of mucin. Fibrin, however,—and this is important,—has been looked for in vain by many investigators ; some of them have found only traces of fibrin ; others, among them von Jaksch, claim to have found somewhat more abundant quantities, but always together with mucin. P. Guttmann is the only one who claims to have found the evacuated masses of membranous tissue to consist exclusively of fibrin. This statement, so far as my knowledge of the literature goes, is isolated, and its value is somewhat impaired by the fact that this author does not describe the chemic reactions that he employed to identify fibrin. Here and there we find the statement made by medical men that " fibrinous " masses were evacuated by their patients ; such statements, however, are of no significance in this discussion, for the reason that the various medical practitioners who report these cases relied simply on the coarse macroscopic appearance of the dejecta. Arnold and Steele have contributed a case which may be cited as an instance of the occurrence of true, pure fibrin in the dejecta : Nervous male, long-standing dyspepsia and constipation ; sudden pain in right of abdomen, with fever ; mass corresponding with ascending colon could be felt, painful on pressure ; injections produced copious evacuations, disappearance of tumor-like mass, and fever. During convalescence passage of egg-shaped masses somewhat resembling Swiss cheese, which Steele determined, beyond doubt, by Weigert's method, to be pure fibrin, without any other cellular elements in it. The origin of the fibrin remained in doubt. There was no reason to think it was blood-fibrin, equally none to believe it came from the ingesta.

Save for the uniform occurrence of these mucous masses, the clinical pictures of these cases, which, as we have repeatedly stated, are all set forth under the name enteritis membranacea, present such marked differences that we deem it best to describe them in groups.

One group is characterized by the occurrence of pain and passage of mucus ; this form occurs, as a rule, in nervous individuals who are at the same time troubled with difficulty in defecation.

Attacks occur more or less suddenly, or may develop gradually to full strength from a mild onset. At times some accidental occurrence is blamed for the attack—as, for example, a drink of cold water ; but most frequently it is some psychic disturbance. Again, the attacks cannot be traced to any extraneous cause. The pains, which are colicky or neuralgic, may be so exceedingly severe that the patients think they are about to die and writhe with groans and cries. Pains are most frequent in left of abdomen, at times in gastric region or over entire abdomen. Sometimes there is a history of abnormal sensations in the bladder and genital organs ; occasionally they are more wide-spread— *e. g.,* into the left leg. Geoffroy states that palpation in the left iliac fossa always reveals the sigmoid flexure tense, swollen, and sensitive to

pressure in consequence of contraction of the part. Transverse colon and cecum are less frequently affected to the same extent. In the same patient there may occur, between the attacks of greatest intensity, passages of mucus without the slightest unpleasant sensation. At times the pains are only moderately severe, or are described as merely unpleasant sensations—a feeling of discomfort, not actually painful. Passage of the mucous masses, generally without actual feces, occurs spontaneously, with great straining or with artificial aid. In exceptional cases the pains follow the passage of the mucus. As a rule, relief follows and the patients are free from pain for some time. The attacks of pain may occur one day, or daily for a week. Schütz has observed a case in which for four weeks the passage of mucus and the colicky pains recurred regularly. The free intervals may last a few weeks or many months. It may happen that the disease is ended with a single attack.

Generally the subjects are constipated, even in the free intervals. The constipation, as pointed out by Westphalen, presents frequently the spastic form. The descending colon and sigmoid flexure are felt as thin, contracted cords. The feces, passed spontaneously with great effort or artificially, are rounded, of hazel-nut size, or very thin, and the patients after defecation experience a sensation of insufficiency of evacuation. The majority of subjects are in varying degrees nervous, neurasthenic, hysteric, hypochondriacal, presenting the corresponding symptom-complex. In women enteroptosis is not infrequent. During the period of the attacks the nervous symptoms may be heightened to an appreciable degree. In the free intervals, in the most typical cases, bowel-action may return to its former status—*i. e.*, it is in the main torpid; the fecal masses may be entirely mucus-free, having only the characteristics of the stools of constipation, of mainly the spastic type. Boas and Schütz lay great stress upon the scarcity of mucus in the free intervals as demonstrating the neurotic origin (colica mucosa, myxoneurosis) of the disease. We believe they are correct in this.

A second group presents different manifestations. As a type of this group I cite the case of Abercrombie. Here again the mucus-production is the foremost symptom. But the paroxysms of pain are entirely wanting. At most there are only feeble unnatural sensations, or the pains are such as occur occasionally in all catarrhs. It may happen in such cases that constipation and diarrhea alternate, and the latter may even be the more frequent. But it is particularly important to note that, unlike the first group, in which the passage of mucus and the violent pains cease together, mucus is passed continuously, either with or without fecal material.

A third group presents other appearances. In this the manifestations of the first and second groups are combined. As illustration I cite the following case from the practice of Dr. Wilhelm Schlesinger— I have seen this subject numbers of times:

Eugenie P., age forty years, first seen in April, 1897, at which time, ostensibly as a result of some failing in digestion, she suffered for five days from violent

diarrhea with fever (up to 39° C.—102.2° F.). Only other objective symptom, tenderness on pressure over sigmoid flexure. Similar attacks said to have been frequent within preceding ten years. In one instance there arose febrile disease with violent pain in right iliac region.

The chronic intestinal symptoms are of equally long standing, beginning with irregularity in stools. Constipation of a day or two, alternating with diarrhea. Generally every morning two or three slight passages of mushy stools, preceded and succeeded by tearing pains in the left iliac region. Patient awakened almost nightly, at 1 or 2 o'clock, by unpleasant sensations in abdomen. Describes these as a feeling of gas-collection, pressing against left iliac region, reaching a point which cannot be passed and there occasioning penetrating pain. At times small quantities of gas escape. Irrigation sometimes brings on stool and passage of flatus and affords relief, but at other times this means is ineffectual.

Finally patient suffers at intervals of months with cramp-like pains, of several hours' duration, in umbilical region. These attacks also have recurred during some ten or twelve years, frequently at time of menstrual period, and are regularly followed by copious evacuations of clear mucous stools. These consist of long (5–10 cm.), band-like, glassy masses that float in water. They show, microscopically, mucus, a few pus-corpuscles, and epithelial cells. In isolated instances this "colic" was followed by a passage of blood to as much as ⅛ liter.

Feces examined in free intervals contained mucus in moderate quantity only, and thoroughly mixed with the feces. Otherwise the only objective symptom is the sigmoid flexure, which is apparently contracted, sensitive, seeming about as large as one's little finger. Genitalia normal.

On strength of the diagnosis—adhesions and appendicitis—laparotomy was performed in the fall of 1899 (Gersuny). As a matter of fact, the vermiform appendix was found displaced downward and outward, and surrounded with adhesions. The appendix was removed, thread-like adhesions to the sigmoid flexure released, and the mesosigmoid shortened. A broad, reticular adhesion, practically inclosing the sigmoid flexure, was fast in the lateral layer of the mesosigmoid. It was apparently of recent occurrence and connected with a not totally undisturbed course of healing. It was removed at a later operation.

After this the patient was almost perfectly well for over half a year. Only after eating foods that readily produce flatulence were there any symptoms similar to her former trouble. During the summer of 1900 she complained of no disturbances of any kind.

It was the autumn of 1900 when the old complaints reappeared: difficulty in evacuation, the nightly irritation with pains in sigmoid flexure, and colicky attacks with mucous discharges as above described. The attacks occurred on an average of once a month. Various therapeutic measures, such as massage, diet, profuse water and oil injections, were ineffectual. Only electric massage gave slight transient relief.

Acting on the hypothesis that adhesions had recurred, the attempt was made, in the autumn of 1901, to inspect them *per rectum*. Examination by the orthoscope (Hochenegg) revealed), about 15 cm. above the sphincter ani, a large free fold of mucous membrane extending downward and to the left into the lumen of the rectum. Above this the mucosa is reddened and shows bleeding areas. Below, the mucosa is pale. At the sphincter ani a varix about the size of a pea.

The attempt to widen the relatively narrowed point by the passage of soft thick bougies has apparently succeeded after a six weeks' treatment. Patient has been well for two months, even the colicky attacks and mucous discharges being absent.

The patient has always been psychically excitable. However, there are no particular nervous symptoms demonstrable except exaggerated knee-jerk.

There is hardly room to doubt that in this case there was mucous colic in addition to chronic intestinal catarrh. The frequent recurrence of enterorrhagia is of interest, the source of which was determined on direct rectal examination. Mathieu regards the enterorrhagia as an accidental complication of the colitis mucomembranosa. Cases such as the foregoing show that intestinal hemorrhage need not stand in patho-

genic relation to the colitis mucomembranosa, but that we generally have to deal with some additional lesion of the diseased intestinal mucosa.

Pathogenesis.—For seventy years past (Roche, 1833) various observers have all used the term enteritis seu colitis, with different qualifying adjectives, and have expressly stated the anatomic basis of the disease to be an inflammatory process, even though perhaps of a special kind (Siredey, DaCosta). On the other hand, I was the first to express the belief, already stated, that in a great number of cases included under this class there is really no inflammatory process at all, and have, therefore, described them as a *colica mucosa* or *mucous colic.* Later, in the first edition of this work, I emphasized the fact that for another, though smaller, number of cases, termed in the literature enteritis membranacea, the inflammatory character must be accepted with caution. I am compelled to maintain this position even to-day, notwithstanding the various publications of recent years, the results detailed in which have just been briefly given.

In our opinion the essential point for the interpretation and judgment of any pathologic condition must be sought, first of all, in the anatomic lesion. We cannot abate from this requirement in the slightest degree. Anatomic biopsies and necropsies, despite their small number, prove that the clinical picture of the so-called enteritis membranacea is not an etiologic entity—that the various conditions grouped under the name cannot be considered from any common standpoint. The condition may exist without any structural evidence of enteritis; in that case it is really not an inflammatory process, but one that may be termed simply colica mucosa, or, as Ewald aptly suggests, *myxoneurosis.* On the other hand, we do find inflammatory changes in some cases, and these, of course, must still be called enteritis membranacea. It is my opinion that any endeavor to force all the varying and different conditions into one general category would show little regard for all the facts.

In the healthy intestine these is a constant excretion of mucus into the lumen of the bowel through the crypts of Lieberkühn, but the small quantity makes it unobservable, and it is lost to view on its way until the excretion of the feces. Some decades ago I observed that every appreciable admixture of mucus in stools indicated conditions that were on the boundary between the physiologic and the pathologic. As examples, take the traces of mucus found on large, but otherwise normal, fecal masses, whose mechanical irritation occasioned a slight increase in mucus-production.

Even the steady formation of mucus in the healthy intestine, though slight in the absolute, can lead to a considerable accumulation of the secretion in case it is not removed by normal regular evacuations of the bowels. This is best seen in the spastic constipation in which the intestine is contracted, a form in which small quantities of feces of slight bulk accumulate in the lumen of the lower large intestine. It is in such cases, as first described by Marchand, that coagula and strands can form : " A viscous intestinal mucus covers the surface ; in the

depths of the longitudinal folds this forms into round cords, which reticulate or present a branched appearance. The rolling together and cohesion, in the longitudinal axis, of smaller cords form larger ones." Hence such appearances seen postmortem, if within reasonable limits, may still be physiologic, and occur in a truely normal mucosa.

That such a production of mucus may occur without any of the anatomic signs we call inflammatory is shown by observations such as those of Jagic-Weigert.

The periodic voluminous secretion of mucus encountered in colica mucosa must be explained in the same way as in the preceding cases, allowance being made for pathologic quantitative differences. The clinical manifestations, the almost constant nervousness of patients in these cases, make conjecture easy as to the nature of the irritant causing such great hypersecretion of mucus. In all probability, as Vanni and Leube have clearly stated, the cause is to be sought in the direct effect of pathologic nervous excitations. Direct experiments aiming to prove the dependence of mucus-production in the intestine upon innervation are not yet available. Nevertheless, the nervous theory of the pathogeny of colica mucosa is perfectly justified by numerous well-known analogies in other nervous hypersecretions. (An instructive contribution to this question is the observation of Strauss and Grote: Youth, sixteen, mitral insufficiency, sudden paralysis and anesthesia of both legs, acute bedsore, retention of urine, etc. Diagnosis: Spinal embolism. For several days there was a profuse flow (over $\frac{1}{2}$ liter a day) of a clear, thin, fluid mucus from the anus which was insufficient).

In fact, it appears to me that the group of symptoms first given can be explained only by neuroses of secretion.

The constipation, mainly spastic, so frequently seen in this affection, and occurring only as a concomitant symptom of the general nervous condition, cannot be proved to be causative of the abnormal production of mucus. As correctly remarked, by Vanni and Mannaberg in particular, if this were so, then the feature of hypersecretion should occur much more frequently. As a matter of fact, it is but rarely observed in the atonic form of constipation. Constipation influences or determines only the outward appearance of the mucus, the formation of tubes, cylinders, etc., but in no way causes the hypersecretion of it.

Besides this group, in which the abnormal mucus-formation is a neurosis of secretion, without inflammatory changes in the intestine, there is a second group, in which there are anatomic-catarrhal changes. In all intestinal catarrhs mucus is formed in pathologic quantity, yet all are not distinguished as enteritis membranacea. To justify the application of this term, the mucus-production must be excessively large, and, in addition, it must be excreted in peculiar forms. But what causes the hypersecretion in such cases? Here, too, a careful analysis of individual cases appears to prove that there is again a hypersecretion of nervous origin. The histories show that the majority of these patients also are nervous. I believe we can summarize these cases as follows: The patient is attacked by a true anatomic intestinal catarrh. Sometimes, we are

told, the attack was of acute febrile onset. But as the patient is a nervous individual, the catarrh in his case may serve to release a hypersecretion of mucus that may even be of some duration. Thus the disease has a two-fold aspect—that of true catarrh and that of nervous hypersecretion.

Different, again, is the third group, of which an instance has already been given. Here, too, there is a true enteric catarrh, with the customary clinical picture. In addition there arises, as a complication, a hypersecretion of mucus, not so continuously, but only in attacks of typical colica mucosa.

The difference between the second and third groups can be formulated as follows : In both there is catarrh, but in group two its aspect is lastingly changed by the continuous mucus secretion. In group three the picture of true catarrh is present, as a rule, being changed at intervals by intercurrent attacks of mucous colic.

I have explained the overpowering pains of mucous colic in the same way as that in colica flatulenta. In the latter case the gas, in the former the excessive mucus, collection causes reflex tetanic contraction of the intestine and thereby the attack of colic. The fact, ascertained in recent years, that in exceptional cases the pain may occur after evacuation of the mucous masses, makes my explanation, at least as a general one, untenable. Rosenheim has suggested that the pain is a hyperesthesia appearing concurrently with the neurosis of secretion. I must acknowledge this theory of Rosenheim's as fitting in certain cases, but in other cases I adhere decidedly to my earlier one. One phenomenon, observed quite frequently, supports my theory. It is that during attacks a strong tonic contraction of the colon can be felt.

It is well to mention also the views, particularly of French authors, on relations between enterocolitis mucomembranacea and intestinal sand. Dieulafoy and Mathieu discuss the not too infrequent occurrence of intestinal sand. It consists usually of calcium salts, particularly calcium phosphate and calcium carbonate ; also magnesium and ammonium phosphate and traces of other substances. The authors above named and also de Langenhagen consider the formation of the latter a consequence of the former. [De Langenhagen,[1] who regards mucous colitis as evidence of the neuro-arthritic diathesis, has met with intestinal sand in 112 out of 1200 cases of mucous colitis, or 9 per cent. ; he believes that it is a complication of the intestinal condition and not an independent arthritic diathesis.—ED.] Eichhorst had two cases (females who, judging from the symptoms given, suffered from enteritis membranacea). In the intestinal coagula he found sand, consisting, in the more carefully examined instance, of calcium carbonate. He remarks that the paroxysms of pain were unusually severe when the stools contained, besides cuticular masses, also quantities of sand. Further than this Eichhorst does not express himself as to the relation of intestinal sand to enteritis membranacea. Hemmeter again believes that uric acid, and particularly its transformation-products, in the uratic

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[1] Maurice de Langenhagen, *Muco-membranous Enterocolitis*, Churchill, 1903.

diathesis and gout may cause enteritis membranacea—the products passing through the intestinal wall and setting up abnormal secretion of mucus, as is done by other irritants. He found larger quantities of uric acid in the stools of two patients during attacks than in free intervals or in healthy individuals. These questions have not yet been definitely decided. (Intestinal sand is considered on p. 94.)

Treatment.—Enteritis membranacea gives a varied prognosis, according to the form. In the catarrhal form, with nervous manifestations, the catarrh is, above all, the first consideration, as explained under Intestinal Catarrh. The nervous forms, especially true colica mucosa, permit a favorable prognosis as to life, but doubtful as to cure, as these forms are apt to be exceedingly resistant to all therapeutic measures.

Treatment at present is being shaped more and more by the theory of the neurotic etiology of the condition. It must be admitted that those measures aimed directly at a hypothetic catarrh show very meager results, excepting, of course, in cases where catarrh really exists. We must separate the therapy of individual cases from that of the condition in general.

The first consideration in an attack is the production of evacuation of the mucous masses. This is best and most rapidly effected by copious irrigation of the intestine, preferably with lukewarm 0.5 per cent. salt solution. A single attack is over with the evacuation. If the 0.5 per cent. salt solution has not the desired effect, stronger purgatives may be used, as infusum sennæ or oleum ricini, even injections of pure oil (Fleiner). Should these means also fail, we must resort to purgatives by the mouth, as one of the bitter waters, tamarinds, rhamnus cathartica. In very severe pains poultices to abdomen or baths may be employed, and, lastly, morphin or belladonna may be necessary.

In any consideration for treatment of the underlying condition there rank first the neurasthenia and then the constipation. The former demand all the available psychic and physical measures, as well as every other means at hand (baths, hydropathy, exhibition of iron, etc.) that the individual case indicates. Besides this, careful provision for regular stools must be made, preferably by regulating the diet and with mechanical-physical methods, as described under Habitual Constipation. These methods are aided by irrigations of the intestines, which must in some cases be carried out regularly. These methods only effect evacuation of the intestines, and not the solution of the mucus, and have no effect on any inflammatory processes or bacteria present. In fact, experience with various antibacterial substances and alkaline and astringent solutions has given no appreciable results. Mineral waters are in most cases not of any great value except as purgatives. It is only in cases of true enteric catarrh that one may expect results from the use of salines, as Kissingen, Carlsbad, etc. In other cases—the purely nervous mucous colic—it will require some caution before Carlsbad is prescribed. [Homburg and Plombière are spas to which patients are frequently sent.—Ed.]

Von Noorden has recently advocated a coarse diet leaving large

residue, that is most successful when suddenly inaugurated. The food consists of coarse bread, pulse, fruits with small kernels and thick skins, and vegetables rich in starch. [In 76 cases of typical mucous colic, 71 of whom had chronic constipation, treatment directed to cure the constipation and improve the condition of the nervous system was successful in 79 per cent. In 50 per cent. there was permanent cure; relapses occurred in 13 per cent.; incomplete success, in 15.8 per cent.; in 15 per cent. the result was not known; in 5.2 per cent. this treatment definitely failed (von Noorden [1]).—Ed.] In all likelihood it is advisable to individualize and specialize the treatment of each case.

[Hale White and Golding Bird [2] have employed surgical treatment for very obstinate cases of chronic colitis with the passage of casts from the bowel. The object of the treatment is to give complete and prolonged rest to the colon, and with this in view right colotomy was performed, and for periods varying from a few months to two or three years the artificial anus was kept open and relief obtained. Subsequently the artificial anus was closed. This treatment is designed by the authors for intractable cases of "membranous colitis" and would hardly be justifiable in the less severe forms associated with neurasthenia.—Ed.]

ULCERATION OF THE INTESTINE (Ulcera Intestinalia).

THE whole intestine, from the duodenum down to the lowest portions of the rectum, may be the seat of ulceration. The histologic forms of the intestinal ulceration vary. The etiologic factors favoring the development of ulcer vary still more, and may be very manifold in character. Following common usage we exclude from the category of "ulcers" those forms of destruction of intestinal tissue that are the direct result of strangulation, of invagination, of neoplasms, of rupture of abscesses into the intestine from without, of erosion by caustic poisons that enter the intestine from the stomach, of the irritating effect of certain agents on the mucous lining of the rectum, etc. But even leaving out all these different possible causes of destruction of intestinal tissue, there is no other internal organ in which so many different processes may lead to ulcerative destruction as the intestine.

From an etiologic point of view ulcers of the intestine can readily be divided into a number of groups as follows:

First Group.—Ulceration as the result of necrotic processes:
    Simple duodenal ulcer.
    Ulcer following cutaneous burns.
    Embolic and thrombotic ulcer (the peculiar ulcers of the intestine seen in patients with multiple neuritis belong to this class).
    Amyloid ulcers.

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[1] C. von Noorden, *Colica Mucosa*, 1903, p. 60 (American translation).
[2] Hale White and Golding Bird, *Trans. Clin. Soc.*, vol. xxix., p. 45; vol. xxxv., p. 183; vol. xxxv., p. 164.

Second Group.—Ulceration as the result of inflammatory processes :
  Catarrhal ulcer.
  Follicular ulcer.
  Stercoral or decubital ulcer.
Third Group.—Ulceration as the result of acute infectious diseases :
  Typhoid.
  Dysentery.
  Diphtheria.
  Anthrax.
  Sepsis.
  Erysipelas.
  Varioloid.
Fourth Group.—Ulceration as the result of chronic infectious diseases :
  Tuberculosis.
  Syphilis.
  Lepra.
  Pellagra.
Fifth Group.—Ulceration as the result of constitutional diseases :
  Gout.
  Scurvy.
  Leukemia.
Sixth Group.—Toxic forms of ulceration :
  Uremic ulcer.
  Mercurial ulcer.

If an attempt were made to arrange the different forms of ulcer of the intestine according to their pathogenesis, so far as it is known, it would be necessary to group them in a somewhat different manner, but as this dissertation is written chiefly from a clinical point of view, it may suffice to consider the pathogenesis when describing different kinds of ulcer. Certain forms of ulceration of the intestine will be discussed under the heading of the different morbid processes causing them. In this section we will limit ourselves to describing in detail only those forms which have a more or less independent existence.

FIRST GROUP.—SIMPLE DUODENAL ULCER.

An excellent monograph by A. Krauss contains the whole literature of this form of ulcer up to the year 1865, a dissertation by Chvostek, the literature up to 1882, and a pamphlet by Oppenheimer, the recent literature up to 1891. The reader should refer to these monographs for bibliographic references.

[Reference may also be made to Perry and Shaw's[1] paper (1893); Cullen's[2] digest of 184 cases (1897); Fenwick's[3] work and that of Hemmeter.[4]—ED.]

Ulcus duodenale simplex s. rotundum s. perforans occupies a

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[1] Perry and Shaw, *Guy's Hosp. Repts.*, 1893.
[2] Cullen, *Scot. Med. and Surg. Jour.*, 1897, vol. i., p. 635.
[3] Samuel and Soltau Fenwick, *Ulcer of the Stomach and Duodenum*, 1900.
[4] Hemmeter, *Diseases of the Intestines*, 1901, vol. i.

peculiar position among the ulcers of the intestine. It is included—and correctly so—under the category of peptic ulcers (Leube). Its etiologic factors are exactly the same as those of the round ulcer of the stomach, and everything that can be said in regard to the pathogenesis of the latter applies to the pathogenesis of round ulcer of the duodenum. In order to avoid repetition, therefore, the reader should refer for the pathogenesis of this lesion to the section on Perforating Ulcer of the Stomach in the volume on Diseases of the Stomach in this series.

The morbid anatomy of this lesion and that of simple gastric ulcer are exactly alike. Simple ulcer of the duodenum and simple ulcer of the stomach are identical regarding their appearances both structurally and histologically (circular outline, terraced walls, complete absence of pus-formation), their method of growth, sequelæ (hemorrhage, perforation, formation of adhesions with neighboring organs), and finally as regards the normal appearance of the mucous membrane in the immediate neighborhood of the lesion. I shall, therefore, refrain from describing all these points in detail, and shall limit myself to emphasizing a few points that are peculiar to simple ulcer of the duodenum.

Ulcer of the duodenum is less frequently met with than gastric ulcer. The relative frequency varies according to different authors : Trier, for instance, states that the proportion is as 1 is to 9, whereas Andral figures it at as 1 is to 40. Occasionally an ulcer of the duodenum and an ulcer of the stomach are found together. Ulcer of the duodenum is most frequently met with between the twentieth and the sixtieth year, and occurs with approximately the same frequency during the different decades of this period.

[In 127 cases dying from duodenal ulcer the average age was thirty-eight years. The Fenwicks, however, draw a distinction between acute duodenal ulcers, 68 per cent. of which prove fatal between fifteen and thirty years of age, and chronic duodenal ulcers, 63 per cent. of which prove fatal between thirty and fifty.—Ed.]

It is remarkable that very small children, who rarely suffer from gastric ulcer, occasionally develop ulcer of the duodenum (Spiegelberg found a duodenal ulcer in a new-born infant only four days and thirty hours old). [T. D. Lister [1] recorded a duodenal ulcer in a child three days old, probably due to embolism from umbilical thrombi, as suggested by Landau in 1874.—Ed.] Another remarkable fact on which all observers are agreed is that the proportion of males affected is very much greater than in ulcer of the stomach. (According to Weir's statistics, there were 30 women in 176 cases ; in Collin's statistics, 52 women in 257 cases.) [Cullen put the ratio as 5.6 to 1 ; the Fenwicks at 4 to 1 ; Perry and Shaw, 3 to 1, or, if burns are excluded, 6 to 1.—Ed.] It is also worthy of notice that chlorosis plays no rôle in the etiology of this disease. Boas seeks explanation for the preponderance of ulcer in men in acid gastritis due to the greater incidence of alcoholism in that sex. The alcohol-containing or acid contents of the stomach pass into the duodenum, where its action on the duodenal mucosa paves

[1] T. D. Lister, *Trans. Path. Soc.*, 1899, vol. l., p. 111.

the way for the causation, by some accidental factor, of a partial necrosis —in other words, an ulcer.

The various causative factors, such as burns and freezing, renal disease, septicemia, and erysipelas, will be dealt with separately ; here we have to deal only with a genuine peptic ulcer analogous to the round ulcer of the stomach.

Duodenal ulcer, as implied by the name, is situated in the duodenum. Ulcers that are pathogenetically closely related to this lesion and resemble it anatomically may also occasionally be found in other portions of the intestine. E. Wagner, for instance, found such an ulcer in the jejunum. [As the result of gastro-enterostomy and the entrance of hyperacid gastric juice into the jejunum, peptic ulcers may be produced ; Watts [1] has collected 14 recorded cases of this kind in man, and has produced them artificially in dogs.—ED.] The ulcer is usually situated in the upper horizontal portion of the duodenum, less frequently in the descending portion, and only exceptionally in the lower horizontal part of the duodenum. If present in the first-named part of the duodenum, the ulcer is usually situated in close proximity to the pyloric fold (this fold usually divides the upper horizontal portion of the duodenum into a duodenal and a gastric half). The ulcer does not seem to show a predilection for any one surface of the duodenum. Statistics in regard to the distribution of the ulcer on the different portions of the wall, anteriorly, posteriorly, etc., vary greatly ; those ulcers of the duodenum situated near the diverticulum of Vater are of special clinical importance, inasmuch as they may lead to the formation of cicatrices and thus occlude the orifices of the bile and pancreatic ducts.

Usually a duodenal ulcer is single ; in exceptional cases two or four may be seen. It has been thought that two ulcers of the duodenum when situated directly opposite each other might originate in the same way as two soft chancres on opposed surfaces of the prepuce and the penis, but this view is, of course, incorrect, as can be readily shown from a study of the pathogenesis of such lesions. In cases in which several ulcers are found, different stages of development may be seen—one may be perfectly cicatrized, one may be in process of cicatrization, or another one may be recent. Chvostek remarks that the statement made by many authors, among them Krauss, that cicatrized duodenal ulcers are relatively rare, is not correct, and that the comparative rarity of this condition is only apparent and due to the fact that cases in which cicatrices are found by chance during autopsy, but in which symptoms were absent during life, are not published.

The formation of cicatrices may lead to a variety of disturbances. Stenoses of the duodenum or stenoses of the duodenal portion of the pylorus are quite frequently met with ; these lead to the same consequences as cicatricial stenosis of the gastric portion of the pylorus. Other sequelæ are occlusion of the common bile-duct with the consequences that result herefrom (icterus, etc.), and occlusion of the duct of Wirsung with atrophy of the pancreas from stasis. French, in one

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[1] S. H. Watts, *Bull. Johns Hopkins Hosp.*, July, 1903, vol. xiv., p. 191.

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case, saw thrombosis of the portal vein from compression of this vessel as a direct result of deep cicatrization of a duodenal ulcer.

Occasionally an ulcer of the duodenum, if it develops rapidly and acutely, may rupture into the peritoneal cavity. If this occurs, death follows from shock or from diffuse perforative peritonitis.

If the ulcer grows slowly, circumscribed peritonitis often results and may lead to adhesions with neighboring organs. The organs involved in this process will, of course, depend on the position of the ulcer in the duodenum. If the adhesions are sufficiently dense and if the ulcer penetrates to the serosa of the duodenum, encysted purulent peritonitis may develop; in other cases in which the adhesions are not so dense, diffuse peritonitis may also result. The process of ulceration may involve other organs, either by extending through these peritoneal adhesions, or, if the ulcer is situated in portions of the duodenum uncovered by peritoneum, by direct continuity of tissue; thus, the liver, the gall-bladder, the pancreas (relatively frequent), the abdominal aorta, the hepatic artery, and the portal vein may all be involved in the ulcerative process. Secondary suppuration following an ulcer of the duodenum has often been known to penetrate through connective tissue and muscle until it perforated posteriorly through the back near the spinal column, or anteriorly through the abdomen in the region of the gall-bladder.

It is important to remember that blood-vessels are quite frequently eroded by an ulcer of the duodenum. Nearly all the arteries that are situated in the upper and the descending portions of the duodenum have been found eroded.

Finally, ulcer of the duodenum, like ulcer of the stomach, has been known to develop into carcinoma (Eichhorst, Ewald).

[Letulle [1] found an extensive ulcer in the first part of the duodenum with colloid carcinoma developing near its center in a man aged fifty-five, who had been under treatment two years previously with melena and other abdominal symptoms. According to Samuel and Soltau Fenwick (1902), [2] at least ten instances of this sequence of events have been reported.—ED.]

Clinical Aspect.—The appearance of simple duodenal tumors is variable, and in the main there is but little characteristic in them. Diagnosis is frequently impossible, or can be made only with more or less probability.

It has already been mentioned that pathogenetically and anatomically ulcer of the duodenum corresponds completely with ulcer of the stomach; the same in a measure applies to the clinical picture presented by the two diseases, for each resembles the other to a great extent.

An ulcer of the duodenum may frequently be present without causing any symptoms—in other words, be latent. Sometimes in performing an autopsy on an individual who has never shown any symptoms or indications of duodenal ulcer during life the cicatrix of a healed

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[1] Letulle, *Bull. Soc. Anat.*, Paris, 1897, p. 721.
[2] Samuel and Soltau Fenwick, *Cancer and Other Tumors of the Stomach*, 1902, p. 286.

duodenal ulcer may be accidentally found. Of course, it is impossible in cases of this kind to state positively that the individual did not at some time suffer from some abnormal sensations that might have been attributed to duodenal ulcer. As no record of such sensations is found in the history of these cases, it must be assumed that they were very slight and remained unnoticed or were forgotten by the patient; at all events, we may be certain that nothing characteristic of ulcer of the duodenum was present during life.

The same applies to another series of cases that run their course without producing any symptoms, or at least without apparently causing any sensations that can be attributed to ulcer of the duodenum, until suddenly perforation of the intestine occurs, peritonitis develops, and the life of the patient is terminated in this way ; or, in other instances, a blood-vessel suddenly becomes eroded, violent hemorrhage occurs, and the presence of a lesion of the intestine (ulcer), so severe as to endanger life, is in this way discovered for the first time.

In yet another series of cases definite symptoms exist for a longer or a shorter time. The following are the most important of these symptoms :

Pain.—As just stated, pain may be completely absent or may be so slight that the patients pay no attention to it. Budd has attempted to explain the frequent absence of pain in ulcer of the duodenum as compared with its great frequency in ulcer of the stomach, by assuming that the duodenum is less motile than the stomach and that, consequently, less traction is exercised on the ulcer than in the gastric form. He also believed that the food irritates a duodenal ulcer less than a gastric ulcer, chiefly for the reasons that it is partially digested when it reaches the duodenum, and especially because it is heated to body-temperature. Occasionally patients complain of very indefinite sensations in the epigastric region, a feeling of tension and pressure that may either occur spontaneously or be experienced very soon after eating, or only on external pressure. In other cases the patients complain of paroxysmal attacks of violent pain indistinguishable in any way from genuine attacks of cardialgia, and sometimes resembling paroxysms of intestinal or gall-stone colic. In the intervals between the paroxysms the patients are either quite free from pain or merely complain of vague discomfort. In some instances the patients suffer from severe continuous pain that is made worse by manual pressure, by the pressure of the clothing, by certain movements of the body, and by eating. These pains are very probably due to peritonitis. Occasionally peculiar sensations are complained of that cannot properly be called pain—for instance, a feeling of hunger, a boring, gnawing sensation as though something were lodged in the gastric region or in the right upper quadrant of the abdomen. Krauss remarks that these sensations are very frequently complained of shortly before hemorrhage or perforation occurs. Nothing is recorded in the literature on duodenal ulcers in regard to the occurrence of radiating intercostal neuralgia ; in gastric ulcer the existence of this form of neuralgia is recognized, but nothing analogous

seems ever to have been observed in duodenal ulcer.   Possibly clinicians
have never looked for this symptom.

[In his papers on the pain of visceral disease Head [1] points out that
the areas affected in gastric disorders correspond to the supply of the
seventh, eighth, and ninth dorsal segments, and that in affections of the
intestine from the pylorus to the end of the colon there is cutaneous
tenderness corresponding more or less to the ninth, tenth, eleventh, and
possibly twelfth dorsal areas.—ED.]

It will be seen that the pain of a duodenal ulcer is in every respect
similar to that of a gastric ulcer, and yet many clinicians argue that the
differential diagnosis between the two can be based on differences in the
character of the pain.   They claim that in ulcer of the duodenum the
pain is located more in the right hypochondriac region; but the pain
may also be felt in this area in ulcer of the stomach, and conversely
the pain and all the abnormal sensations in ulcer of the duodenum may
be chiefly situated in the mesogastrium.   Another difference has been
artificially created and is purely theoretic—namely, that the pain pro-
duced by ulcer of the duodenum occurs later after the ingestion of food
than the pain of ulcer of the stomach.   Many authors claim that this
duodenal pain does not occur until four to six hours after eating.   As
a matter of fact, however, the pain is frequently experienced two to
three hours, or even half an hour, after the ingestion of food.   It is
also stated that in ulcer of the stomach the pain is mitigated by vomit-
ing, whereas the pain of duodenal ulcer is not relieved hereby (John-
ston).   I consider this differential sign to be very uncertain.   Both this
sign and the estimation of the time of pain are altogether unreliable.
Chvostek employs the following sign in making the diagnosis of duodenal
ulcer.   In a case that was correctly diagnosed during life he discovered
that the pain that occurred two hours and a half after breakfast stopped
if a little wine was given, and that the pain occurring about three hours
after the noon-day meal also disappeared, at least for a time, if some
of this beverage was administered.   He explains this phenomenon in
this way, that the wine acted as an irritant to the stomach and reflexly
caused closure of the pylorus, and so prevented the passage of stomach-
contents into the duodenum, and that in this way the pain was stopped
temporarily after a large meal and permanently after a small meal.
Chvostek himself remarks that this behavior of the cardiac pain appears
to occur in duodenal tumors only in exceptional cases.   Boas observed
the same phenomenon, but does not state whether the reason is to be
sought in the reflex closure of the pylorus or in the dilution, by the
ingested fluid, of the excess of hydrochloric acid.

The appetite and the stools may be perfectly normal.   Dyspeptic
symptoms are rarely seen.   In exceptional cases the stools are diarrheic,
but more frequently constipated.

Vomiting is rare.   When it does occur, it usually terminates the
attack of cardialgia; or it is caused by some complicating disease of
the stomach.   In cases of healed ulcer it may be due to stenosis of the

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[1] Head, *Brain*, vol. xvi.

duodenum. The vomit, unless it contains blood, presents no character-istic features. But little has been adduced to date concerning the action of hydrochloric acid in the gastric juice in cases of duodenal ulcer. Leube and Reckmann and Robin found it absent or diminished. Devic and Roux, Boas, and Georgiewski, on the contrary, found hyper-chlorhydria.

[Hemmeter found normal acidity in two cases and excessive acidity or hyperchlorhydria in one case; in three cases A. Robin found an absence of free hydrochloric acid, but a considerable amount of organic acids.—ED.]

Hemorrhage is quite a frequent symptom. Apart from small insig-nificant hemorrhages that remain undiscovered, severe hemorrhages occur in about one-third of the cases. The blood is evacuated by vomiting alone, is exclusively passed in the stools, or leaves the intes-tine in both directions. In some instances death may occur before any of the blood is evacuated. The material vomited after a hemor-rhage or the feces show all the various possible changes that are seen in any case of gastric or intestinal hemorrhage. When large quantities of blood are passed, it may be more or less unchanged in the vomit or the feces, or its appearance may be greatly altered by the action of the gastric juice, or finally the blood may have the tar-like consistence so frequently seen when blood is contained in the feces. It is impossible to decide, from the character of the hemorrhage, whether in any given case the blood comes from a gastric or a duodenal ulcer.

[The Fenwicks estimate that hemorrhages, either hematemesis or melena, occur in 26 per cent. of the acute and in 40 per cent. of chronic ulcers, and that melena is not necessarily, as is generally supposed, more common than hematemesis. In both the acute and chronic cases the death-rate from hemorrhage was 36 per cent.; Cullen found that fatal hemorrhage occurred in 20 to 30 per cent., and Perry and Shaw, in 13 per cent.—ED.]

These are all the symptoms that can be produced by ulcer of the duodenum *per se,* for there is nothing characteristic about the numerous secondary symptoms that may be caused by cicatrization or other ter-minations of the ulcer (icterus, atrophy of the pancreas, suppuration, diffuse peritonitis). It will be seen, therefore, that the recognition of an ulcer of the duodenum is exceedingly difficult, and that in the majority of cases the lesion must remain unrecognized.

The only secondary symptom requiring a brief reference is icterus, chiefly because an exaggerated diagnostic importance has been attributed to this sign. Icterus is exceedingly rare in ulcer of the duodenum, so that from its very rarity it may be concluded that it has nothing to do with the ulcerative process itself. In the few cases in which it occurred in the course of duodenal ulcer other complications existed, or icterus was caused by cicatrization following healing of an ulcer, so that it can hardly be considered a true symptom of duodenal ulcer in either case.

Course.—Apart from that form of ulcer of the duodenum that fol-lows cutaneous burns (compare the following section), simple ulcer

of the duodenum almost always runs a chronic course. The process may extend over many years. Recurrences are quite as frequent in ulcer of the duodenum as in ulcer of the stomach. Occasionally different stages of the process may be seen side by side at the autopsy. The description of the clinical picture and of the pathologic anatomy readily explains why the course and the termination of this lesion vary so much. I will merely insist again that death occurs with exceptional frequency from acute and unexpected perforation into the peritoneal cavity and from erosion of blood-vessels and hemorrhage.

The prognosis, provided the diagnosis can be made at all, is always very serious.

The treatment is essentially the same as that advised for simple ulcer of the stomach, so that the reader should refer to the section on the treatment of the latter lesion.

[In perforation of a duodenal ulcer the fluid passes down into the right iliac fossa and the symptoms of acute appendicitis are often produced ; in fact, in 19 out of 51 cases of perforating duodenal ulcer the appendix was cut down upon after a diagnosis of acute appendicitis had been made (Moynihan).[1]—Ed.]

ULCERATION OF THE INTESTINE IN CUTANEOUS BURNS.

That hyperemia and inflammatory changes in the intestine may follow extensive cutaneous burns was known at the beginning of this (last) century. Macfarlane called attention to this etiologic factor in his discussion on duodenal ulceration. Curling, in 1842, however, was the first to attract universal attention to the occurrence of duodenal ulcers after cutaneous burns.

After extensive and wide-spread burns (Greenwood, it is true, also reports a case in which the arrest of a hemorrhage, following amputation of the penis, by hot water produced the same effect), ulceration of the intestinal canal may occur. In most exceptional cases the ulcers appear in the stomach (in addition to other ulcers in the intestine) ; in the majority of cases the duodenum is affected, and very rarely the lower portions of the intestine. My reason for not discussing this form together with simple duodenal ulcers, like all other authors, is that it is distinguished from the latter lesion both by its specific etiologic factor and by its course. I feel justified, therefore, in giving a separate account of his form of ulcer of the duodenum.

An ulcer of the duodenum that follows an extensive burn is usually situated in the upper horizontal portion of the duodenum, rarely lower down. One isolated ulcer may develop, or from three to six may be seen together ; occasionally in different stages of development, when several ulcers are present, there may be either simple hemorrhagic erosions, on the one hand, or very considerable loss of substance and destruction of tissue, on the other ; in addition there may be, though not necessarily, diffuse inflammation of the mucous lining of the intes-

[1] Moynihan, *Med.-Chir. Trans.*, vol. lxxxvi.

tine. These structural changes, even apart from other peculiarities, differentiate ulcers following burns from simple duodenal ulcer. The form of ulcers from burns is frequently irregular and dentate, or the lesions may be very long and quite narrow ($5''' : 1\frac{1}{2}'''$, $1\frac{1}{2}'' : \frac{1}{2}''$). Another and still more conspicuous difference is the very rapid development of ulcers from burns. This feature is so marked that this lesion has frequently been called acute ulcer of the duodenum. In general the lesion becomes manifest seven to fourteen days after the burn; occasionally, however, it appears as early as forty-eight hours afterward.

The fatal issue usually occurs within thirty-six to forty-eight hours after the appearance of the first definite symptoms of ulcer. The latest onset of the symptoms on record was seventeen days after the burn.

The symptom caused by ulcer of the intestine following burns is hemorrhage, usually from erosion of the pancreatico-duodenal artery, or peritonitis from perforation.

Great interest attaches to the mode of origin of these ulcers, and why they show such a particular predilection for the duodenum. The origin of these ulcers and their preference for the duodenum are undoubtedly due to the same cause, but so far it is impossible to offer as adequate explanation of these facts.

The hypothesis has been suggested that the constitution of the bile is changed in burns, or that the blood-vessels of the duodenum become occluded by embolic processes, but no proof for these views has so far been forthcoming. W. Hunter has reported a series of experiments with toluylendiamin, from which he draws certain analogies with duodenal ulcers produced by burns. He found that subcutaneous injections of toluylendiamin produced the same lesions of the duodenum and the same clinical picture as cutaneous burns. He concludes from this that both in burns and after injection of toluylendiamin certain substances are poured into the duodenum with the bile, which are capable of producing inflammation of this portion of the bowel. This view is purely hypothetic. Ponfick and Sonnenburg expressed the opinion that the small hemorrhagic erosions that are seen throughout the whole digestive tract in burns are the primary cause of the development of ulcer, since autodigestion of tissue occurs in these areas. This theory, however, does not explain why the stomach is so rarely involved and why the duodenum is the chief site of hemorrhagic infiltrations.

Treatment of ulcer of the duodenum following burns and the treatment of the symptoms caused by this lesion must be carried out according to well-known indications (see intestinal hemorrhage and peritonitis from perforation). I hardly believe that any treatment will ever be successful in these cases.

Krauss, quoting an observation of Förster's, raises the question whether the ulcer of the duodenum may not also develop after frost-bite. Unfortunately, very little clinical material of this character is available, so that, for the present, this question must remain undecided.

[The incidence of duodenal ulceration after burns is estimated by

the Fenwicks,[1] on the basis of the statistics of T. Holmes, Erichsen, Perry and Shaw, to be 6.2 per cent. of all cases of fatal burns. For a recent consideration of the visceral changes which follow rapidly fatal burns the reader should refer to C. R. Bardeen's[2] paper.

Ulceration of the duodenum has been observed in pneumonia (Griffon).[3] Dieulafoy[4] has observed multiple small gastric ulcers of pneumococcal origin, and Griffon and Bezançon[5] have produced ulceration of the stomach by pneumococcal septicemia.

Ulceration of the duodenum has been observed in chronic kidney disease. Barié and Delaunay[6] report a case with very severe intestinal hemorrhage a few hours before death, and have collected 18 cases. The first part of the duodenum is nearly always affected. The patients are usually between forty and sixty years of age.—Ed.]

EMBOLIC AND THROMBOTIC ULCERS.

Embolism of the main trunk and of the larger branches of the mesenteric artery produces a clinical picture that will be discussed in another section. But if small branches of this blood-vessel are occluded, particularly some of the branches running in the intestinal wall itself, certain changes may result from this thrombosis which lead to circumscribed destruction of tissue—so-called embolic ulcers. In the older literature these ulcers were only briefly mentioned. Ponfick has described their origin with great clearness. Parenski was the first to write a monograph on this subject and to introduce the name. I have reported one case of this disease that was submitted to careful histologic examination.

The primary causes of embolism are either endocarditic processes or atheromatous degeneration of the aorta. In my case there were capillary emboli of the arteries of the intestinal wall derived from abscesses of the lungs. The patient had been in the habit of injecting morphin for a good many years, so that the skin was covered with infiltrations and small ulcerations, and the axillary glands were suppurating. The woman also suffered from albuminuria and died with symptoms of pyemia. The autopsy revealed large white kidney filled with small pus foci, abscesses in the lungs, and numerous miliary abscesses in the intestine. Some of the latter had developed into ulcers. Embolic ulcers of the intestine may be found from the duodenum downward as far as the cecum, but are very rarely met with in the colon.

The pathogenesis and the anatomic appearances of this disease vary according to the size of the vessel occluded and the character of the embolus. An aseptic embolus necessarily produces different consequences than one of an infectious nature. Embolism of a very small

[1] Samuel and Soltau Fenwick, *Ulcer of Stomach and Duodenum.*
[2] C. R. Bardeen, *Johns Hopkins Hosp. Repts.,* 1898, vol. vii.
[3] Griffon, *Bull. Soc. Anat.,* Paris, 1899, p. 611.
[4] Dieulafoy, *La Presse Méd.,* November 4, 1899.
[5] Griffon and Bezançon, *Bull. Soc. Anat.,* Paris, 1899, p. 409.
[6] Barié and P. Delaunay, *Soc. Méd. des Hôp.,* January 16, 1903, p. 45.

vessel can usually be detected in the submucous layer of the intestine, where it gives rise to little swelling and a slight degree of hemorrhagic reddening of the surrounding tissue. If larger plugs occlude larger vessels, small-celled infarction of the neighboring tissues will be seen, together with hemorrhagic infiltration. In the central portions of these areas necrosis is frequently present, and an ulcer may be thus produced. Parenski furnishes the following description of a case of this kind: " In the cecum several (about 5) folds are seen to be hyperemic, swollen, solid, infiltrated with blood, and almost black in color. The submucous tissues corresponding to this area are also infiltrated. In the middle of these folds the mucous membrane is seen to be suppurating." The peritoneum is often found to be hemorrhagic and swollen in the areas corresponding to the affected portions of the intestinal wall.

Ulceration of the intestine occurs from necrotic degeneration of the tissues in the area of distribution of the occluded blood-vessel. The ulcers formed in this way are, as a rule, small, and rarely acquire large dimensions. Sometimes they are circular, in other cases they are girdle-shaped, and in still others irregular in outline. As necrotic disintegration of these areas occurs very rapidly, the whole thickness of the intestinal wall may become involved, so that fibrinous, purulent, or perforative peritonitis finally develops. Within the intestine diphtheric or croupous inflammation of the mucosa is occasionally seen in the neighborhood of the necrotic portions of the wall.

As a rule, a number of ulcers are found, for the process is such that it necessarily leads to the development of more than one ulcer. In addition infarction of some of the other organs (the spleen, the kidneys, etc.) may be found.

In pronounced cases of capillary embolism that occur in the course of septic processes small nodules (miliary abscesses) are seen in the submucosa. These nodules consist of a conglomeration of numerous closely congregated round-cells surrounding a blood-vessel. The accumulation of round- (pus-) cells may infiltrate the serosa on the one side, or the mucosa on the other; it may destroy the latter, and produce ulceration of the intestine with a free opening into the intestinal lumen.

Parenski has reported a case in which firm cicatrices of healed ulcers were disseminated throughout the whole wall of the jejunum. These cicatrices were pigmented and almost black. Examination of the whole case showed that the ulcers which had produced these cicatrices were of embolic origin.

The symptoms of ulcer due to embolic necrosis of the intestine are the same as those produced by other ulcerative processes of the intestine, for every ulcer, after it is once formed,—and it is immaterial what its etiology,—produces a certain typical train of symptoms that will be considered below. When it is possible to diagnose an ulcer of the intestine at all, the question arises, whether or not it is due to some embolic process. In any given case the following circumstances will favor this diagnosis:

In the first place, some focus must be discovered in the body from

which emboli can originate (endocarditis, arteriosclerosis, pyemia with the probability of abscesses of the lungs). In the second place there must be evidence more or less certain of the presence of emboli in other organs (spleen, kidneys, skin). A very important point is the appearance of symptoms that indicate embolism of the trunk or of one of the larger branches of the mesenteric artery (compare the section on this condition), particularly if these symptoms precede the appearance of symptoms of ulcer. Another important point is the development of acute peritonitis after symptoms suggesting the presence of an ulcer. At best, however, the diagnosis of embolic ulceration of the intestine is rarely positive, and only in exceptional cases can it even be made with any degree of probability.

In multiple degenerative neuritis ulcers of the intestine have occasionally been observed which are without doubt pathogenetically related to the preceding group. These ulcers must be called thrombotic ulcers of the intestine. Kussmaul and Maier described wide-spread degeneration of the mucosa both of the large and small intestine, with stasis, ecchymoses, inflammatory infiltration, ulceration, and superficial loss of substance in a case that was called periarteritis nodosa at the time. This case has been frequently quoted in the literature, and while it is described as a periarteritis, it properly, according to our present views, belongs to the present group. Kussmaul himself called attention to the similarity of the general features in this case with the syndrome of embolism of the mesenteric artery. Minkowski also describes a peculiar condition of the intestinal mucosa in multiple neuritis. In his case the intestine seemed to be irregularly pigmented, and throughout the whole bowel numerous ulcers were disseminated that were either longitudinally or transversely placed and had an irregular dentate outline. These ulcerations were arranged along the whole circumference of the intestinal lumen, but did not involve the lymphatic follicles. Some of these ulcers extended as far as the muscular coat, and one of them produced perforation. Minkowski expresses the opinion that this formation of ulcers in the intestine was due to some circulatory disturbance in the bowel-wall which, in its turn, was caused by the disease of the blood-vessels observed in this case. Many thrombotic and obliterated blood-vessels could be found in the immediate vicinity of the ulcers. Lorenz also reports a case of multiple neuritis in which extensive lesions were found involving wide areas of the jejunum, some of these ulcers penetrating as deep as the serosa ; in addition there were numerous small foci of ulceration ; the bases of these ulcers consisted of dark, brownish-black, softened tissue, and their margin was formed by injected and somewhat swollen folds of mucous membrane. The small branches of the mesenteric artery that supplied these areas were thickened to such an extent by arteritis that in some cases their lumen was almost occluded.

All these cases have, in common, changes of the arterioles leading to occlusion of the lumen of the blood-vessels. This peculiar arterial condition has lately been found to be quite frequent in infective degenerative multiple neuritis, consequently the intestinal ulcers met with

in this form of neuritis must be included in the group of ulcers due to circulatory disturbances. Their presence may be suspected on fair grounds when more or less typical intestinal symptoms (diarrhea, peritonitis from perforation) develop in a patient with multiple neuritis.

Mouisset states that ulcers of the large intestine are far from rare in arteriosclerosis, and are probably due to vascular changes. It must be remembered that Mouisset's two patients also had nephritis, and that these hypothetic arteriosclerotic ulcers are exceedingly rare as compared with the frequency of arteriosclerosis. His view, therefore, appears to me to be ill founded.

Acland and Hale White have called attention to the possibility that ulceration of the intestine may develop in certain diseases of the spinal cord. The lesions in these cases would be comparable to other trophic lesions that follow diseases of the spinal cord—for instance, bed-sores ; in other words, this form of ulceration must be regarded as trophic. These authors studied 3 cases in which this origin of intestinal ulceration seemed possible. I do not think, however, that the cases they describe were sufficiently typical to justify any definite conclusions, or that this question can be considered solved. So far as I know, no other observations of this kind have been made.

[Targett [1] and C. Ogle [2] have described two cases in which fracture of the spine was followed by death in five days, in both cases diarrhea beginning on the second and third days after the accident, and a high temperature preceded death and the colon was extensively ulcerated. In Targett's case the spine was fractured at the level of the third and fourth lumbar vertebræ, and in Ogle's at the sixth dorsal vertebra. In neither were there scybala in the colon.—Ed.]

AMYLOID ULCERS.

The majority of authors consider amyloid ulcers of the intestine to be very rare. Friedreich, Aufrecht, and some other authors have described isolated cases of this condition. Colberg and Courtois-Suffit, on the other hand, consider the formation of ulcers in the intestinal mucosa after it has undergone amyloid degeneration to be frequent even in the absence of tuberculosis.

It seems that amyloid ulcers may be found in any portion of the intestine. They are usually multiple, and the ulcerative process may involve large portions of the intestines. Small erosions are found that are no larger than a lentil, together with large, girdle-shaped ulcers which occupy the whole circumference of the intestine. Some of the latter may be 5 to 15 cm. long. The margins of these lesions are perfectly smooth and occasionally somewhat elevated. The outline of the smaller ones is so distinct and their margin so sharp that they seem to be punched out of the mucosa. The large ulcers have a pale base covered with a number of radiating strands. The latter consist of blood-

[1] Targett, *Trans. Path. Soc.*, 1892, vol. xliii., p. 73.
[2] C. Ogle, *ibid.*, 1897, vol. xlviii., p. 97.

vessels whose walls are stiff and rigid, owing to amyloid degeneration. Amyloid ulcers penetrate as far as the muscular coat—may even penetrate this layer itself. No inflammatory changes are seen in the areas of intestine situated between the different ulcers; but, as a rule, however, these portions are in a state of amyloid degeneration. Colberg calls particular attention to the fact that the tendency to heal or to form cicatrices is almost completely lost in ulceration that occurs in an amyloid intestine.

Leube is inclined to attach special importance to the circulatory disturbances that supervene in the small arterioles of the intestinal wall in these cases. He claims that these ulcers originate from circulatory disturbances which in their turn are due to amyloid degeneration of the blood-vessel walls. According to Colberg, these ulcers are produced by two factors: in the first place, the blood-supply must be deficient and the general nutrition of the parts impaired by the amyloid degeneration; in the second place, some mechanical or chemic irritant must be operative that can be made directly responsible for the development of the ulcer. He argues that the ingesta themselves constitute such a mechanical and chemic irritant in these cases, for the villi are necessarily fragile, owing to the anemia and rigidity of the degenerated tissues of the mucosa, and are therefore easily broken off by the passage of the food, and that this leads to ulceration of the mucosa. In addition he believes that certain urinary decomposition-products are always present in the intestinal contents in these cases, owing to the frequency of amyloid disease of the kidney.

The symptomatology of amyloid ulcer will be dealt with in a subsequent paragraph in connection with other forms of intestinal ulcer.

SECOND GROUP.—CATARRHAL AND FOLLICULAR ULCERS.

Ulceration of the intestine occasionally occurs in the course of catarrhal inflammation of the intestinal mucous membrane. These ulcers are usually divided into simple catarrhal and follicular ulcers, this classification depending on the situation in which the ulcers begin. An ulcer is spoken of as follicular ulcer when it starts in the lymphatic follicles of the intestine; as catarrhal, when it develops in the mucous membrane. However justifiable this separation may be from the point of view of pathologic anatomy, clinically both forms must be considered together, for they are frequently found in association, especially when the catarrhal inflammatory process in the intestine is particularly severe. Both forms of ulcer may originate in catarrh of the intestine, whether in the acute or in the chronic form. They are particularly frequent in the chronic form when an acute exacerbation occurs.

Virchow has vigorously objected to the term "catarrhal ulcer." While indorsing his opposition to this term, and while admitting that his objections are logical, I do not see how we can very well do away with this title, since these ulcers do actually develop as a result of catarrh of the intestinal mucosa.

Simple catarrhal ulcers are usually situated in the large intestine ; less frequently, in the small intestine. They may be isolated, or they may be present in large numbers and extend over considerable portions of the intestine. In milder degrees of ulceration the lesions constitute small superficial circular erosions that are no larger than a lentil and rarely extend downward into the tissues as far as the submucosa. As the process develops, several of these small ulcers may become confluent, and in this way large, irregularly shaped ulcers are formed that may extend to the muscular layers or through the muscular coat, and may even lead to perforation of the bowel. While the ulceration is still in process of development, the margins are usually somewhat thickened and show infiltration with small hyperemic cells, and are also slightly edematous and may be undermined. The base of the ulcer is usually seen to be suppurating. The mucous membrane between these larger ulcers forms islands of catarrhal, swollen tissue.

These ulcers, provided they are not too numerous and too large, may heal by cicatricial contraction. The process of healing, however, usually leads to more or less noticeable degrees of stenosis. The mucous membrane in the immediate neighborhood of the lesions frequently forms polypous ridges and folds (Rokitansky).

The histology of this form of ulcer and the exact mode of origin are interpreted somewhat differently by various authors. Rokitansky, Leube, and others state that suppuration may begin either on the surface of the mucosa or in the substance of the mucous membrane of the intestine. When suppuration begins on the surface, it extends downward into the intestinal wall ; when it begins within the mucosa, it perforates into the free lumen of the intestine. According to Woodward's observations, simple catarrhal ulcers of the intestine always begin on the surface. I have examined this point carefully in many cases, and fully indorse the view of Woodward. I have never seen a case of simple acute or chronic catarrh of the intestine in which histologic examination showed that the catarrhal ulcer originated from primary submucous collections of pus-cells (compare, on the other hand, embolic ulceration of the intestine).

The first step in the development of ulcers of the mucous membrane of the intestine after the commencement of catarrhal changes is an accumulation of round- (pus-) cells in the mucosa. These pus-cells surround the glands of Lieberkühn. The epithelium begins to desquamate, the glands either slough off completely or, in very shallow ulcers, only slough off in part, so that in the latter case the superficial half of the glands may be absent, while the fundus may still be seen in the mucosa. Gradually the destructive process extends downward into the intestinal wall. At the same time the accumulation of round-cells continues. The follicles may remain quite unaffected, so that, in some instances, superficial ulceration of the mucosa may be seen in immediate proximity to a well-preserved follicle.

Hale White has recently described a form of disease that he calls "simple ulcerative colitis." No exact histologic description is given,

but, so far as I can judge from his description of the lesions, this simple ulcerative colitis is nothing more than the simple catarrhal form of ulceration of the intestine just described, which should be distinguished from follicular ulceration.

[ULCERATIVE COLITIS.

This term has, at any rate in England, been used to describe a special form of ulceration of the colon and not any form of ulceration met with in the large intestine. It has been specially described by Hale White,[1] and occurs sporadically in general hospital practice and in epidemics, which are very formidable, in the asylums for the insane. The form met with in asylums has been specially dealt with by Gemmel,[2] who in 1898 gave an account of an epidemic in the County Asylum of Lancaster in which there were 114 deaths and 80 autopsies. His work, which perhaps has been rather overlooked, led him to the conclusion that the disease is indistinguishable from dysentery. From the bacteriologic researches of Goodliffe, which were incorporated in Gemmel's monograph, it appeared that the disease was due to a specific bacillus closely resembling the colon and typhoid forms. Mott[3] also (1902) came to the conclusion that the so-called ulcerative colitis of asylums was dysentery. It appears that the identity of the asylum disease with dysentery was definitely settled by Vedder and Duval (1902),[4] who, working under Flexner, found that the sporadic and institutional (asylum) outbreaks of acute dysentery are due to the same micro-organism as that causing acute epidemic dysentery in Japan (Shiga), the Philippine Islands, and Porto Rico (Flexner). The blood-serum of their patients agglutinated bacilli from these sources and also Kruse's micro-organism from cases of what he called the pseudodysentery of lunatic asylums in Germany. It is, therefore, unnecessary to give a separate description of the disease, since it may be regarded as dysentery.—ED.]

Follicular ulcers may occur in both the small and the large intestine, but are much more frequently met with in the latter portion of the bowel. Follicular ulcers may appear as isolated lesions or may be present in such large numbers and reach such a size that the whole colon is covered with ulcers. In fact, the ulcerated portions of the mucous membrane of the colon may occupy as much of the surface of the bowel as the intact mucous membrane.

In the earliest stages each ulcer appears as a minute opening leading into an excavated follicle ; later the lesion becomes funnel shaped. The base gradually extends, the edges of the ulcer become infiltrated, and the mucosa undermined for some distance from the lesion. Sometimes a piece of undermined mucosa containing well-preserved glands of

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[1] Hale White, *Guy's Hosp. Repts.*, 1888, vol. xlvi. ; *Allbutt's System of Med.*, 1897, vol. iii., p. 950.

[2] Gemmel, *Idiopathic Ulcerative Colitis* (*Dysentery*), Baillière, Tindall and Cox, 1898.

[3] Mott, *Trans. Epidemiological Soc.*, 1902 ; Mott and Durham, *Report on Colitis or Asylum Dysentery*, 1901.

[4] Vedder and Duval, *Jour. Exper. Med.*, February 5, 1902, vol. vi., p. 181

Lieberkühn may seem suspended over the ulcer or hang down into it. To begin with, these ulcers are circular in shape, owing to the fact that the lesion is limited to one follicle. As the disease progresses, the process of destruction involves the perifollicular tissues. The ulceration advances into the submucosa, the mucous layer lying above the affected areas of the mucosa becomes necrotic, and in this way irregularly shaped ulcerative lesions are developed. At this stage it is impossible to determine whether the ulcers were originally follicular or simply catarrhal in character. In the early stage these lesions may heal, and if the destruction of tissue is only moderate, cicatrization with stenosis of the bowel may occur. In other cases the ulcerative process extends downward through the intestinal wall and finally leads to perforation. If the destruction of tissue is very great, the lesions do not heal.

The histology of follicular ulceration of the intestine is simple. The catarrhal irritation of the intestinal wall leads to great accumulation of cells between the glands of the mucosa and in the uppermost layers of the submucosa, as well as in the follicles. The cellular infiltration of the follicles leads to the formation of a small nodule that projects into the lumen of the intestine. As the accumulation of cells progresses necrosis occurs, the swollen follicle discharges, and in this way the ulcer originates.

STERCORAL OR DECUBITAL ULCERS.

Stercoral ulcers, or, as Grawitz has lately called them, decubital ulcers, are produced by the pressure of hardened stagnating intestinal contents on the mucosa. In this way necrosis of the superficial layers of the mucous membrane and later purulent inflammation of the submucosa are produced. This form of ulceration consequently rarely develops in the lowest portions of the small intestine and is most frequent in the large intestine, particularly in those portions of the colon in which stasis of fecal matter is most liable to occur—for instance, the hepatic and splenic flexure, the cecum, the sigmoid flexure, the rectum, and particularly the appendix. In the rectum they present the appearance described as proctitis ulcerosa (*cf.* syphilitic enteric ulcers). The ulcerations that usually develop above enteric stenoses, and are described by Kocher as distention tumors, belong only partly in this group. (For further treatment of them see p. 348.) Stercoral ulcers are also frequently found above stenoses of the intestine. When this occurs, a very characteristic anatomic appearance results, for below the stenosis the mucosa is seen to be perfectly normal, whereas above the stenosis it is found to be ulcerated. Stercoral ulcers in the rectum produce the morbid condition known as proctitis ulcerosa (compare also syphilitic ulcers of the intestine).

Dickinson and, subsequently, Grawitz have called particular attention to the circular outline of these decubital ulcers, particularly in those portions of the colon where the bowel is kinked. The ulcers frequently heal by cicatrization and produce stenosis of the colon, and Grawitz remarks that such cicatricial strictures are often erroneously regarded as

of carcinomatous or syphilitic origin. I should like to supplement this statement by calling attention to the fact that these small circular stenoses in these portions of the bowel are, in point of fact, very frequently found to be carcinomatous, and may, nevertheless, have originally developed from the cicatricial tissue of an ulcer of the colon which, in the first instance, was purely decubital. This secondary development of carcinoma is analogous to the development of cancer in the cicatricial tissue formed by a healed ulcer of the stomach.

[The condition of simple ulcer of the colon described by Quénu and Duval[1] as analogous to gastric ulcer probably belongs to this category. They described 27 cases, chiefly in the pelvic part of the colon. The symptoms were vague until perforation occurred, which was met with in 19 instances out of the 27.—ED.]

THIRD GROUP.—ULCERATION IN ACUTE INFECTIOUS DISEASES.

Ulceration of the intestine develops in a variety of acute infectious diseases. In some of these infections ulceration of the intestine constitutes one of the characteristic features of the whole of the disease-process. For this reason these specific ulcers, so to say, are discussed under the heading of the different infectious diseases—for instance, typhoid ulcers in typhoid fever, dysenteric ulcers in dysentery. In the latter section so-called diphtheric ulcers will also be described. In anthrax there is a hemorrhagic pustulous infiltration of the intestine, with sloughing and ulceration. In a number of other acute infectious processes ulcerative lesions of the intestine are observed in exceptional cases only. They are not chance complications of these diseases, but must be considered intimately and directly connected with the primary process. They are distinguished from the former group merely by the fact that they do not occur constantly. I will briefly consider some of the latter forms of ulcer.

It is well known that in septicemia diarrhea is a frequent symptom (compare catarrh of the intestine); nevertheless lesions of the intestine will rarely be found that have progressed to ulcerative destruction of bowel tissue. This complication then is exceedingly rare. In 27 of the cases of this disease that were examined postmortem in my clinic during the last few years there was no ulceration of the intestine.

Billroth has reported the case of a subject who was suffering from fatty degeneration of the heart and cirrhosis of the liver. An operation on the thyroid was performed, and two days later the patient developed intestinal symptoms, passed bloody stools, and died on the sixth day with an antemortem rise of temperature to 104.2° F. At the autopsy a number of ulcers were found in the duodenum. Billroth believes that these ulcers were septicemic in origin. He explained the occurrence of this accident as follows: It is known that in experimental septicemia the mucous membrane of the intestine is found to be hyperemic, inflamed, and hemorrhagic. If in a human subject with septicemia

[1] Quénu and Duval, *Rev. de Chir.*, 1902, vol. xxv., p. 692.

certain additional factors are operative, such as fatty degeneration of the heart and cirrhosis of the liver, that lead to venous stasis in the intestine, portions of the hyperemic inflamed or hemorrhagic intestinal mucosa may undergo autodigestion, and in this way become ulcerated.

According to this view, septic ulcers of the duodenum must be considered the result of certain accessory factors, not of sepsis itself. The same undoubtedly applies to those cases in which hemorrhages of the intestine, embolism of the mesenteric artery, and ulceration of the intestine develop in the course of septic endocarditis. All these ulcers are without doubt embolic in character. We can only say that an ulceration of the intestine is directly dependent on the general septic process if no other complications are present which can be made responsible for the development of the ulcers. Pathogenetically, therefore, we must attribute the formation of ulcers in sepsis to the presence of a hemorrhagic septic catarrh of the intestine leading to local necrosis of the intestinal mucosa.

Duodenal ulcers have occasionally been discovered in erysipelas (Larcher, Petitbien), but is so rare that very little can be said about its occurrence. Pathogenetically the same arguments apply as in the case of ulceration of the intestine in septicemia. In varioloid, sloughing of the follicles of the intestine with secondary tumefaction of the mesenteric glands is occasionally seen combined with catarrh of the intestine and intestinal hemorrhages. It is very doubtful whether ulceration of the intestine ever develops from pustules (Curschmann). Finally, it may be mentioned that in pemphigus acutus and in pellagra ulceration of the intestine has, in isolated instances, been observed. It remains undecided whether the occurrence of ulcers in these diseases is an accidental complication or is directly dependent on the primary disease.

[Duodenal ulceration in pneumonia has been referred to on p. 248. —ED.]

FOURTH GROUP.—TUBERCULOUS ULCERS.

Tuberculosis is one of the most frequent causes of ulceration of the intestine. This disease is of the same importance in this respect as typhoid, dysentery, and catarrh of the intestine. Bayle (1810) was the first to call attention to the occurrence of tuberculous ulceration of the intestine and to emphasize the frequency of this complication in tuberculosis.

Tuberculous ulcers are chiefly found in the cecum, and particularly in the lowest part of the ileum, and may, in fact, be found only in the latter situation. From this point they usually extend downward toward the colon and as far as the rectum, or upward into the jejunum and even the duodenum. Occasionally they seem to develop primarily in the colon; this impression, at least, is given in many cases in which the postmortem examination shows that the ulcers are most numerous in the colon, and that the stage of development of the ulcers in this portion of the bowel as compared to the development in other portions

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seems to indicate that this was the primary starting-point of the process. The number of tuberculous lesions in individual cases may vary greatly.

The development of intestinal tuberculous ulcers is preceded by the formation of a miliary tuberculous nodule. The tuberculous process always begins in the lymphatic follicles of the intestine, and not, as was formerly believed, in other portions of the mucosa. The proliferation of cells causes swelling of the solitary follicles. When caseous degeneration of the central portions of the follicle begins, the swollen tuberculous follicle opens and in this way a small, crater-like ulcer is developed which is about as large as a millet-seed or a pea (Rokitansky's primitive tuberculous ulcer). The same eruption of tubercles and formation of ulcers occurs in the agminate follicles as in the solitary ones, the process developing in the same manner. A characteristic feature of this tuberculous swelling, which is exactly the reverse of the first stage of typhoid and other catarrhal ulcers, is that only isolated follicles of the plaques are involved in the tuberculous infiltration, whereas in typhoid and catarrhal swelling the whole Peyer's patch is uniformly affected. In the tuberculous form some of the follicles of a plaque may, therefore, be infected, while others remain perfectly free from infection. As a result, ulcerative softening occurs only in certain points of a plaque ; finally, of course, the whole patch becomes involved in the process and undergoes typical ulceration.

The growth of the primitive ulcer may occur in two ways : the neighboring tissues may either undergo purulent infiltration or fresh tuberculous nodules may develop around the ulcer in the different layers of the intestinal wall ; these finally undergo caseous degeneration and in this way cause enlargement of the original ulcer.[1] The surface of the ulcer usually extends along the transverse axis of the intestine— namely in a direction that corresponds to the areas supplied by the different blood-vessels and their branches (Rindfleisch). In the small intestine the growth of the ulcer proceeds in a direction that is parallel to the valvulæ conniventes, so that finally an ulcerating surface is formed which is placed transversely to the longitudinal axis of the intestine or forms a ring-shaped lesion involving the whole circumference of the bowel. The latter form is called tuberculous girdle ulcer.[2] Occasionally the ulcers are elongated or quite irregular in form. These varieties are usually due to the confluence of a number of single ulcers. The downward growth of the ulcer is frequently arrested as soon as the muscular coat is reached. As a rule, however, small tuberculous nodules are seen in the muscular coat which frequently follow the distribution of the lymphatic vessels. In some instances the muscular coat of the intestine is involved in the destructive process, so that the ulcer extends to the serosa ; when this happens, ulceration into the peritoneal cavity may occur (compare the serosa below).

[1] Woodward, on pages 587 and 588 of his beautiful work, gives two very instructive photomicrographic illustrations of this lesion.

[2] The terms tuberculous ulcer of the intestine and girdle ulcer of the intestine are not synonymous by any means, for ulcers due to embolism or thrombosis, to amyloid degeneration, to syphilis, or decubital ulcers may all be girdle or ring-shaped.

A fully developed large tuberculous ulcer has an irregular margin which may be perfectly smooth, swollen, or undermined. As a rule, the margin is of a light-red color. The base of the ulcer is greasy, and consists of tissue in all stages of degeneration. Occasionally patches of swollen mucosa are seen on the base of the ulcer. There is often tuberculous infiltration of the base and margins of the ulcer. The surrounding tissues are very often the seat of catarrhal changes.

The peritoneal covering of the bowel corresponding to the ulcer is usually changed in so characteristic a manner that the position and the nature of the ulcer can often be recognized on simple inspection of the outside of the intestine. The serosa will be found in a condition of chronic inflammation, reddened, thickened, and covered with layers of fibrin; at the same time it is usually adherent to other loops of intestine—the mesentery, the urinary bladder, etc.—by strands of connective tissue. In these areas of circumscribed peritonitis more or less numerous tubercles are almost always present. Peritoneal adhesions form so constantly in this disease that peritonitis from perforation is comparatively rare (as compared with the great frequency of tuberculous ulcerations).

Another very common change in this disease is swelling and tuberculous infection of mesenteric lymph-glands.

We may mention here a particular form in which tuberculosis of the intestines is occasionally seen. This is the tuberculous ileocecal tumor recognized only within the past ten years. As this form differs anatomically and clinically from ulcers, it will be described with the group of intestinal diseases which it resembles more closely—viz., stenoses (see p. 468).

[**Chronic hyperplastic tuberculosis of the intestine** is a special and peculiar form of change characterized by great thickening of the walls of the bowel and some narrowing of the lumen. There is diffuse infiltration of the wall of the bowel with adhesions to the surrounding fat. Conrath,[1] in 1898, collected 85 cases, mostly in the ileocecal region; it may attack the small intestine, the cecum, colon, and rectum. There may be multiple strictures. In 77 cases analyzed by Conrath 41 were women and 36 men; the majority of the patients are between twenty and forty years of age. The extreme hyperplasia is due either to an attenuated condition of the tubercle bacilli or to a mixed infection. Tubercle bacilli are rare. A certain number of the cases are primary tuberculous infections of the intestine (Lartigau).[2] Some of the reputed cases of syphilitic strictures of the rectum are really tuberculous, as shown by Sourdille,[3] and Lapointe[4] has collected 9 cases of stenosing tuberculous proctitis. (For an exhaustive account of hyperplastic tuberculosis of the intestine the reader should consult Lartigau's article).—Ed.]

As a rule, tuberculous intestinal ulcers evince a tendency to be pro-

[1] Conrath, *Beitrag z. klin. Chirurg.*, 1898, vol. xxi., p. 1.
[2] Lartigau, *Jour. Exper. Med.*, November 29, 1901, vol. vi., p. 23.
[3] Sourdille, *Arch. gén. de Méd.*, 1895.   [4] Lapointe, *Thèse*, Paris, 1897.

gressive, as is shown by the continued eruption of new foci in their bases and margins. Nevertheless, there is no doubt that cicatrization does occur in some cases, and leads to considerable stenosis on account of the circular shape of the ulcer. König emphasizes the fact that these stenoses are distinguished by their narrowness and relatively great length; and that they are not infrequently localized and isolated, at least so far as this is possible in tuberculous conditions.

We distinguish a primary and a secondary intestinal tuberculosis. In the former, the intestine is the only organ affected; in the latter, the intestinal tuberculosis is the result of a similar condition in other organs, most frequently the lungs. Other organs are not so often affected, but the genital organs are the most important in this connection. The primary form does occur, but is exceedingly rare. It is shown conclusively in nurslings and young children, but in older children and adults intestinal tuberculosis is almost exclusively secondary. The most recent statistics show that it is associated with tuberculosis of other organs in from 56 to 98 per cent.

[Koch [1] has seen primary intestinal tuberculosis on only two occasions; he quotes the following statistics in support of his views—at the Charité Hospital, Berlin, there were 10 cases in five years; in 933 cases of tuberculous children Baginsky never found intestinal tuberculosis without simultaneous disease of the lungs and bronchial glands; in 3104 autopsies on tuberculous children Biedert found only 16 cases. English statistics show a much higher percentage of primary intestinal tuberculosis. In 120 autopsies on tuberculous children Carr [2] found the disease primary in the bowel or mesenteric glands in 20; in 77 similar cases Guthrie [3] found 19; in 269 Still [4] recorded 53, and in 355 Shannon [5] found that in 28.1 per cent. the infection was primarily intestinal.—ED.]

Our ideas in regard to the origin of tuberculous infiltration and ulceration of the intestinal canal have undergone many changes within recent years. Formerly the literature was full of discussions on this subject; nowadays the matter has been simplified by the discovery of Koch's tubercle bacillus, for with this discovery the cause of the pathologic changes in tuberculous ulceration of the intestine was revealed. We also know how this germ enters the intestine, or at least are familiar with the conditions essential for intestinal infection by the tubercle bacillus. There are two chief possibilities for consideration : Klebs was the first advocate of the theory that tubercle bacilli are carried into the intestine chiefly by the sputa swallowed by patients with pulmonary tuberculosis. This is undoubtedly the most frequent form of infection, and Klebs called attention to this possibility even before the true character of the tuberculous virus was recognized. As a matter of fact, I believe that the great majority of cases of secondary tuberculosis of the

[1] Koch, *Brit. Med. Jour.*, 1901, vol. ii., p. 189.
[2] J. W. Carr, *Lancet*, 1894, vol. i., p. 1177.
[3] Guthrie, *ibid.*, 1899, vol. i., p. 286.
[4] Still, *Brit. Med. Jour.*, 1899, vol. ii., p. 455.
[5] Shannon, *Edinburgh Hosp. Repts.*, 1900.

intestine are due to the swallowing of sputum. Even before the tubercle bacillus was known, a number of authors performed experiments with sputa, and the results of these older investigations alone force us to accept this view. Finally the more recent investigations by Wesener and others have established the possibility of infection of the intestine by tuberculous sputum.

A second important source of tuberculous infection is the meat and the milk of tuberculous cows. Orth, Bollinger, Baumgarten, and others have shown that the meat and the milk of tuberculous animals can produce tuberculosis, notably primary tuberculosis of the intestine. This possibility was considered twenty years ago, but had very little support from physicians. Within the last decade, however, it has been definitely decided that this is a certain and a prolific source of infection. I need hardly call attention to the great hygienic and sanitary importance of this discovery. Tuberculous infection of the intestine from the food is undoubtedly one of the most important causes of primary tuberculous disease of the intestine and the mesenteric glands which is relatively so common in small children. No discussion will be entered into here of the questions started by R. Koch's recent contributions to this subject, as they are still open.

[At the Tuberculosis Congress in London in 1901 R. Koch made the startling announcement that bovine tuberculosis differs from human tuberculosis; that infection of man by bovine tuberculosis hardly ever occurs, and that it is unnecessary to take precautions against the transmission of tubercle to man by meat or milk. Though startling, the idea was not entirely novel, as it had been recognized that bovine tuberculosis played a less important part in the production of human tuberculosis than it was generally credited with. Koch's statement, however, raised much discussion and great dissent. Nocard,[1] J. Mc-Fadyean,[2] and among others Ravenel,[3] of Philadelphia, showed reasons why Koch's views could not be accepted. At present commissions are considering this important question both in England and in America, and pending a definite result the practical course should be to act on the assumption that bovine tuberculosis can be transmitted to man.—ED.]

A great number of very careful investigations are on record in regard to the path that the tubercle bacillus travels in order to enter the intestinal wall, and on the histogenesis of the anatomic changes that the bacillus produces in this location (Gottsacker, Orth, Baumgarten, Dobroklonski, Herxheimer). It is important to recognize that injury or desquamation of the superficial epithelium of the intestinal mucosa does not seem to be a necessary condition for tuberculous infection, for bacilli seem to be able to invade the mucosa even though the epithelium is intact. Undoubtedly, however, removal or injury of the epithelium favors this infection. In exceptional cases tuberculosis of the intestine from feeding (sputa, milk, meat) begins in the lymph-follicles. The formation of the tubercle in these structures is usually accompanied by

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[1] Nocard, *Brit. Med. Jour.*, 1901, vol. ii.   [2] J. MacFadyean, *ibid.*, 1901, vol. ii.
[3] Ravenel, *Univ. Penn. Med. Bull.*, September, 1901; *Veterinarian*, October, 1900.

the development of tubercles in the mesenteric glands. I must refrain from going into more exact details of this process.

SYPHILITIC ULCERS.

Syphilitic ulceration of the intestine is rarely met with. Such lesions are exceedingly rare in the small intestine as compared with the frequency of lues. Occasionally a syphilitic ulcer of this portion of the bowel is seen in a new-born infant. They develop chiefly in late lues, but follicular ulcers are seen in exceptional cases in the secondary period. In these cases there is either an isolated ulcer or a large number of ulcers scattered throughout the whole of the small intestine. They start from the lymphatic follicles, from the mucosa, or from the submucosa, the intestinal wall being infiltrated with a gumma which gradually disintegrates. Meschede, Klebs, Oser, Birch-Hirschfeld have all reported cases of luetic ulceration of the small intestine in adults. The base of these ulcers is hard, the margin grayish white in color, and usually more or less hard and infiltrated.

Syphilitic ulcers of the large intestine, or, to be more precise, of the rectum, are more frequently encountered, and are, therefore, clinically more important. As a rule, the ulcerative process is limited to the lowermost portion of the bowel, and in the great majority of cases to the few centimeters of rectum that are situated immediately above the anus. Occasionally, however, they are found higher up in the colon.

[Fournier [1] regards syphilis of the bowel as extremely rare, having recognized only 12 cases. Miliary gummata, thick infiltration, stenosis, cicatrices, or perforations may occur. There is nothing very characteristic about the symptoms. The diarrhea, which is very resistant and yields only to antisyphilitic treatment, may last for many years.—ED.]

Very vigorous discussion has taken place as to the pathogenesis of this form of syphilitic ulceration of the rectum. Anatomic and clinical investigation has now made it certain that syphilitic ulcers of the intestine may originate in different ways: in the first place they may be simple primary chancres. Erskine Mason, for instance, studied a number of cases of venereal strictures of the rectum in which there was no constitutional syphilis; in two of these cases the symptoms of stricture were observed before any symptoms of constitutional lues developed. Bäumler calls attention to the fact that these strictures are most frequent in the female sex, particularly in women between seventeen and thirty. Others again—for instance, Muron—attribute specific stricture of the rectum to the development of broad condylomata and the conversion of these lesions into cicatricial tissue. Finally, rectal ulcers may develop from the disintegration of tertiary lesions—gummata, as has been proved by numerous observers.

I have already mentioned that the most common seat of syphilitic ulcers is the lowest part of the rectum. Virchow calls attention to

[1] Fournier, *Acad. de Med.*, July, 1900.

another feature of this form of specific ulcers by which he distinguishes them from dysenteric ulceration of this portion of the bowel—namely, their slight depth and smooth base. Birch-Hirschfeld finally called attention to certain changes in the immediate vicinity of these ulcers and to the differences between syphilitic and dysenteric ulcers. In the former he claims that the cicatricial induration of the neighboring parts is relatively much more pronounced than in the latter.

Luetic ulcerations of the lower portion of the bowel are clinically very important from their tendency to lead to the development of severe rectal strictures. There is a popular belief to the effect that syphilitic ulcers have a special tendency to produce rectal strictures. This view has quite recently been opposed by Poelchen and Nickel, for these authors showed that syphilitic tissue-changes can only rarely be demonstrated with certainty in the majority of cases of rectal ulcer and rectal stricture; they also showed that many forms of rectal ulceration that are supposed to be syphilitic are frequently of quite a different origin. Poelchen, who, by the way, also calls attention to the enormous prevalence of this lesion in women (in 219 patients with ulcers of the rectum leading to stricture 190 occurred in women), explains the origin and development of a large portion of the ulcerations of the intestine producing stricture in a somewhat different way. He claims that gonorrheal abscesses of the glands of Bartholin often lead to sloughing and ulceration of the rectum. He believes that this process is due to infection of the submucous tissue of the rectum following the suppurative adenitis and periadenitis that exist in these cases. Nickel, on the other hand, is inclined to the belief that many of the so-called syphilitic ulcers are in reality traumatic ulcers due to injury from the nozzles of syringes, etc., or that the ulcers are decubital (see above). This author, in addition, accepts and indorses Poelchen's views.

[Some cases regarded as syphilitic strictures of the rectum are in reality tuberculous. Sourdille [1] showed that 5 cases of the type usually spoken of as syphilitic were really tuberculous, and Lapointe [2] has added 4 more, making 9 examples of stenosing tuberculous proctitis. —Ed.]

FIFTH GROUP.

The forms of intestinal ulcer belonging to this group are of subordinate practical importance.

In leukemia soft lymphatic tumors may occur in the intestine, particularly in the ileum, which may become ulcerated and form ulcers with high circumvallate margins. In chronic leukemia intestinal ulceration is the exception; in acute leukemia, a much rarer disease than the chronic form, ulcerative destruction of the intestine, together with serious involvement of the whole digestive tract, is seen with relative frequency (Lauenstein, Fränkel, Ebstein, Askanazy, and others). In this form of ulceration the soft parts are usually infiltrated with lymphatic elements. This infiltration is then secondarily followed by necrosis and ulceration.

[1] Sourdille, *Arch. gen. de Med.*, 1896.   [2] Lapointe, *Thèse*, Paris, 1897.

The intestinal symptoms seen in scurvy correspond to the cutaneous manifestations of the disease. Postmortem hemorrhagic spots and erosions which may form ulcers are often seen. In a few instances extensive sloughing and ulceration of the follicles have been seen, in addition to the above-named forms of ulcer, which were due to hemorrhages occurring into the intestinal wall. The follicular ulcers are small, and their margins are frequently infiltrated with blood.

A few isolated statements occur in the literature with regard to the ulceration of the intestine in gout. Personally, however, I have never seen such a lesion, and the reports are so scanty and so isolated that they hardly deserve serious consideration.

SIXTH GROUP.

In this group of toxic ulcers those caused by the direct effect of corrosive poisons on the intestine, after their introduction either *per os* or *per rectum*, are not included. This group has already been considered in the introductory remarks to this section, and hence the description will be limited to those forms of ulcer of the intestine that are produced indirectly—in other words, constitutionally.

UREMIC ULCERS.

Many of the older authors speak of the occurrence of ulceration of the intestine in Bright's disease. Frerichs, for instance, says : " Here and there round follicular ulcers or shallow large catarrhal areas of sloughing are found in the intestine, the latter particularly in the sigmoid flexure and in the rectum." It seems established that these ulcerations are directly dependent on the existence of nephritis in the same way as the diarrhea and other catarrhal changes in the intestinal mucosa that are seen so frequently in affections of the kidney. Treitz's statistics show that among 220 cases of nephritis there was " croupous and ulcerative dysentery " 19 times and "ulceration, partially recent, partially gangrenous," in 12 cases.

The ulcers in rare cases are follicular in character and involve the solitary follicles and Peyer's patches. Some of them are small, with distinct, undermined margins (Rosenstein, Dickinson). At the same time the mucous membrane of the intestine will be found to be in a state of catarrh. More frequently, however, the small and the large intestine show diphtheric changes which may involve large or small areas, may produce superficial or deep destruction of tissue, and occasionally may even become gangrenous.

These ulcerous growths are found most frequently in the large bowel, the rectum, and the lower ileum, but they may spread over the entire small intestine. It is noteworthy that at times they are localized in the duodenum, as has recently been stated by several observers, among them Dickinson and Perry and Shaw. [See also Barié and Delaunay, p. 265.—Ed.]

Dickinson has advanced the view that these "albuminuric ulcers"

with follicular suppuration originate from small hemorrhages into these parts, but he is quite alone in his view. The changes in the blood produced by nephritis can undoubtedly be made responsible for the occurrence of diarrhea and the development of catarrh of the intestine in Bright's disease; it is also very probable that they cause ulceration. The most wide-spread belief is that they are caused by the irritation of the ammonium carbonate which is developed from the urea excreted in the lumen of the intestine. Hlava believes the necroses and ulcers are caused by primary capillary thromboses. Other authors seek to explain them as the result of irritation by retained urinary constituents excreted by the intestine, but do not state which constituent it is.

[Mathieu and Roux,[1] finding no evidence of acute or subacute inflammation, regarded the ulceration as due to necrosis of the mucous membrane due to a toxic body either produced by micro-organisms or manufactured in the body. Mouisset[2] describes ulceration due to arteriosclerosis of the intestine, the first stage being a raised patch in the mucous membrane which subsequently ulcerates; he considers that the reason why intestinal ulceration is rare in young subjects dying with uremia from scarlatinal nephritis is because arterial changes have not had time to develop. Mathieu and Roux, on the other hand, state that uremic ulceration almost always occurs between eighteen and twenty-five years of age. The ileum is the part usually attacked. Roux and Mathieu describe a case in which there was a continuous ulcerated area thirty inches long in the ileum. Barié and Delaunay[3] have collected 18 cases of uremic ulceration of the duodenum which nearly always attacks the first part, and though it may extend into the second portion, is very rare in the third part of the duodenum.—ED.]

MERCURIAL ULCERS.

I must refer the reader for the etiologic and anatomic details of this lesion to the section on Enteritis Crouposa (p. 211), and will confine my remarks here to one point—namely, that mercurial ulcers are found both in the small and in the large intestine, and usually produce a symptom-complex similar to that produced by dysentery.

[INTESTINAL MYIASIS.

Schlesinger and Weichselbaum[4] report a fatal case in which there was fecal impaction due to ulceration and atony in parts of the colon. The ulcers had undermined edges and occupied the whole circumference of the colon, and were regarded as due to maggots which bored their way into the intestinal wall and set up necrosis.—ED.]

[1] Mathieu and Roux, *Arch. gén. de Méd.*, 1902, p. 14.
[2] Mouisset, *Lyon Médical*, vol. xcv.
[3] Barié and P. Delaunay, *Soc. Méd. des Hop.*, January 16, 1903, p. 45.
[4] Schlesinger and Weichselbaum, *Wien. klin. Wochenschr.*, January 9, 1901.

SYMPTOMATOLOGY OF INTESTINAL ULCERATION.

Ulcers of the intestine do not produce a uniform clinical picture. The syndrome varies according to the etiology of the ulcers, and still more according to their seat and extent.

The symptomatology of ulceration of the duodenum (ulcus duodeni simplex, ulcer from burn) has already been described. The symptom-complex produced by typhoid, dysenteric, and diphtheric ulcerations will be discussed in the sections on these different diseases. The clinical picture of all the other forms is represented by the syndrome produced by catarrhal, follicular, and tuberculous ulcerations of the intestine.

It is an important clinical rule to remember that ulcers of the intestine frequently produce no symptoms whatever and may remain quite latent. In any given case of phthisis, therefore, we are never justified in excluding the existence of ulceration of the intestine from the absence of all symptoms pointing directly to this lesion. It will be seen in the description of the positive symptoms that may be produced by ulceration of the intestine why these lesions frequently remain latent. The positive symptoms produced are particularly important because, of course, only those ulcers which can be directly seen and diagnosed by inspection are situated in the lowest portion of the rectum. The most important symptoms are the following :

Diarrhea—that is, frequent and at the same time watery evacuations of the bowels. The view is generally prevalent that ulcerative destruction of the intestinal wall must necessarily produce increased peristalsis and diarrhea. This opinion is based on the supposition that the nerves of the intestinal wall, being deprived of their protective sheaths, are frequently directly exposed to contact with the intestinal contents. Some authors believe that the seat of the ulcer is immaterial, and that general peristaltic contraction of the intestine may be started by irritation in any portion of the bowel (Traube, Cohnheim). Another factor that is believed to give rise to diarrhea, at least in very extensive ulceration of the large intestine, is impairment of the absorptive power of the altered intestinal wall. In this way the bowel contents are still excessively watery. These two factors, as a matter of fact, unquestionably explain the occurrence of diarrhea in the dysenteric form of ulceration of the large intestine and in the follicular form which is primary in character and frequently involves very large areas of the bowel. The second factor mentioned—namely, interference with the absorption of water—is without doubt a prime factor in the production of diarrhea in all forms of intestinal ulcer. The first factor spoken of, namely, irritation of the exposed sensory nerves at the base of the ulcer, can, however, hardly be considered a constant cause of diarrhea in all forms of ulcer.

Clinical experience shows that intestinal ulcers can produce diarrhea, but do not necessarily do so in every case. Ulcers of the intestine are often present without the slightest diarrhea—in fact, the bowels may act daily with great regularity or there may be a slight degree of constipa-

tion, so that the bowels are evacuated only once every few days. Again in other cases constipation and diarrhea alternate. As early as 1847 Virchow wrote: "Neither in typhoid nor in tuberculosis has the number or size of the tumors any demonstrable direct connection with the diarrhea." The interesting question arises, What are the factors which determine the presence or absence of diarrhea in ulceration? The number of individual ulcers *per se* certainly cannot be regarded as responsible for the diarrhea; neither does the rapidity of the development of the individual ulcers exercise any marked effect in this respect. The situation of the ulcer, however, is of paramount importance. When ulceration occurs in the small intestine and is limited to this portion of the bowel, the bowels may act in a perfectly normal manner whatever the cause of the ulcers. The same applies to ulceration of the cecum and of the first portion of the ascending colon. This peculiar fact is readily explained. As long as the greater portion of the large intestine is healthy, the absorption of water proceeds in a normal manner. In this way the second factor that has been spoken of above is not brought into play. The old idea that irritation of any portion of the intestine may produce general peristalsis of the whole intestine is erroneous. Even irritants affecting the large intestine do not necessarily produce an evacuation of the bowels (with regard to this question see the discussion beginning on pages 70, 121, 171). It is well known, for instance, that ulceration of the intestine may be present in typhoid fever without producing diarrhea, and, conversely, cases of typhoid with severe diarrhea in which the autopsy shows only a few isolated small ulcers are commonly met with. Cohnheim, in his day, called attention to the fact that in typhoid fever the specific typhoid poison and not the ulcers must be considered responsible for the occurrence of diarrhea. Cases of tuberculous, follicular, and other forms of ulceration of the cecum and the first portion of the colon in which constipation exists during life are common. Ulcers may even be found in the lower part of the colon without increasing peristalsis as long as there are only a few. Still more striking conditions may occur: Kortum, for instance, reported the case of a tuberculous woman who suffered from diarrhea for ten weeks before she was admitted to the hospital. After admission to the wards this diarrhea continued for nine days more. After this, for five weeks, until the death of the patient, the bowels acted, with a few exceptions, only once a day, the stools, it is true, being somewhat watery. At the autopsy the whole of the large intestine as far as the anus was found covered with innumerable ulcers. Some of these, particularly in the colon, extended over wide areas, so that in large portions of the bowel only small islands of mucous lining were left. There is only one explanation for an occurrence of this kind—namely, that the ulcerative process of the mucosa had completely destroyed the nerves situated in the ulcerated areas. It is still more probable, of course, that the nerves were not anatomically destroyed, but that they became inactive owing to the constant irritation; in other words, that the repeated stimulation of the nerve-endings rendered them

insensitive to the ordinary irritants which would normally have produced peristaltic movements.

We see, therefore, that the presence or absence of diarrhea in ulceration of the intestine is primarily determined by the site of the lesions. If the ulcers are situated in the small intestine, cecum, or ascending colon, they probably never produce diarrhea unless the disease is complicated by certain other conditions, as catarrh of the intestine, amyloid disease, special forms of infection, or other factors that can exercise a direct influence on the peristaltic movements of the bowel. If the ulcers are situated in the lower portions of the colon and the rectum, diarrhea occurs with greater regularity; but even when the ulcers are in this part of the bowel, diarrhea may be absent under the following conditions : In the first place, when the number of ulcers are very small; in the second place, if the nervous irritability of these portions of the bowel is for some reason or another lowered or inhibited.

While diarrhea is considered a fairly important symptom of ulceration, much more importance is usually attached to the passage of blood, pus, shreds of tissue, and of peculiarly shaped particles of mucus in the stools.

Blood may appear in the dejecta in various forms of ulceration. In some cases pure blood may be passed by the bowel. This occurs, for instance, in simple duodenal ulcer, in ulcer of the intestine from burns, and in typhoid ulceration. In many other cases quantities of blood more or less large are passed either with fecal matter or with mucopurulent masses. The blood may either be intimately mixed with the other constituents of the stools, so that the color of the whole mass is modified by the coloring-matter of the blood, or it may not be intimately mixed with the feces, but be present in a separate portion of the evacuated material. All these variations depend largely on the source of the hemorrhage; the same applies to the changes that the blood-pigment undergoes (compare the section on Intestinal Hemorrhage).

Finally, there are other cases in which mere traces of blood derived from an ulcer are mixed with the intestinal contents. The quantity of blood may be so small, however, that it is not visible to the naked eye in the stools. Cases of this kind are much more frequent than is usually supposed. I have on many occasions succeeded in discovering very small collections of red blood-corpuscles on microscopic examination of the feces, even in cases where simple inspection of the feces would hardly have led me to suspect the presence of blood, and in many of the cases in which red blood-cells were found microscopically, autopsy subsequently showed the presence of ulcers in the intestine. In rare instances nothing may be discoverable in the dejecta but crystals of hematin, and these may be the only sign of a small hemorrhage high up in the intestine.

The frequency with which hemorrhage of the intestine occurs is another point for consideration. Different forms of ulcer vary in this respect. Simple duodenal ulcers occupy an exceptional position; from analysis of the features of typhoid and dysenteric ulcers, on the one

hand, and catarrhal and tuberculous ulcers, on the other (only to mention the more frequent types of ulcer), marked differences are apparent. In the former class large hemorrhages are very much more frequent than in the latter. It is possible, of course, that traces of blood that can be recognized only microscopically may often be passed in the stools in cases of tuberculous and catarrhal ulceration. This question requires more extended investigation. The difference in the frequency of hemorrhage does certainly not depend on the situation of the ulcers nor on the histologic form of the lesions, for both tuberculous and typhoid ulcerations originate in the follicular apparatus. It is possible that the rapidity of the development of the ulcers plays some part, for we know that typhoid and dysenteric ulcers develop more rapidly than tuberculous ulcers and the ulcers of chronic catarrh. It is also possible that the typhoid and dysenteric process *per se* in some way predisposes to hemorrhage.

The diagnostic value of the passage of stools either consisting of pure blood or containing a large quantity of blood may be summed up as follows : When blood appears in the stools under conditions that make the presence of ulcers possible, the diagnosis of ulcer is very probable. We should never draw more definite conclusions than this from the appearance of blood in the stools, for this symptom does not establish the diagnosis of ulcer by any means even in cases in which the existence of ulcers is etiologically probable, for we know that even when ulcers are present, the blood that appears in the stools may still be derived from other sources (compare the section on Intestinal Hemorrhage).

Conversely, I must expressly emphasize the fact that extensive ulceration of the intestine may be present without any blood in the feces. This is a fact that should be carefully remembered, as it is a matter of daily experience. The absence of blood, therefore, in the stools never militates against the diagnosis of ulceration of the intestine.

The appearance of pus in the dejecta is much more important than the appearance of blood. In order to understand the true diagnostic significance of the passage of pus in the feces it must be remembered that the material evacuated in catarrh of the intestinal mucosa is not really pus—that is, does not consist of closely congregated masses of round-cells. This is in contradistinction to the truly purulent or mucopurulent secretion passed in catarrh of the bladder or expectorated in catarrh of the bronchial mucosa. It is important to remember this in diagnosing ulcer of the intestine, for in this lesion true pus is found in the stools, and the appearance of purulent material must be considered one of the most positive signs of ulceration of the intestine. This includes, of course, not only all the ulcers of the intestine properly so called,—*i. e.*, those described above,—but also ulceration of new growth, and extra-intestinal abscesses that have perforated into the lumen of the intestine.

The quantity of pus evacuated in genuine ulceration of the intestine is usually small, and it is frequently necessary to examine the stool very carefully before any pus is found. Larger quantities of pus are

evacuated only in cases of abscess opening into the intestinal lumen, in suppurative carcinoma, and in croupous, "diphtheric" inflammation of the intestine, particularly in dysentery (compare p. 215). In all other forms of ulceration of the intestine very small quantities of pus are found, frequently in the form of minute grayish-white lumps which appear under the microscope as closely congregated masses of pus-cells.

In many instances, even though genuine ulceration of the intestine exists, pus may be completely absent from the stools. There are two possible explanations for this phenomenon—viz., either no pus whatever is formed, as, for instance, in duodenal ulcer, or the minute quantity produced on the surface of some small ulcer disappears. This is particularly apt to occur when the ulcers are situated high up in the intestine, for the distance this minute quantity of pus must travel being very long, the pus-cells may either become so intimately mixed with large masses of fecal matter that they cannot be discovered, or they may actually be destroyed—*i. e.*, digested by the intestinal secretions. If the presence of ulcers of the intestine is suspected and small specks or lumps of material are discovered in the stools, which on microscopic examination are found to be pus, the diagnosis of ulcer may be considered to be practically established.

Certain conclusions in regard to the nature of the process can also be drawn from the relative amounts of pus, blood, and mucus that are present in the stools. In uncomplicated "diphtheria" of the bowel pus alone is found, and the same applies to abscesses perforating into the intestinal wall. Approximately equal amounts of pus, blood, and mucus are evacuated only in dysentery and ulcerating carcinoma when the morbid processes are located in the rectum or in the lower part of the colon. The blood, pus, and mucus may be passed either alone or with feces. When the bowel-contents present this peculiarity, it may usually be assumed, with reasonable certainty, that one of these two conditions is present. The differential diagnosis between the two is, therefore, usually a comparatively easy matter.

Shreds of tissue passed with the feces are very important from a diagnostic point of view. Unfortunately, they are only rarely evacuated. It is essential and usually easy to differentiate the various membranous and shred-like particles derived from the food—for instance, elastic fibers, etc.—from the shreds of the intestinal wall now under consideration. Shreds of tissue—that is, membranous masses derived from the disintegrating tissues of the intestinal wall—are never found in slowly developing forms of intestinal ulcer, as the catarrhal, the tuberculous, and the amyloid form, nor in those forms which develop rapidly but remain small—as, for instance, typhoid ulcers. Shreds are most frequently passed in the stools in the dysenteric form of ulcer of the intestine. In this disease they may appear for many days in succession.

The examination of the feces for tubercle bacilli is an important laboratory aid to the diagnosis of tuberculous ulcers. Lichtheim was the first to call attention to the significance of tubercle bacilli in the feces. Giacomi also called attention to the occasional presence of

tubercle bacilli in the stools and to the significance of this observation in the diagnosis of tuberculous ulceration of the bowel. Boelo examined fecal matter from three cases of wide-spread intestinal tuberculosis, but failed to find any tubercle bacilli. On the other hand, he examined the fecal matter in three phthisical cases without lesions of the intestine and succeeded in discovering tubercle bacilli. In three other cases of intestinal tuberculosis the examination was also positive. From these data he argues that we can never draw any conclusions as to the presence or absence of intestinal tuberculosis from the presence or absence of tubercle bacilli in the stools. Further systematic researches in late years (*e. g.*, Biermann's) all agree that the presence of the bacillus does not justify a dogmatic conclusion, and that its absence does not exclude intestinal tuberculosis. Every observer will probably substantiate this.

Occasionally peculiar lumps of mucus are found in the stools which possess great significance in the diagnosis of ulcers of the intestine. These have been described as " small lumps of mucus resembling frogs' eggs or boiled grains of sago." I need hardly emphasize the fact that mucus *per se* in the stool has nothing whatever to do with ulceration. Formerly it was believed that the little round lumps of mucus just spoken of could be considered a positive sign of follicular ulceration, but this is by no means the case. Ulcerating surfaces, in the first place, never secrete mucus, but only pus. Heubner argued that the only mucus that could be cast off from the surface of a follicular ulcer would have to enter the ulcerated area from without. Kelsch also called attention to the fact that in exceptional cases one of the glands of Lieberkühn might become involved in such an ulcerative process.

Virchow many years ago demonstrated that these peculiar structures, resembling boiled grains of sago, are, as a matter of fact, almost always of vegetable origin. Woodward and I have arrived at the same conclusion (compare p. 88). I examined a great many dejecta containing these little balls of mucus, and found, almost without exception, that they were of vegetable origin and showed vegetable structure—in other words, that they consisted either of starch or of small pieces of fruit, etc. I am definitely of the opinion that these round lumps of mucus do not possess the diagnostic significance in ulcer of the intestine that they are traditionally credited with. Kitagawa agrees with my view that the structures resembling grains of sago are, as a matter of fact, frequently mucous in character, and arrives at the same conclusions as to the clinical significance of these bodies in the stools.

The occurrence of pain is also of little value in the diagnosis of ulcers of the intestine. In many cases pain is completely absent both in the acute and in the more chronic forms of ulcer. The same applies to a certain extent, though not so universally, to ulcers of the stomach. While the absence of pain may be surprising in gastric ulcers, it is not astonishing that pain is absent in intestinal ulcers, for they are not irritated by the bowel contents in the same way that gastric ulcers are irritated by the stomach-contents, for the latter are acid and possess more

marked chemically irritating properties. In the stomach, moreover, peculiar conditions obtain, for at one time the stomach is empty, at another it is full, so that the irritation of the nerves is not continuous. It can be imagined, from a physiologic point of view, that interrupted irritation of exposed nerve-endings would produce more pain than continuous irritation. The former condition would be present in the stomach and consequently cause pain ; the latter, in the lowest portions of the ileum, the cecum, the colon, and the rectum (the most frequent situations of ulcer), and would probably not lead to such severe paroxysms of pain.

When pain does occur in ulcer of the intestine, the character of the paroxysms may vary according to the origin of this pain. In the first place, pain may really be produced by irritation of the nerves at the base of the ulcer. In cases of this kind the pain is apparently spontaneous and limited to a small area of the bowel—namely, the site of the ulcer ; this pain, moreover, is not very intense ; occasionally, however, it assumes a colicky character and extends over wide areas of the intestine (compare the paragraph on intestinal pain). In other instances the pain is elicited only by external pressure on the abdomen. In this case pain may, of course, also be considered due to direct irritation of the ulcer, but in the great majority of these cases it is due to the mechanical irritation of areas of circumscribed peritonitis, which is a very common complication in chronic ulceration. Both the spontaneous pain and the tenderness on pressure are, as a rule, due to involvement of the serosa, although this is not the only source of the pain.

We see, therefore, that the symptom " pain " can be utilized only with great care in the diagnosis of ulceration of the intestine. In many instances it may be completely absent; in others, it may be present, but of doubtful significance. The symptom is of most use when the pain is always present in the same location, and can be made worse or produced by external pressure over these areas. The presence of a constant pain on pressure, which is strictly circumscribed, may be of use in the differential diagnosis between catarrh of the intestine and ulceration in so far that, *ceteris paribus,* it is in favor of ulceration.

I have already described the peculiar pain that is experienced in ulcer of the duodenum. Ulcers of the lowest portions of the rectum also produce a peculiar form of pain that is characterized as tenesmus.

Ulcers of the intestine *per se* probably never produce fever ; if a rise of temperature is observed in the course of ulceration of the intestine, this rise is usually due to the primary etiologic process.

The general nutrition is not disturbed by the presence of a few small ulcers in the intestine. Wide-spread and severe destruction of tissue, however, such as is occasionally seen in the follicular and the tuberculous forms of intestinal ulcer, may lead to great emaciation. This result is brought about by the rapid propulsion of the intestinal contents and the interference with normal absorption which is the natural result of the destruction of such large portions of the intestinal mucosa.

The involvement of the small intestine is much more important in this respect than involvement of the large intestine.

In the paragraphs on the anatomy of ulcer of the intestine I have already dealt with the other possible sequelæ of this lesion, such as stenosis and perforation of the bowel and the formation of peritoneal adhesions, etc.

The following is a brief summary of what has been said in the preceding paragraphs with regard to the diagnosis of ulceration of the intestine :

Ulcers of the intestine may produce no symptoms whatever ; even when large numbers of ulcers are present and extend over a wide area of the intestine, the symptoms produced are in many instances altogether out of proportion to the intensity of the structural changes present. The only reliable sign of intestinal ulceration, if we can exclude perforation of an abscess into the intestinal lumen, is the appearance of pus and of shreds of intestinal tissue in the stools. Another very important sign, which, however, must be utilized with great care, is the passage of blood in the stools. No definite conclusions can be drawn from the watery consistence of the stools and the increased frequency of defecation. The discovery of circumscribed fixed points in the abdomen that are painful to pressure may, under certain circumstances, aid in the diagnosis. When perforative peritonitis occurs, the diagnosis under given conditions may be considered established.

I shall not enter into a discussion of the different signs that can be utilized in determining the etiologic character of the ulcers and their pathogenesis in each individual case, as I have already mentioned most of these factors in discussing the different forms of ulcer of the intestine. The most important thing to do, of course, is to consider carefully the symptoms as a whole. Even if this is done, it may be very difficult or even impossible to arrive at a diagnosis in any given case. In the first place many accidental complications may obscure the general aspect of the disease ; in the second place it may be difficult—for instance, in a tuberculous case in which bacilli are not found in the dejecta—to decide whether the hypothetic ulceration of the intestine is tuberculous or amyloid.

PROGNOSIS.

A number of important points in regard to the prognosis of ulceration of the intestine have been dealt with in the paragraphs on the etiology and the anatomy of this disease, and it would be quite superfluous to enter into all these details again. There is no such thing as a universal prognosis in all forms of ulceration of the intestine. The prognosis will be different in each individual case, but will be dependent essentially on two factors—namely, the primary etiology of the ulcers and the extent of the ulcerative process. The seat of the ulcers is of small significance in the prognosis. Typhoid ulceration offers a better prognosis than amyloid ulceration. From three to five small catarrhal ulcers are comparatively free from danger. When large areas of the

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intestinal mucosa are destroyed by the same form of ulcer, the life of the patient is seriously endangered. Ulcers at the lower end of the rectum, which are visible and accessible to direct local treatment, offer a better prognosis than those situated in other portions of the bowel; but it makes very little difference from the point of view of prognosis whether they are situated in the small intestine or in the colon. Some forms of ulcers, such as the amyloid variety, show no tendency whatever to heal; others, like the tuberculous and the embolic-thrombotic form, show only a slight inclination to heal, while others, such as the typhoid and dysenteric forms, usually undergo rapid cicatrization, the latter even when the ulcerative process involves large areas.

TREATMENT.

For some forms of ulcer of the intestine there is no special therapy, as the treatment of the ulcer coincides with the treatment of the primary disease that produces it. This applies to ulceration occurring in acute infectious diseases, in constitutional diseases, and in certain intoxications. Duodenal ulcers should be treated like round ulcer of the stomach. The treatment of dysenteric ulceration of the intestine will be discussed in a special section. Syphilitic ulceration should, of course, be treated by specific antisyphilitic methods. Practically speaking, therefore, we are confronted by the question in this section, how to treat catarrhal and tuberculous ulcers of the intestine. The treatment of all other forms, apart from those mentioned above, need hardly be discussed, for embolic-thrombotic ulcers are exceedingly rare; stercoral ulcers, unless they are situated in the rectum, can rarely be diagnosed, or are diagnosed only when it is too late—that is, after perforation of the bowel has occurred or stenosis has developed. The treatment of catarrhal and tuberculous ulcers is divided into two parts: In the first place, symptomatic treatment—namely, the treatment of diarrhea, hemorrhage, pain; in the second place, general treatment directed against the ulcerative process itself.

For the symptomatic treatment the reader should refer to the sections on Diarrhea, Intestinal Hemorrhage, Intestinal Pain, and Chronic Peritonitis. Cases of this character should remain in bed. The application of warm compresses (dry or moist) to the abdomen is, as a rule, grateful. In exceptional cases, if there is much peritonitic irritation, the patients prefer a cold compress. Lukewarm and warm baths are also a useful adjuvant to the treatment.

The regulation of the diet is of extreme importance. Careful supervision of the diet meets one of the primary indications in the treatment of ulcerations of the digestive canal—namely, prevents interference with the natural process of healing by stopping the ingestion of irritating or otherwise harmful articles of food. The diet should, in general, be regulated according to the same principles as have been enunciated in the description of the treatment of chronic catarrh of the intestine with diarrhea. The diet should, in the first place, be non-

irritating, both chemically and mechanically, and at the same time, of course, should be as nourishing as possible. It is well to remember that a diet which fulfils all these conditions, and is, so far as possible, liquid, meets all the chief indications.

The direct treatment of ulcers, or rather an attempt to treat ulcers directly, can be undertaken only when they are in the rectum, the lower portions of the colon, or possibly in the upper portion of the large intestine. Since ulcers above the ileocecal valve cannot be reached by rectal irrigation, the treatment of ulcers in the small intestine must be carried on by medication *per os*.

Unfortunately, however, we do not possess any internal remedies given by the mouth which can cure an intestinal ulcer or even influence it favorably in any way. Louis was in the habit of giving lead acetate with opium in cases of phthisis with severe diarrhea due to intestinal tuberculosis. It is possible that he succeeded in a few instances in arresting the diarrhea, but he certainly never cured the ulcers. It is even doubtful whether the preparation of lead which he administered can be used for any length of time without interfering with general digestion. Alum, preparations containing tannic acid, and so-called styptic preparations of iron are all worthless in this respect. More benefit may perhaps be expected from subnitrate and salicylate of bismuth, given in doses of 15 grains three, five, or eight times a day with or without the addition of opium; for, in the first place, bismuth has a certain constipating power which is probably chiefly due to its property of decomposing the sulphureted hydrogen that is present in the intestine (Boaki); in addition, some authors (Traube) assume that bismuth powder forms a protective covering over the surface of the ulcer and in this way favors its healing. It is difficult to imagine, however, how a small amount of bismuth powder administered by the mouth can become deposited on a few isolated ulcers scattered over the course of the long intestinal tract, particularly if they are situated in the lower portion of the ileum or in the transverse colon. On the other hand, when numerous ulcers are present, the small quantity of powder swallowed cannot possibly be enough to cover them all. Nevertheless, bismuth is a harmless remedy and to a certain extent acts as a styptic; again, there is no better remedy, so that no harm can be done by employing it in ulceration of the intestine, provided no delusions are cherished as regards its value in the treatment of this disease. Tannigen, tannalbin, and tannocol (*cf.* Enteritis Chronica) do no more, or even less, than bismuth.

[Sublimed sulphur, given in doses of 20 grains, with 5 grains of Dover's powder several times a day, which had a marked effect in curing dysentery in the South African War, has been employed with benefit in tuberculous enteritis,[1] but requires further trial.—Ed.]

Ulcers in the lower part of the colon and the rectum are under much more favorable conditions for treatment, as they are to a certain degree amenable to direct local treatment. Consideration must be

[1] G. E. Richmond, *Lancet*, 1901, vol. ii., p. 1408.

chiefly limited to the treatment of catarrhal, stercoral, and tuberculous ulceration of this part of the bowel ; the syphilitic and dysenteric forms will be considered in a special section. In addition to attacking these ulcers by the general means already detailed, they can be treated by irrigation with disinfecting and astringent lotions. Of the disinfecting solutions that can be employed, the following are the most useful : Thymol, 1 : 2000 or 1000 ; salicylic acid, 1 : 300 ; boric acid, 1 : 500. An express warning is necessary against irrigations with carbolic acid or solutions of corrosive sublimate 'because there is always danger of producing general poisoning by their use. The best so-called astringent lotions that can be used are a 0.2 to 1 per cent. solution of silver nitrate and solutions of tannic acid of the same strength.

Ulcers of the large intestine which can be directly seen should be treated according to the well-known rules for the treatment of ordinary ulceration.

AMYLOIDOSIS OF THE INTESTINE (Degeneratio amyloidea intestini).

Amyloid degeneration of the intestine was originally described by Virchow in 1855.

The intestine is one of the organs most frequently involved in amyloidosis, and thus resembles the kidneys, the liver, and the spleen. [In 468 cases of lardaceous[1] disease obtained by combining the statistics given by H. P. Loomis,[2] Dickinson,[3] and Goodhart,[4] the kidneys were affected in 302, the spleen in 273, the liver in 201, and the intestines in 163 instances.—ED.]

There is no occasion to discuss the pathogenesis and the etiology of this form of intestinal disease here. Detailed description of these points will therefore be omitted, and it will be sufficient to point out that amyloid degeneration of the intestine, like amyloidosis of other organs, is always a secondary phenomenon to a number of well-known primary diseases, among which the following may be mentioned : tuberculosis, syphilis, suppuration in connection with bones, malarial and other forms of cachexia ; these diseases are the most frequent causes of amyloid degeneration ; in addition, however, a variety of processes which lead to loss of tissue fluids and produce great exhaustion must be mentioned, such as suppuration of all kinds, bronchiectasis with profuse bronchorrhea, abscess of the lung, chronic diarrhea, exhausting forms of gastric ulcer, etc. ; furthermore, the following conditions may produce amyloidosis—leukemia, gout, and chronic parenchymatous nephritis. In exceptional cases it has been impossible to discover any primary disease to account for the occurrence of amyloid degeneration

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[1] The term lardaceous is widely used in England, and is indeed recommended in the nomenclature of diseases issued by the Royal College of Physicians of London.

[2] H. P. Loomis, in *System of Practical Medicine* (Loomis and Thompson).

[3] W. H. Dickinson, *Allbutt's System of Med.*, vol. iii., p. 264.

[4] Goodhart, quoted in Hilton Fagge and Pye-Smith's *Text-book of Medicine*, vol. ii., fourth ed., p. 645.

of the intestine. Again, in other cases the intestine was primarily diseased and became affected with amyloidosis before any evidence of the affection could be discovered in the liver or the kidneys; instances of the latter kind must, however, be considered rare.

Anatomy.—Amyloid degeneration may involve the whole intestine (occasionally the whole digestive tract throughout from the tonsils to the rectum may be affected). When only part of the intestine is diseased, the small intestine, especially the ileum, is the part affected; in exceptional cases, however, the small intestine escapes and the amyloid degeneration is limited to the colon.

Virchow gave an accurate description of the appearance of the intestine when affected with amyloid degeneration; he speaks of an anatomic, pale-grayish, translucent, somewhat swollen appearance, and also mentions the fact that the amyloid intestine gives certain characteristic color reactions—viz., a brownish-red color with iodin, turning blue or violet when treated with sulphuric acid, and a brilliant rose-red color with methyl-violet. In advanced amyloid degeneration the villi of the intestine are very frequently absent: they are either atrophied or sloughed off. The formation of ulcers in an amyloid intestine is referred to elsewhere.

Since the days of Virchow, Friedreich, Neumann, and Hayem, the histologic changes characteristic of amyloid degeneration of the intestine have been made the subject of most exhaustive study—special mention should be made of Kyber's extensive and thorough investigations. All the essential points of the histology may be considered fully worked out, with the possible exception of a few minor and insignificant details.

Amyloid degeneration of the intestine involves primarily and chiefly the blood-vessels of the bowel-wall; of these, the capillaries and the smallest arteries seem to be chiefly affected, although occasionally the veins are also involved in the process. The first vessels to be affected are the vessels of the intestinal mucous membrane; in many instances the vessels of the submucosa are also affected, and in some cases the vessels throughout the whole thickness of the intestinal wall, including serosa and subserosa, are affected. Occasionally, as Friedreich long ago pointed out, the degenerative process may be found in the vessels of the mesentery, and may, in fact, be very pronounced there. In the smaller arteries the amyloid degeneration chiefly affects the muscular coat; occasionally the whole wall of the blood-vessels is in a state of amyloid degeneration, but in all these cases the amyloid process always begins in the media. In many instances the blood-vessels alone are diseased, but frequently the muscularis mucosæ (Bruecke's muscle) is affected in addition. The muscular layers of the intestinal wall proper —*i. e.*, the longitudinal and the circular fibers—may be involved in the process of amyloid degeneration, and the change need not be confined to the blood-vessels which run in these muscle-fibers. The follicles and Peyer's patches usually remain free from amyloid degeneration even in those cases in which the bowel-wall is extensively involved; it is only in exceptional cases that the parenchyma of these structures is

affected. The tissues proper of the mucous layer and the glands of the intestine are also rarely degenerated and seldom give an amyloid reaction, so that in many cases the progress of the amyloid process can be followed up to the boundaries of the glandular epithelium, but does not involve the latter. The epithelial cells forming the surface layer of the mucosa are also rarely involved in the degeneration; but in rare and isolated cases they may occasionally give the amyloid reaction. Hayem distinguishes a distinct form of amyloid degeneration of the intestine occurring in children, in which the follicles are alone or almost exclusively involved in the degeneration; whereas in the ordinary form the blood-vessels are primarily or exclusively affected.

It is unnecessary to give a detailed description of the microscopic appearances presented by the vessels of the bowel-wall when in a state of amyloid degeneration. In order to make it easier to understand the clinical features of the disease I need only point out here that the chief change consists in a thickening and stiffening of the affected blood-vessels. As a result of this rigidity the vascular supply of the parts is interfered with, and the physiologic variations in the amount of blood passing through the various layers of the bowel are interfered with or entirely brought to a standstill.

The occurrence and mode of origin of amyloid ulcers have been described in a previous section (see p. 251); I will merely add here that Kyber does not recognize any connection between amyloid degeneration of the intestinal blood-vessels and ulceration; he maintains, moreover, that the ulcers found in cases of amyloid disease of the intestine are simple and of catarrhal origin.

Clinical Features.—Amyloidosis of the intestine when it is slight and involves only small portions of the intestine cannot be discovered and is not recognized clinically; advanced and extensive degrees of amyloidosis properly speaking produce only one symptom—viz., diarrhea. In cases of this character the diarrhea is very obstinate and persistent. The evacuations of the bowels at the same time vary in consistence and frequency. They are usually thin and liquid, but are otherwise not in any way remarkable and show no characteristic properties. The genesis of this form of evacuation is interpreted in different ways. Traube claims that the diarrhea is dependent on anemia of the intestinal wall, and tries to draw an analogy with Schiff's well-known experiment in which violent peristaltic movements of the intestine followed compression of the abdominal aorta; this interpretation is probably incorrect, for in Schiff's experiment the anemia of the intestinal wall is produced suddenly, whereas in amyloidosis the onset of the anemia is gradual. Cohnheim attributes the diarrhea to an excessive passage of fluid through the diseased capillaries. Other authors believe that the absorption of the bowel contents is interfered with owing to the diseased condition of the bowel-wall, and that the diarrhea is due to this cause, since the intestinal contents remain liquid as a result of deficient absorption of fluids from the bowel. It is difficult to prove which of these views is the correct one.

The ordinary view indorsed by Traube is that in simple amyloidosis of the intestine the motions are not bloody. To judge from personal experience I can substantiate this statement. Grainger Stewart, it is true, describes intestinal hemorrhages even without a loss of tissue, and attributes the appearance of blood in the motions to rupture of the blood-vessels of the intestinal wall, which in this disease are abnormally fragile. Lastly it is important to note that in amyloidosis of the intestine without complications there is neither spontaneous pain nor tenderness on pressure over the abdomen.

For an account of amyloid ulcers of the intestine the reader is referred to page 251.

The **diagnosis** of amyloidosis of the intestine is always difficult, and should in most cases be made very cautiously ; it must be considered in cases of obstinate diarrhea in which there is no blood in the stools, no spontaneous abdominal pain or tenderness, and in which, in addition to diarrhea, there are enlargement of the liver and spleen and albuminuria. But even in cases of this kind the diagnosis may be difficult and uncertain, since the same clinical picture may be simulated by some kinds of intestinal ulceration, such as tuberculous or catarrhal ulcers.

The **prognosis** is unfavorable, and cure of the disease seems to be impossible.

Treatment is limited to regulation of the diet (see also p. 274). Where the motions are very profuse, opium, bismuth subnitrate, bismuth salicylate, bismuth tannate, tannigen, tannalbin, etc., may be administered. The results of treatment are generally poor in this disease.

DISEASES OF THE BLOOD-VESSELS OF THE INTESTINE.

EMBOLISM AND THROMBOSIS OF THE MESENTERIC ARTERIES.

VIRCHOW was the first to recognize and to explain embolism and thrombosis, and our knowledge of these processes as they occur in the mesenteric arteries dates from the publication of his admirable investigations on this subject, as the first three cases of embolism and thrombosis of these vessels were recorded by him. The total number of cases reported in the literature of the last forty years does not exceed 30, and while unquestionably a certain number of cases have not been published, the affection must, nevertheless, be considered a rare one. [According to Welch,[1] at least 70 cases of embolism or thrombosis of the mesenteric arteries have been published. Gallavardin[2] has collected 83 cases. In Watson's series 8 occurred in one year at Boston.—ED.] The occurrence of thrombosis in the intestinal arteries is, however, of such clinical importance that it requires a detailed description.

The majority of cases (20) were published before 1875, and were summarized by Litten and Faber. [The reader should refer to Welch's

[1] Welch, *Allbutt's System of Med.*, vol. ii., 1899.
[2] Gallavardin, *Gaz. des Hôp.*, Paris, 1901, pp. 929 and 957.

article in *Allbutt's System of Medicine,* vol. iv., for a full and up-to-date account, or to Gallavardin's critical review.—ED.]

Etiology.—Occlusion of the mesenteric arteries in the great majority of cases is due to embolism and only rarely to the formation of an autochthonous thrombus. [In 83 cases of embolism or thrombosis of the mesenteric arteries collected by Gallavardin, 63 were cases of embolism.—ED.]

The source of the embolism is usually the heart, for in nearly all the cases endocarditis with valvular disease, or thrombosis without endocarditis, was found. In some instances the starting-point is atheromatous degeneration of the aorta, and in one case the embolus was detached from a clot in one of the pulmonary veins (the result of gangrenous hemorrhagic infarction of the lung (Virchow).

Until a few years ago embolic occlusion of the mesenteric arteries was the only form of occlusion recognized. Litten, however, has since then described 2 cases of occlusion of the arteries by thrombosis due to local changes in the vessels. On postmortem examination he found the cause of this local thrombosis to be a form of circumscribed ("gitterförmig," interlacing) endarteritis of the mesenteric artery, which will not be described in detail here. [Welch considers that there is room for suspicion that Litten mistook the lattice-work markings sometimes seen after detachment of an adherent thrombus for a special form of endarteritis. It does not appear that he observed this "latticed endarteritis" except after the removal of adherent thrombi.—ED.]

Firket and Malvoz also reported an example of spontaneous local thrombosis of the mesenteric artery, which, from their description, was probably the case, but cannot be regarded as settled beyond doubt; for in addition to atheroma of the mesenteric arteries there was wide-spread atheroma of the aorta and the endocardium.

[Syphilitic endarteritis of the mesenteric artery, in rare cases, has been thought to account for thrombosis. T. E. Gordon [1] has recorded a case in point. Injury may give rise to occlusion of the mesenteric artery (Marsh).[2] The free mobility of the mesentery probably prevents thrombosis being set up by the pressure of tumors, but thrombosis has been thought to be set up by the pressure of calcareous glands (Adenot).[3] —ED.]

Finally, it may be interesting, from the standpoint of pathology, to refer to the so-called verminous aneurysm of horses produced by *Strongylus armatus* (Bollinger, Csokor), which is met with in the main trunk of the colic artery. From the aneurysm emboli frequently enter the branches of the colic arteries and produce "attacks of colic," paralysis of the intestine, and intestinal occlusion. [In man a rare condition, periarteritis nodosa, in which multiple acute aneurysms occur, may involve the intestines and give rise to pain, colic, diarrhea, and ulceration.—ED.]

Anatomy.—Occlusion of the superior mesenteric artery is much

[1] T. E. Gordon, *Brit. Med. Jour.,* 1898, vol. ii., p. 1447.
[2] H. Marsh, *ibid.,* 1902, vol. i., p. 963.   [3] Adenot, *Rev. de Méd.,* 1890.

commoner than occlusion of the inferior mesenteric artery, which has been observed only in a few isolated cases.

There is complete occlusion of the trunk of the artery, blocking of some of its larger branches, or, lastly, of some of the smaller branches. In addition there is a form of obliteration of the smallest and terminal arteries embedded in the wall of the intestine. These may become occluded as a result either of embolism or of endarteritis obliterans, a disease which has been seen several times in association with multiple neuritis. When the minute ramifications of the colic arteries become occluded, embolic or thrombotic ulcers of the intestinal wall develop. As these lesions of the intestinal wall have been described above (see p. 248), the reader should refer for the details to these paragraphs.

The extent of the intestinal lesion following the occlusion of the blood-vessels supplying a given area of the intestine (and I refer here only to the superior mesenteric artery) varies according to the seat of the embolus. When the trunk of the vessel is occluded close to its origin from the aorta, its entire area of distribution is involved—that is, the whole small intestine (with the exception of the middle and upper horizontal portion of the duodenum), and in addition the cecum and the ascending and the transverse colon. Cases of this kind have been described by Oppolzer, Faber, and Kaufmann. When one of the main branches of the mesenteric artery becomes blocked, which is relatively more frequent, the lesions of the intestine may occur in various portions of the bowel corresponding to the exact area of distribution of the occluded vessel. The intestinal lesion invariably involves a continuous segment of the gut and is of considerable size, usually in the ileum and lower part of the jejunum; but any other situations may, of course, be imagined. Finally, when some of the smaller branches of the mesenteric artery are occluded, a number of different portions of the bowel may be affected by local interference with their vascular supply. In this instance the affected portions of the intestine are not necessarily continuous, but may be scattered irregularly over the bowel, and may be separated by healthy portions of intestine. In occlusion of these smaller branches, moreover, a number of emboli are usually found. In a case published by Firket and Malvozd, for instance, there were four distinct necrotic foci in the intestine. Embolism of the minute terminal branches of the mesenteric artery situated in the substance of the intestinal wall produce secondary changes in the intestine which are even more widely distributed; for example, the small multiple ulcers of the intestine described in a preceding section.

The parts of the mesentery and intestine supplied by the blocked vessel present the following appearances: there is venous hyperemia, with hemorrhage, edema, and necrosis; the arteries are contracted and empty, the veins of the serosa and of the mesentery full and distended to bursting, the mucosa dark red, and all the tissues succulent and swollen. Throughout the mesentery and the intestinal wall there are small, often innumerable, hemorrhages, and the lumen of the intestine contains extra-

vasated blood, either fresh or so old that it forms a tarry mass. In very severe cases the walls of the intestine and the mesentery are in a condition of hemorrhagic infiltration, which may be so extreme that a distinct mass can be felt inside the mesentery. When the morbid process has persisted for some time, necrosis of the intestine results, and fat-necrosis is seen in the mesentery. When the smaller branches of the mesenteric artery are occluded, the necrotic process involves only smaller portions of the intestine and of the mesentery ; when one of the main branches is blocked, large portions of the intestine and of the fat in the mesentery become necrotic. In the former instance localized ulceration occurs in different places ; while in the latter case the mucous membrane of the intestine becomes discolored and of a dirty brownish-green color in extensive and continuous areas of the bowel, so that it can be stripped off like mucus. The mesenteric glands are frequently unchanged, but in some cases are swollen and infiltrated with blood. Oppolzer and, recently, Deckart, in their studies on arterial embolism, have encountered a thrombosis (secondary) in the mesenteric veins which even led to emboli and hemorrhagic infarcts in the liver.

The serous covering of the bowel is not only changed in the affected area, but also in other parts of the peritoneal cavity. When death does not occur too soon after the occlusion of the intestinal artery, the serosa is inflamed over considerable portions of the intestine which are otherwise healthy, and as a result various loops of intestine become adherent, lose their sheen, and are covered with fibrin. In addition the peritoneal cavity contains blood-stained fluid or a purulent exudate. [Welch and Flexner,[1] in a case of hemorrhagic infarction of the jejunum, found gas in the peritoneal cavity due to the Bacillus aërogenes capsulatus. During life tympanites was thought to be due to perforation. Emphysema of the mucosa has also been observed in hemorrhagic infarction.—Ed.]

It is unnecessary to give a detailed description of the emboli and thrombi themselves. They may be either single or multiple, and differ in no respect from those occurring in other parts of the circulatory system, so that a detailed description would be superfluous. One point, however, is worth mentioning—namely, that in many of the cases of embolism of the intestinal vessels there is also hemorrhagic infarction of other organs, especially of the kidneys and spleen.

The anatomic appearances of the intestine after embolism of the mesenteric artery are practically those of a hemorrhagic infarct. The lesions secondary to occlusion of the mesenteric arteries, as we have seen, may occupy either large or small areas of the intestine, the extent of which entirely depends on the seat of the embolus or thrombus— in other words, whether the obstruction is in one of the large branches or in one of the fine terminal ramifications of the vessels in the intestinal wall itself. After Cohnheim had published his well-known theory on the genesis of hemorrhagic infarction it seemed remarkable that hemorrhagic infarction could occur in the area supplied by the mesen-

[1] Welch and Flexner, *Jour. Exper. Med.*, 1896.

teric artery, for this vessel is not an "anatomic end artery" in the sense adopted by Cohnheim; on the contrary, the colica sinistra artery arising from the inferior mesenteric artery forms the largest anastomosis in the body with the colica media artery coming from the superior mesenteric artery, and from above the superior mesenteric artery communicates with the celiac axis through the gastroduodenal branch of the hepatic artery.

In order to explain this anomaly Litten made a series of experiments, and arrived at the conclusion that the superior mesenteric artery, although not an end artery in the anatomic sense, is nevertheless practically an end artery in the "functional" sense, a conclusion in which Faber agreed. Anatomically it is true that all the branches of the mesenteric artery, even the smallest ramifications, can be injected with wax from the aorta through their numerous anastomoses, even after ligation of the main trunk of the artery. In living subjects and in experiments on living animals, however, this does not hold good. The area supplied by the mesenteric artery is so extensive that when the artery is suddenly occluded, the pressure falls to zero. The small quantity of blood which can pass into the occluded vessel and its branches from the comparatively insignificant inferior mesenteric and pancreaticoduodenal arteries is insufficient to fill the collapsed vessels. The blood, however, from the portal area can regurgitate backward into these vessels because the blood in this area is under positive pressure, even though this pressure is reduced. In this way venous hyperemia, infarction of the tissues supplied by the mesenteric artery and its branches, and, finally, if all arterial blood is excluded, necrosis supervene.

[According to Welch and Mall,[1] the blood which produces the hemorrhagic infarction is derived from anastomosing arteries, and not by reflux from the veins. Directly after experimental occlusion of the main trunk of the superior mesenteric artery in dogs violent tonic contraction of the intestine sets in; the bowel remains contracted and anemic for from two to three hours, the circulation ceasing; the bowel then relaxes, venous hyperemia occurs, and hemorrhagic infarction follows in from three to six hours after occlusion of the artery.—ED.]

Occlusion of the inferior mesenteric artery is extraordinarily rare. Litten succeeded in collecting only 2 recorded cases, 1 of which (von Gerhardt's case) was associated with occlusion of the superior mesenteric artery. [In 83 cases of embolism and thrombosis of the mesenteric arteries, the inferior mesenteric artery was obstructed in 5 instances—on 4 occasions by emboli, and in 1 case by thrombosis; in 2 of the 5 cases the superior mesenteric artery was also occluded. Cases of thrombosis of the inferior mesenteric artery, with a similar change in the superior mesenteric artery, have also been met with by Hale White[2] and by Monro.[3]—ED.]

In the only uncomplicated case, reported by von Hegar, the mucous

. [1] Mall, *Johns Hopkins Hosp. Repts.*, vol. i.
[2] W. Hale White, *Allbutt's System of Med.*, vol. iii., p. 961.
[3] Monro, *Lancet*, 1894, vol. i., p. 147.

membrane of the descending colon (Hegar writes "ascending"), of the sigmoid flexure, and of the rectum was somewhat loosened and detached, very red, and covered with some small and some large hemorrhages. The bowel was not, properly speaking, necrotic, nor was the condition one that could be called hemorrhagic infarction. This condition corresponds with the experimental results obtained by Litten, for he found that the inferior mesenteric artery is in no sense to be regarded as an end artery, either anatomically or functionally. [In Monro's case of thrombosis of the inferior mesenteric artery there was infarction of the sigmoid mesocolon.—ED.] Yet in a few other cases (Adénot, Elliott) gangrenous ulceration and perforations were found in the colon.

Clinical Features.—In view of the small number of cases of embolism of the superior mesenteric artery on record it is impossible to give an exhaustive and complete clinical account of this lesion ; this task is rendered still more difficult by the fact that a great number of the described cases were discovered postmortem and were not observed during life. Nevertheless a few definite points can be gleaned even from these scanty data : Kussmaul and Gerhardt, in fact, many years ago gave a very clear and succinct account of this lesion and the clinical picture it produces. It is surprising to note that the symptoms differ greatly in different cases and are by no means constant ; careful analysis, however, of all the available reports explains this apparent discrepancy. Two distinct clinical types must be recognized in embolism of the superior mesenteric artery—viz., one characterized by hemorrhages from the intestine, which is the more frequent of the two ; the other by the appearance of symptoms which simulate intestinal obstruction with or without signs of peritonitis.

The onset of the disease is frequently sudden or at least rapidly progressive in character, so that the symptoms, which may be slight at first, soon become grave and severe. This also applies to those cases which are not really embolic, but are due to spontaneous local thrombosis of the artery ; Litten, for instance, reports a case of the latter category in a young man in whom the disease began suddenly and unexpectedly with intestinal hemorrhage. Two interesting observations by Schnitzler and Lépine have shown another clinical appearance, which may be important in the diagnosis of autochthonous thrombosis (see below).

In other instances the initial symptoms of the disease are obscured, and the onset of the disease is not so sudden nor so acute and may occasionally escape detection altogether ; this is particularly liable to occur when the disease is not ushered in with intestinal hemorrhage, or when embolism of the superior mesentery artery occurs as a complication of some other grave condition.

In several recorded cases the onset of the disease was characterized by colicky pains which either radiated all over the abdomen or were localized in the lower portions of the abdomen ; while in other cases the pain was confined to a spot above the umbilicus or to one side of the abdomen. [This early colic can be explained as due to the tonic contractions described by Welch and Mall.—ED.] The situation of this

pain probably corresponds to the portion of the bowel in which the acute disturbance of the circulation of the blood supervenes. If the patient survives sufficiently long, the character of the pain may change, and may become continuous and be spontaneous; at the same time there may be some tenderness on pressure in different parts of the abdomen. When this occurs, it may be assumed that the case is now complicated by peritonitis. I wish, however, to call particular attention to the fact that in many cases there was a complete absence of pain, although peritonitis was present; this refers both to the initial colicky pain and to the pain of peritonitis which, as I have said, may supervene later in the course of the disease. This peculiar absence of pain was observed in a case under my care in which there were infarction of fifteen and three-quarter inches of the ileum and suppurative peritonitis. Vomiting may accompany the pain or follow very soon after its onset.

In some cases vomiting begins either with the onset of pain or soon afterward. Diarrhea may begin at times shortly after the pain and vomiting, though sometimes it may be delayed two to four days. Sometimes the diarrhea is very violent, the stools mucous, watery, and at last bloody. In a number of cases the stools contain blood from the first.

The leading symptom of the disease is hemorrhage from the intestine. The source of the bleeding is apparent from examination of the anatomic lesions. In several cases the quantity of blood passed *per rectum* was very profuse, and even excessive; as a rule, a number of motions containing blood are passed in succession; in fact, melena has been observed for several days after the onset of the first symptoms (Moos); the motions are usually dark, blackish-brown, and of a tarry consistence; several clinicians expressly call attention to a peculiar musty odor. Gerhardt has stated, on more or less theoretic grounds, that the blood passed *per rectum* in embolism of the inferior mesenteric artery is bright red in color, whereas that passed in cases of embolism of the superior mesenteric artery is dark and of a tarry consistence; at present, however, the data are not sufficient to prove whether this assumption is correct.

The blood that accumulates in the bowel after an intestinal hemorrhage due to embolism of one of the mesenteric arteries is not always passed *per anum*, but may be retained. It is not, however, of much importance whether or not the blood is passed, as the diagnosis can be made from the presence of other symptoms which are characteristic of profuse intestinal hemorrhages, such as a sudden fall of temperature, collapse, and other symptoms described in detail on page 158, to which the reader may refer.

Besides these cases in which intestinal hemorrhage is the most prominent clinical feature, there is another group of cases which bears a striking resemblance to acute intestinal obstruction. [In 49 cases of embolism and thrombosis of the mesenteric vessels collected by Borzeky,[1] there were blood-stained motions in 14, in the remainder obstruction

[1] Borzeky, *Beitrag z. klin. Chirurg.*, 1901, vol. xxxi.

was complete from the first.—ED.] Examination of some of the earlier recorded cases brings out illustrative examples of this association, although the older authors did not recognize their true nature. Kaufmann recorded a typical example of this kind in which the patient had been suffering from constipation for a number of days, the abdomen was distended, painful and tender on pressure, and feculent vomiting was also present. The diagnosis of "ileus" was made, and laparotomy performed, during the course of which the patient died from collapse. At the autopsy there were infarction of twenty and one-half feet of the intestine and peritonitis. In the case which I reported above the symptom-complex of peritonitis with signs of paralysis of the intestine was also present, I have also been told of two other similar cases and the literature of the past decade contains several others, which have been collected by Deckart, together with 2 cases of his own. The similarity to the clinical appearances in acute intestinal occlusion may be so pronounced that Deckart, in his work on *Thrombosis and Embolism of the Mesenteric Vessels*, adopted the subtitle, "A Contribution to the Study of Ileus." The patient is seized with sudden abdominal pain, collapse, vomiting, meteorism, absolute constipation, and at times stercoraceous vomit. In fact, the entire symptomatology exactly resembles that of the acute intestinal obstruction (internal strangulation, volvulus), namely, the so-called strangulation-ileus. It is due to intestinal paralysis (ileus paralyticus), the cause of which is doubtless the acute nutritional disturbance which must occur after stoppage of the flow of arterial blood into the intestinal wall. But it is not yet possible to determine the factors which produce diarrhea, hemorrhage, or constipation and intestinal paralysis.

The clinical picture in a case of Schnitzler's, in which autochthonous thrombi were found, was entirely different. A woman of fifty-five years had suffered for six months from almost daily, spontaneous, violent, cramp-like pains in the abdomen. There was no palpable objective sign in the abdomen; no abnormal peristalsis; and bowel-evacuations occurred only with artificial aid. Laparotomy was without effect. After death there was blocking of the superior and inferior mesenteric arteries by tight fibrous, connective-tissue clots. In a similar case of Lépine's the patient had suffered for fifteen years from "abdominal neuralgia." Large branches of the mesenteric artery were occluded. It can readily be understood that in a case of this kind with an acute onset ushered in by pain, in which constipation has existed for some time and there is not the slightest sign of intestinal hemorrhage, the diagnosis of acute intestinal obstruction is extremely likely to be made and may seem perfectly justified. The symptoms of acute intestinal obstruction in these cases is produced by paralysis of the bowel, which may be limited to the infarcted portion of the bowel and be due to this accident, or it may involve larger portions of the bowel and be the direct result of complicating peritonitis (compare also the section on Paralysis of the Intestine and on Peritonitis). Kaufmann attributes the occurrence of feculent vomiting in his case to the fact that the paralysis

of the bowel persisted for so long a time.   [A palpable tumor due to the mesentery and intestine being infiltrated with blood has been detected in a few cases during life and may increase the resemblance to intussusception.—ED.]

Gerhardt and Kussmaul have drawn up some rules for the diagnosis of occlusion of the superior mesenteric artery.   The rules laid down by these authors are still of value and can be advantageously followed. It should be possible to determine the origin of the embolus, but even when this cannot be done, it does not follow that embolism can be excluded, for the embolus may be derived from the aorta when this vessel is atheromatous ; or again the clot may be autochthonous.   As a rule, there is embolism of some other artery before the appearance of symptoms of embolism of the superior mesenteric artery, or evidence of embolism of other arteries appears simultaneously with the embolism of the superior mesenteric artery—it is not, however, essential that this should be the case, and embolism of other arterial areas may be absent. There is copious intestinal hemorrhage, which is not due to any other independent intestinal process, nor, in all probability, to any form of obstruction of the venous circulation in the bowel-wall.   The temperature usually falls when hemorrhage occurs.   Either before or at the same time as the hemorrhage there are attacks of colicky pain which occasionally may be extremely severe—these attacks of pain, however, may be absent.   The other symptoms which may appear are tympanitic distention of the abdomen and exudation of fluid into the abdominal cavity.   When all these signs and symptoms are present, the diagnosis of occlusion of the superior mesenteric artery may be surmised.   In cases, however, in which intestinal hemorrhage in particular is absent and the syndrome of acute occlusion of the bowel is presented, the diagnosis cannot possibly be made ; and the most skilful diagnostician may easily be baffled.   [According to Makins,[1] the onset of symptoms is rapid in thrombosis and embolism of the mesenteric artery and slow in thrombosis of the mesenteric vein.—ED.]

The **prognosis** of this affection is very grave and its course, as a rule, unfavorable.   Death may occur within from twenty-four to forty-eight hours, and is usually accompanied by symptoms of collapse and of acute anemia.   In other instances the course of the disease is somewhat more protracted, and peritonitis finally develops, or the patient dies with symptoms of acute or subacute intestinal obstruction.   There is, however, both anatomic and clinical evidence to show that occasionally a case of occlusion of the superior mesenteric artery may terminate favorably.   Virchow, for example, in one instance found the superior mesenteric artery converted into a hard and solid cord, being occluded by a large, dry, and adherent mass of clot which extended into the aorta ; there was no change in the jejunum or the ileum.   Cohn has reported a case in which, to judge from the anatomic data, recovery followed occlusion of the superior mesenteric artery *intra vitam ;* in this case one of the colic branches of the superior mesenteric artery was

[1] Makins, *Brit. Med. Jour.*, 1898, vol. i., p. 1137.

blocked by an embolus; in the transverse colon there were areas of slate-gray discoloration and also a number of ochre-yellow spots which appeared to be the remains of old hemorrhages. Moos has also reported a case that was carefully studied, in which, as far as I can see, there is no reason to doubt the accuracy of the diagnosis; here too the patient recovered. [In a patient with mitral stenosis acute symptoms occurred two months before death; the superior mesenteric artery was blocked by an embolus (Karcher).[1]—ED.]

A favorable outcome can be expected only if a sufficiently free collateral circulation is established to keep up the nutrition in the affected parts. This occurs under two conditions—viz., in the first place, when the arterial area involved is not too extensive, and, in the second place, when the occlusion of the main trunk, or at least of a large branch, of the artery comes on very slowly; when this occurs, there is time for the reëstablishment of the circulation in the parts by means of the collateral vessels, and in this way the anemic portions are kept supplied with arterial blood.

Treatment.—The disease is not amenable to direct treatment. Faber is probably correct when he says that excessive hemorrhages are probably produced by increase of pressure in the portal system; this is the chief danger, and if the arterial blood-pressure can be raised by stimulating the heart, this danger can to some extent be eliminated; stimulation of the heart is, therefore, one of the therapeutic measures at our disposal. All other measures are purely symptomatic. The most important one is blood-letting.

Of recent years an operative treatment (resection, enterostomy) has been advocated and even attempted, with favorable result (Elliott). Further experience alone will decide definitely as to the value of this treatment.

[Gordon excised two feet of the small intestine, with recovery. Another case is reported by Tyson.[2] According to Watson,[3] about one-sixth of the cases are suitable for resection.—ED.]

THROMBOSIS OF THE MESENTERIC VEINS.

Pilliet, E. Grawitz, and Eisenlohr have recently reported cases of this condition. This subject is interesting not only from an anatomic, but also from a clinical, point of view, particularly since reports of cases that were studied during life are available. In view, however, of the small number of cases that have been reported so far we must content ourselves with a short summary of this disease. [Welch[4] has collected 32 cases, and proves that the superior mesenteric vein is far more often affected than the inferior mesenteric vein.—ED.]

The anatomic appearances of the intestine in thrombosis of the

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[1] Karcher, *Correspondenzbl. f. Schweiz. Aerzte*, 1897.
[2] Tyson, *Trans. Clin. Soc.*, vol. xxxv., p. 114.
[3] F. S. Watson, *Boston Med. and Surg. Jour.*, vol. cxxxi., p. 552.
[4] Welch, *Allbutt's System of Med.*, vol. v., p. 218.

mesenteric veins are the same as in obstruction of the mesenteric arteries ; the intestinal wall is thickened, the mucous lining of the bowel is loosened so as to be almost free and is necrotic ; the folds of the intestinal mucosa are dark red and edematous ; in a case reported by Grawitz the inner surface of the intestinal wall was gangrenous and almost "diphtheritic." The intestinal contents are blood-stained and discolored. In a few cases peritonitis, either of the simple fibrinous form or complicated by hemorrhagic exudation, has been present. The hemorrhagic infarction of the intestine cannot be considered as due to venous engorgement, but as the result of arterial fluxion.

[There may be wide-spread thrombosis in the mesenteric veins without any hemorrhagic infarction. In a case reported by the editor [1] there was thrombosis in both the inferior and superior mesenteric veins, but no hemorrhagic infarction of the intestine. In cases in which thrombosis begins in the portal vein and subsequently spreads into the trunk of the superior mesenteric vein, hemorrhagic infarction may be absent ; while, conversely, acute portal obstruction may cause hemorrhagic infarction of the intestine without thrombosis of the mesenteric vein (Welch). Thrombosis of the superior mesenteric vein is often part of pylethrombosis or thrombosis of the portal vein, and the effects of the two are not always capable of being rigidly apportioned.—Ed.]

The mesenteric veins corresponding to the diseased area are blocked by thrombi ; the arterial branches, on the other hand, are all free and patent. In the case recorded by Grawitz and in several of the cases reported by Pilliet the thrombi could be followed far up into the portal vein.

In this condition the thrombotic process begins in the peripheral branches of the mesenteric veins—that is, in those portions of the vessels situated either close to or actually in the intestinal wall ; from this point the process extends in an ascending direction ; in other words, it does not begin in the portal vein and extend downward into the mesenteric veins. Occasionally, however, as we know, the thrombus, in cases of primary pylethrombosis, will extend for a considerable distance downward into the tributaries of the portal vein and sometimes even as far as their origin—*i. e.*, the commencement of the mesenteric vein. Alexander has reported a case of this character in detail. The best proof of the primary involvement of the portal vein in these instances is the fact that other tributaries of the portal vein are thrombosed, and that the mesenteric veins are greatly dilated. Nearly all the other cases hitherto reported show other conditions that might be of etiologic importance.

[Thrombosis of the mesenteric veins is often the early stage of suppurative inflammation of the portal system and may occur as the result of infection in appendicitis, ulceration of the intestinal mucous membrane, dysentery, etc. It may also occur in malignant disease of the colon. Traumatism may produce inflammation and thrombosis in the mesenteric veins ; the same result may occur after operation (Mayland).[2]

[1] Rolleston, *Trans. Path. Soc.*, vol. xliii., p. 49.
[2] Mayland, *Brit. Med. Jour.*, 1901, vol. ii., p. 1454.

Raymond Johnson[1] has recorded a case of tuberculous peritonitis in which acute intestinal obstruction probably depended on thrombosis of certain tributaries of the superior mesenteric vein. In Bradford's[2] case thrombosis was probably secondary to disease of the mesenteric glands. In Taylor's[3] case of a girl aged five years the superior mesenteric artery and vein were both thrombosed at a point where they were constricted by a mass of fibrous tissue in the mesentery. In Gull's[4] often-quoted case of a man aged twenty-three who had recently contracted syphilis, the cause of intestinal hemorrhage and the passage of valvulæ conniventes by the rectum was diagnosed as thrombosis of the mesenteric veins due to the pressure of a gumma, but as recovery occurred, there is no proof of this.—ED.]

The clinical picture, as deduced from the available material, is exactly like that of embolism or thrombosis of the mesenteric arteries, even in the existence of various types showing diarrhea, hemorrhage, and obstruction. For a description the reader should, therefore, refer to the previous section. In fact, it is exceedingly difficult, if not impossible, to distinguish primary venous thrombosis from arterial occlusion, apart from the point that in one there is a primary change in the intestinal blood-vessels and in the other, a source for the arterial embolism. In Grawitz's case there were symptoms of severe intestinal catarrh with enterorrhagia and shortly before death severe pains in the abdomen, so that Mosler's diagnosis of typhoid fever with perforative peritonitis was regarded as probable.

[Mayland draws attention to a peculiar excitability of the nervous system suggesting hysteria. From a survey of the published cases he finds that while there is much variation in the symptoms, the following points are more or less characteristic : (1) Intra-abdominal pain is invariably present : it may come on acutely or gradually, and varies in position. (2) Loose motions ; blood may be present, but this depends on the extent of bowel involved and on the degree of congestion. (3) Vomiting is inconstant. (4) The general condition of the abdomen is negative ; there is no tenderness, rigidity, or distention. (5) Weak and rapid pulse. (6) Temperature presents no certain character, though it is usually reduced in severe cases. (7) Great excitability of the nervous system.—ED.]

No **treatment** for thrombosis of the mesenteric vein is known, except perhaps a surgical one when the process begins with symptoms of intestinal occlusion and can be operated on early. In such a case reported by Elliott 48 inches of intestine were resected and the ends sewed into the wound. Fourteen days later union of the resected bowel was complete and recovery followed.

[1] R. Johnson, *Trans. Clin. Soc.*, vol. xxxi., p. 212.
[2] J. R. Bradford, *ibid.*, vol. xxxi., p. 203.
[3] F. Taylor, *Trans. Path. Soc.*, vol. xxxii., p. 61.
[4] W. W. Gull, *Guy's Hosp. Repts.*, vol. xxvii., p. 15.

VENOUS HYPEREMIA OF THE INTESTINE.

Whenever the return of blood from the intestine is interfered with, venous engorgement results. The most direct cause of this occurrence is obstruction to the flow of blood in the trunk of the portal vein and its intrahepatic branches, due to various causes (as in cirrhosis hepatis); another cause of venous hyperemia of the intestine and of venous stagnation in the intestinal vessels is interference with the free passage of blood out of the inferior vena cava in many forms of heart disease and in some pulmonary diseases.

In the local infarctions following acute occlusion of branches of the mesenteric veins, described in the previous section, there is not so much venous hyperemia as hemorrhagic infarction, the blood coming from the afferent arteries of the infarcted area and causing flooding of the part with blood. Another form of venous hyperemia which must also be mentioned occurs in paralysis of the intestine, provided the stasis of blood in the paralyzed intestinal walls is not prevented by the overdistention of the parts.

The intestine is never involved alone in these various forms of hyperemia. This is obvious from consideration, on the one hand, of the anatomic changes in this condition, and, on the other, of the various causes of intestinal venous hyperemia. In addition, other areas supplied by the portal vein which pour their blood into this vessel are involved at the same time; finally, when hyperemia of the intestine is due to the backward pressure of heart disease, the whole area drained by the inferior vena cava may be in a similar state of passive hyperemia.

In chronic venous hyperemia the intestine and the mesentery assume a dark-bluish and cyanotic color; the veins of the mucous, submucous, and serous coats are dilated and filled with blood. The whole wall of the intestine may be thickened, succulent, and swollen and edematous. These changes, however, are not evenly distributed throughout the whole length of the intestine, nor are they uniform in severity in different portions of the bowels; in some instances the large intestine is greatly engorged, while the small intestine is only slightly affected; or, again, different portions of the large or of the small intestine may be found in different stages of hyperemia, so that light-colored areas alternate with darker ones. Dilatation of the hemorrhoidal veins will be considered in the next section.

The important question arises here, which must be answered if possible, whether venous hyperemia is a cause of intestinal catarrh. That this does not necessarily follow I have been able to determine in many cases of heart disease that I have examined and in which there was hyperemia of the bowel-wall, but no catarrh. In many of these subjects there were considerable degrees of cyanosis, advanced venous engorgement of both the large and the small intestine, but no signs whatever of catarrh, even on microscopic examination.

It is impossible to make a direct clinical diagnosis of venous hyperemia of the intestine. The only functional evidence of this con-

dition which is occasionally observed is constipation, which, as we know, is not infrequent in patients suffering from various heart lesions, particularly during the stage of failing compensation in very chronic cases. It is true that physiologic experiments show that venous engorgement brought about artificially by occlusion of the portal vein or of the inferior vena cava may set up intestinal peristalsis, which, as a rule, is very slight; we also know that the circulation of dyspneic blood through the bowel-wall may produce fairly vigorous peristaltic movements. On the other hand, under certain circumstances, dyspneic blood has been known to have the reverse effect—of inhibiting intestinal movements. In the instances just mentioned, however, the abnormal conditions are brought on acutely, and are entirely different from venous engorgement of the intestine due to chronic conditions, such as heart disease and general cyanosis. Apart from the fact that these chronic forms of venous hyperemia may directly produce structural changes in the wall of the bowel, there must be some other factor capable of producing constipation under these conditions—viz., impairment of the nerves supplying the bowel-wall. It is clear that an insufficient supply of blood to the nerves and ganglia of the bowel-wall, particularly when this diminished supply is of long standing and when the blood is, in addition, altered in quality from its diminished oxygen value, must necessarily seriously interfere with the normal function of these nervous elements; the direct result of this impairment will be a loss or a reduction of automatic activity. I consider that this perversion of the normal nervous mechanism of the bowel-wall is the direct result of chronic venous hyperemia of the parts; and that this perversion is the cause of the constipation in these cases.

It is well known that in some cases of heart disease diarrhea alternates with constipation. In these cases microscopic examination shows distinct evidence of intestinal catarrh; this condition was well marked in the cases I examined, which showed an abundant round-cell infiltration in the mucosa. Whether or not chronic venous hyperemia determines a predisposition to catarrh by lowering the resistance of the intestine remains to be proved; from analogy this explanation is not improbable, since this sequence of events is certainly seen in other parts of the body.

HEMORRHOIDS (Phlebectasia Hemorrhoidalis; Hemorrhois).

The hemorrhoidal veins surround the lower portion of the rectum and there form the hemorrhoidal plexus. The majority of these veins enter into the inferior or external inferior hemorrhoidal veins and from there into the common pudic veins and the internal iliac vein; others enter the median inferior hemorrhoidal veins and pass from there directly into the internal iliac vein and so into the area of the inferior vena cava.

A small number of these veins pass *via* the superior hemorrhoidal veins and the inferior mesenteric veins into the portal system. It will be seen, therefore, that the blood of the hemorrhoidal veins has

two destinations. In portal obstruction the blood from the hemor-rhoidal veins can still pass into the vena cava. On the other hand, back pressure in the system of the inferior vena cava, of cardiac origin, becomes manifest throughout the whole hemorrhoidal system of veins. In this connection it is noteworthy that all the hemorrhoidal veins are devoid of valves.

In the language of the physicians of antiquity the word "hemor-rhoids" literally signified hemorrhage, and in the clinical sense was applied, for instance, to bleeding from the mouth, the uterus, etc. ; the ancient writers, therefore, spoke of hemorrhoids of the mouth, of the uterus, etc. At the present time the term is used only in an anatomic sense to designate pathologic dilatation of the hemorrhoidal veins ; between the old term as applied by ancient writers, and the term as applied by modern authors, there lies a long and interesting historic development.

Laymen speak of hemorrhoids and of a hemorrhoidal state, and even now the latter expression carries with it something of the mystical, at least in the minds of the public. This is the result of a very old tradition first formulated as a hypothetic system of Georg Ernst Stahl two hundred years ago (1698). Stahl's work was entitled " de Vena Portæ Porta Malorum Hypochondriaco-splenetico-hysterico-colico-hæmorrhoid-ariorum." Even at the present day traces of this theory are occa-sionally seen in the writings of some authors. It would be an inter-esting task to follow the development of the doctrine of hemorrhoids through different periods of history, but I am obliged to refrain from doing so here ; and for the sake of general information will merely make the following statements :

Formerly hemorrhoids were not regarded as a local disease of the rectum, but as a general constitutional state (dyscrasia vel diathesis hemorrhoidalis) ; the hemorrhoids themselves and hemorrhoidal bleed-ing were considered to be local manifestations of the general disease. Stahl advanced the theory that the hemorrhoidal veins were a reservoir intended to hold any excess of blood in the organism, and were so constructed that they could readily fulfil this purpose ; hence hemor-rhoids were coupled with another old and hallowed conception—viz., " plethora abdominalis " ; in some of the older writers we also hear of " Anschoppungen, Stockungen, Physkonien im Pfortadergebiet " (arrest of the circulation, stasis, congestion in the portal area), these terms being used as synonymous with abdominal plethora. At a given moment it was believed the excess of blood was removed by bleeding and the patient benefited in this way ; the hemorrhage was considered a crisis which relieved the patient's distress (hence also the name " the golden artery ").

This view has not yet been completely dispelled. Even now some medical men distinguish between hemorrhoids due to mechanical causes and hemorrhoids which they call constitutional ; the latter they believe to be merely a local manifestation of some general pathologic condition. I unhesitatingly adopt the standpoint of the majority of modern

medical men in denying any constitutional basis for hemorrhoids, as no proof has ever been adduced to show that there are any grounds for this belief.

An unusual view was recently put forward by O. Rosenbach to the effect that the hemorrhoids are not always the result of local causes, but that "the point of visible stasis and the point of the primary vascular disturbance are not always identical. Thus hemorrhoids are often only a symptom of a synchronous overwork of the specific protoplasm of individual abdominal organs with the processes of chemic metabolism, and this overwork (greater excitation) of the liver and intestine leads eventually to mechanical insufficiency and to circulatory disturbances in the venous system, when the propelling forces are not able to overcome the increased resistance involved in returning the blood." (For a further explanation of Rosenbach's theory the reader must refer to his book.)

In order to have a solid foundation for our description of the etiology of hemorrhoids, a description of their anatomy will preface this account.

Anatomy.—In describing the anatomy of hemorrhoids it is well to remember that in their initial stages and before they are fully developed, hemorrhoidal ectasies are much more readily detected during the life of the subject than after death; in making postmortem examinations only marked and fully developed hemorrhoids are found.

External and internal hemorrhoids are distinguished according to their position: the former are readily visible with the naked eye, are situated below the sphincter ani, and are arranged in groups around the external orifice of the anus; the former can be discovered only by digital examination or with the aid of the rectoscope, and are situated above the sphincter muscle. External and internal hemorrhoids are frequently present together. As a rule, hemorrhoids are limited to the immediate vicinity of the sphincter, but occasionally dilated rectal veins are found much higher up, and in exceptional instances they have been found in the sigmoid flexure of the colon.

Two forms of dilatation of the hemorrhoidal veins can be distinguished—viz., a diffuse and a circumscribed nodular form. The diffuse form is often only an initial stage of the hemorrhoidal process; occasionally, however, it persists as such throughout the disease. In the latter instance, that is, in the permanently diffuse form, the veins of the rectum become thick and tortuous and are converted into elongated cords lying in close proximity to one another. The circumscribed form of hemorrhoids—the so-called hemorrhoidal nodule—constitutes a true varix. These varices vary in size from that of a lentil to that of a walnut, and occasionally are larger still. The nodules may be roundish or flattened or irregular in outline and angular. Sometimes there is only one pile, sometimes only a pair of them; occasionally they are multiple and form a ring or a semicircle around the anal opening; there may be two rings of hemorrhoids—namely, an external and an internal ring higher up, while in some instances a third ring may be found still higher up. In other instances the hemorrhoids are irregu-

larly distributed throughout the rectum. In nearly all instances the clumps of piles have a broad base of attachment; occasionally, however, they become pedunculated, especially when they are forced out by the contractions of the bowel in such a way that the rectum becomes everted at the same time; when this occurs, they may become strangulated at the margin of the anus and so become pedunculated.

Opinions as to the anatomic character of hemorrhoids vary greatly. Originally they were simply taken for venous ectasies; later, however, certain anatomic peculiarities were discovered in isolated cases and led to the adoption of different views as regards their anatomic structure; these views may now be considered disproved and hardly require discussion, as they are thoroughly refuted. At the present time it may be stated that hemorrhoids originate from simple venous ectasies and are genuine varices; this view is generally accepted by all modern investigators. But recently this has again been disputed. For instance, Mainzer maintains that they are true cavernomata in the sense that that there is an actual new formation of vessels, originating in the submucous adipose tissue, with subsequent cavernous transformation. Reinbach arrived at the same conclusion from examination of piles removed at operations. He described a new formation of blood-vessels, developed by gemmation, in the form of cavernous, tumor-like new growths—a development of angiomata, for which the anal region appears to possess some predisposition. Signs of venous stasis and inflammation may be associated. Raschkow advocates the theory of a local angiomatous tumor formation.

In old hemorrhoids secondary changes may supervene. Dilated blood-vessels, or, in other words, venous varices, in the immediate vicinity of a group of hemorrhoids may become inflamed, adherent, and coalesce; as a result the walls of these dilated vessels may undergo atrophy and tumors of considerable size may result, which resemble cavernous tumors and are multilocular. Tumors as large as an apple have been described. The inflammatory and hyperemic processes just mentioned may also bring about another change in the clumps of piles—viz., instead of forming cavernous sinuses within the piles, they may convert the external covering of each varix into a thick shell with a hard, solid, and resistant surface. In other instances, when there is no inflammation or congestion inside the pile, its outer covering may become extremely thin—no thicker than paper; this is specially frequent in external hemorrhoids. In other cases the blood coagulates, forms thrombi within the pile, thus causing atrophy of the tissues of the varix. Lastly, phleboliths are not uncommon in old hemorrhoids. When the circulation of the blood in the hemorrhoidal varix is interfered with by coagulative and inflammatory changes inside the pile, its cavity may contain a certain amount of blood which still remains fluid, and in this way a sort of blood cyst is occasionally formed.

The mucous membrane of the rectum in the immediate vicinity of the clumps of piles is always hyperemic, and, as a rule, in a state of catarrhal inflammation. This and some other important clinical conse-

quences will be referred to in the section on Symptomatology of Hemorrhoids.

Finally, attention should be called to the fact that of recent years Allingham has revived some of the older views as to the anatomy of hemorrhoids by distinguishing between venous, capillary, and arterial hemorrhoids. The observation that true arterial telangiectasis occasionally occurs in the arteries of the rectum is old, but does not justify the recognition of a special form of arterial hemorrhoid ; I consider such a subdivision to be impractical. Bardeleben, moreover, has called attention to the fact that arteries of medium size, or even fairly large arterial trunks, may be forced forward together with a varix which is growing from the submucous tissues, and may thus apparently form an integral part of the hemorrhoidal swelling ; on the other hand, two or more varices or clumps of piles may gradually enlarge and finally coalesce, as shown above, and in so doing may include the artery within the cavernous space thus formed ; in this way the artery actually comes to form an integral part of the hemorrhoid.

Etiology.—Dilatation of the hemorrhoidal veins, as I have already shown, is entirely a local disease. The reasons why some writers still adhere to the old idea of a hemorrhoidal diathesis and accept the arguments brought forward to support this theory will be mentioned in the closing paragraphs of this section.

There can be no doubt that mechanical factors are the primary causes of dilatation of the rectal veins. At the same time we must carefully consider the question whether local disease of the walls of these veins may not be the primary factor in the production of hemorrhoidal varices ; in other words, an attempt must be made to find out whether conditions are present in this local disease of the veins which are similar to those present in aneurysm of the arteries—viz., localized dilatation of the blood-vessels. Von Recklinghausen has carefully considered all the points bearing on this question, and has arrived at the conclusion that there is no proof to support the proposition that local changes in the vessel-wall are the primary and sole starting-point of the hemorrhoids. I must refer the reader to his paper for the details ; he recognizes the occasional appearance of minute anatomic changes in hemorrhoidal phlebectasies, but does not consider them to be the primary cause of the lesions ; he believes, moreover, that gravity is the prime factor ; in other words, that the determining and the essential factor in the production of hemorrhoidal phlebectasies is purely physical in character and consists in the accumulation of blood in the veins of the rectum and the difficulty opposed to the return of the blood from a part of the body where the laws of gravity oppose the return of the blood. In whatever position we may be, he argues, whether sitting, lying, or standing, the blood accumulates in the hemorrhoidal veins, as these vessels are always in a dependent position and the return of blood must, therefore, always be against the force of gravity.

In addition to this factor a number of concomitant and accessory factors, which all appear to play a part in causing the development of

hemorrhoidal varices, must be considered ; the main question to be decided is why the veins of the rectum should be particularly liable to varicosity, while the veins of the lower extremity, which are equally exposed to the factors enumerated above—viz., the opposition of the force of gravity to the return of the blood—are not so apt to undergo dilatation. The chief reason for this difference is the fact that the veins of the lower extremities have numerous valves, whereas the rectal veins have not. In addition the walls of the veins of the lower extremity contain a quantity of muscular tissue, whereas the walls of the intestinal veins contain few muscle-fibers and are thin in comparison with their size. Another factor that favors the veins of the lower extremities is the frequent contractions of the muscles of the parts ; these contractions without doubt help the return of the blood toward the heart ; exactly the opposite conditions are present in the rectal veins where the fecal masses compress the blood-vessels of the parts, while contraction of the abdominal muscles during defecation interferes with the return of blood from the rectal veins. These various factors, taken as a whole, to a certain degree unquestionably support von Recklinghausen's dictum that "Fluid contained in a flaccid and dependent sac always sinks to the bottom, and thus both dilatation and elongation of this sac occur."

Analysis of the various factors concerned in the clinical etiology of hemorrhoids from this point of view shows that everything of real etiologic importance as apart from purely hypothetic considerations— in other words, the results of actual observation as opposed to speculative deductions—can be combined under this common point of view.

A variety of factors of a purely local character—that is, factors that interfere directly and mechanically with the circulation of the blood in the veins of the lower part of the rectum—all favor the development of ectatic dilatation of these vessels. As a good example the occurrence of hemorrhoids during pregnancy, which, as we know, is by no means uncommon, may be cited ; when the uterus undergoes postpartum involution, the hemorrhoids may rapidly disappear. Permanent enlargement of the uterus, the ovaries, or of the prostate gland acts in a similar way and may produce hemorrhoids by local interference with the circulation of the blood through the hemorrhoidal veins. The accumulation of fecal material in the rectum necessarily interferes with the return of blood from the veins of this part of the intestine, and, as a matter of fact, constipation is frequently associated with hemorrhoids. The fact that hemorrhoids are not constantly present in all cases of chronic constipation, as the above considerations might lead us to expect, must be attributed to the fact that in constipation accumulation of fecal material does not occur in the rectum, but higher up in the bowel—in the sigmoid flexure or in the colon. Duret, moreover, has called attention to the fact that the pressure exerted by the feces is not the chief factor in bringing about dilatation of the rectal veins, and that the excessive pressure exerted by the abdominal muscles in their attempt to expel the bowel contents in chronic constipation is much more important. Strictures of the rectum may also have the same effect in a purely mechan-

ical way.   On page 433 I have already called attention to the occasional development of hemorrhoidal dilatation of the rectal veins and hemorrhages from these vessels in cases of carcinomatous stricture of the rectum and to the fact that the hemorrhoids may in a sense apparently be considered an early sign of the disease.   I wish, however, to call particular attention to the fact that the inter-relationship between hemorrhoids and constipation of any kind may be quite different from the one described above ; constipation may not only produce hemorrhoids, but, conversely, from the pain and the difficulty of defecation caused by the hemorrhoids, the patient may avoid passing a motion as long as possible ; in a case of this kind, where the hemorrhoids are due to some cause other than chronic constipation, they may be regarded as responsible for the constipation.   Lastly, in other cases, constipation and hemorrhoids may be the joint effect of some common cause.

   "Stasis in the portal system" is systematically given as one of the most important causes of dilatation of the veins of the lower part of the rectum ; portal engorgement is chiefly due to cirrhosis of the liver (occlusion of the portal vein itself may be considered so rare an event that it need hardly be considered here) and certain affections of the heart and lungs leading to general engorgement of the venous system. There can be no doubt that hemorrhoids, when they do develop in the course of one of these diseases, must be considered the result of obstruction in the portal vein preventing the return of blood from the rectal veins.   It is remarkable, however, that hemorrhoids seldom develop in cases of chronic engorgement of the portal veins.   Many years ago Frerichs expressed his views on this point as follows : "Hemorrhoidal varices, in my experience, are by no means frequent in this form of disease of the liver (*i. e.,* cirrhosis)" ; Monneret, Sappey, Damaschino, Thierfelder have indorsed this view, with which I fully agree.   This statement applies with equal force to chronic venous engorgement in the inferior vena cava occurring in some forms of heart and lung disease, where hemorrhoids are by no means frequent or specially prominent. It appears to me that the view so widely held as to the frequent incidence of hemorrhoidal dilatation of the rectal veins in chronic venous engorgement of the portal vein and in backward pressure in the inferior vena cava in heart and lung disease is based more on theoretic reasoning than on actual observation.   But even the purely theoretic considerations which might lead to the *a priori* adoption of this view are open to question, for in cirrhosis of the liver and in cardiac insufficiency the pathologic increase in the blood-pressure is distributed uniformly over such an enormous area, in which the collateral vascular communications are so free, that there is no special and exaggerated engorgement in the region of the hemorrhoidal veins.   I am distinctly of the opinion that chronic engorgement of the portal vein and of the inferior vena cava under the conditions mentioned above is only of subordinate importance in the production of piles.

   Certain habits and walks of life influence the development of hemorrhoids by producing mechanical conditions which interfere with the

return of blood from the hemorrhoidal veins, or at least directly predispose to overdistention and overfilling of the veins of the rectum. The predisposing factors include sitting down for long periods of time, particularly on soft, upholstered, or warm seats, riding on horseback a great deal, etc. In cases of this kind the various factors which may produce hemorrhoids are often combined; one and the same condition may, for instance, directly predispose to hemorrhoids in the way just mentioned and may also cause constipation, which in its turn leads to the development of hemorrhoidal dilatation of the rectal veins. Eichhorst has recorded the existence of piles in a number of persons who played wind-instruments and naturally had to make excessive expiratory efforts; he maintains that in these cases the formation of hemorrhoids was due to the continuous and forcible increase of the intra-abdominal pressure.

A number of other factors are said to produce hemorrhoids by causing " loosening of the tissues " in the neighborhood of the hemorrhoidal veins. This, again, is a purely local factor, which would act merely by causing local hyperemia of the mucous membrane in the lower part of the rectum, and therewith dilatation of the rectal veins. It was believed for a time, for instance, that persistent catarrh of the rectum or the abuse of drastic purgatives might act in this way. It is not easy, however, to form a cautious and definite decision on this question. This applies particularly to cases in which both a catarrhal state of the mucous membrane of the lower part of the rectum and hemorrhoids are found to be present when the patient is first examined. In a case of this kind the exact sequence of events cannot be made out, and it is difficult or even impossible to decide whether the hemorrhoids and the catarrh developed consecutively or simultaneously; it may very well be that the hemorrhoids were the primary factor and that they indirectly and secondarily led to the development of catarrhal inflammation in the affected portions of the intestinal mucosa. And, lastly, in cases where no external piles are present, the rectum is not examined : the veins of this portion of the bowel may be in a state of dilatation from inflammatory hyperemia, but the rectum is rarely examined, as the diagnosis of " catarrh " is made. How are we to determine positively in a case of this kind that the catarrh of the rectum was the primary factor and produced the hemorrhoidal ectasy of the rectal veins? Besides, " catarrh " is unquestionably often diagnosed in practice when there is in reality nothing more than habitual constipation, which may be due to entirely different causes. Again, how is it possible to prove that the abuse of drastic purgatives ever causes hemorrhoids? For, in the first place, these remedies are administered for nothing more or less than for chronic constipation. It seems much more rational to assume that in these cases the constipation and not the drastic purgatives that were administered for its cure was the direct cause of the hemorrhoids.

Some other factors which are said to cause piles or to play an important rôle in the etiology are also of questionable importance, such as excesses *in venere,* the abuse of alcoholic drinks, of spiced

foods, and the indulgence in excessive quantities of food and drink in general. The importance of all these factors in the production of hemorrhoids was explained by the tendency to local rectal hyperemia and of general plethora that they were said to favor. It must always be remembered, however, that in habitual drunkards and in gourmands there is, as a rule, a tendency to chronic constipation as a direct result of the sedentary life they usually lead; this constipation in itself is much more apt to produce the supposed local hyperemia of the rectal mucosa than the excesses in drinking and eating *per se.* [Lauder Brunton [1] says that in some persons a glass or two of champagne will bring on an attack of piles, and regards this as analogous to the effect of champagne in bringing on gouty manifestation, such as phlebitis.—ED.] " General plethora," it is true, is almost universally considered a prolific cause of hemorrhoidal dilatation of the rectal veins and as an important factor in the pathogenesis of this lesion. It is currently believed that any further increase in the quantity of tissue fluids following the abundant absorption of pabulum from the intestine into the mesenteric veins has a tendency to increase this plethoric condition of the blood. If we leave the doubtful question as to the existence of such a state as plethora entirely on one side and adhere strictly to what can be learned from actual observation and experience, it appears in the first place that hemorrhoids are very frequent in subjects who are in an average state of nutrition, and may, in fact, be spoken of as thin, and that in practice piles are, at least in my experience, more frequently found in such subjects than in very stout, obese, or " full-blooded " individuals. In the second place, that hemorrhoids are by no means constant in all truly obese or " full-blooded " subjects. I fully indorse the statement made by von Recklinghausen to the effect that " hemorrhoids are by no means particularly common in plethoric subjects,—as was formerly taught on purely theoretic grounds,—but are, on the contrary, usually found in subjects with small motor energy and in individuals who lead a sendentary life, and who, while not taking too little nourishment, nevertheless do not succeed in replacing the loss of tissue in the muscular system owing to their defective general metabolism; these individuals, moreover, are usually anemic; the reason why hemorrhoids are apt to develop in these persons is that the blood is not driven through the vessels with the necessary force. It can readily be understood that with a defective circulation and a weak cardiac impulse the blood will follow the laws of gravity and accumulate in the dependent portions of the body; in this way stagnation of venous blood may be assumed to occur in the blood-vessels of the lower part of the rectum, followed necessarily by dilatation of the rectal veins."

This really exhausts everything that can be said as to the etiology of hemorrhoids. A number of other factors which have been regarded in the light of disposing causes, such as constitutional influences or the hemorrhoidal diathesis, either do not exist or must be interpreted to mean something else. Thus it is stated that Oriental nations (Turks, Egyp-

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[1] T. Lauder Brunton, *Brit. Med. Jour.*, 1892, vol. i.

tians) are particularly prone to suffer from hemorrhoids. Even if this statement could be verified, it would not prove that there is such a thing as a hemorrhoidal diathesis; all that the frequent occurrence of hemorrhoids among Orientals would show would be that their mode of life predisposes to hemorrhoidal disease; as a matter of fact, we know that Moslems lead very sedentary lives, and are in the habit of sitting with folded legs on soft cushions. The fact that hemorrhoids are often hereditary has no bearing whatever on the existence or non-existence of a hemorrhoidal diathesis; it appears to me that the supposition is much more rational that in those persons who suffer from hemorrhoids and in whom a history of hemorrhoidal trouble in previous generations can be obtained (and this is undoubtedly often the case) there is a hereditary transmission of certain anatomic peculiarities in the structure of the venous system. It is well known that definite anatomic peculiarities may be transmitted through several generations of one family.

The fact that hemorrhoids are particularly frequent in the middle years of life can readily be explained from the greater frequency during these years of the various etiologic factors which are responsible for hemorrhoidal dilatation of the rectal veins.

Clinical Features.—Hemorrhoids are a local disease, and their characteristic features are almost exclusively local in nature. Remote symptoms of the disease are only rarely seen, and when they do appear, must be considered as due to some accidental complication occurring in the course of hemorrhoidal disease, or as the direct effect of the same causes as are responsible for the local disease.

The essential features of hemorrhoids are the dilated and varicose veins. If the anal folds are drawn apart and stretched, clumps of external piles appear as large or small tumors of the character described above; they are usually round, bluish in color, and shiny; they may either be full of blood and distended almost to bursting, or they may be collapsed and flaccid; occasionally they do not appear until the anal folds are stretched very far and the anal orifice is widely opened, and in other cases it is necessary that the patient should strain vigorously before they appear. Clumps of internal piles can be detected only by digital examination of the rectum or by inspection of the rectum through a speculum. Occasionally there are dilated cylindric veins running a tortuous course in the mucous membrane of the rectum, a condition which may occur either in association with hemorrhoidal varices or alone.

Hemorrhage may occur from these cylindrically dilated or varicose veins of the rectal mucosa, or from capillary ectasies that are occasionally found in this situation in hemorrhoidal disease; as a matter of fact, these hemorrhages have given the name hemorrhoids to this affection. In some cases hemorrhoids last for years or even throughout the life of the patient without at any time producing hemorrhage, and are popularly spoken of as "blind" hemorrhoids. In other instances bleeding occurs occasionally at long intervals. Again in another group of cases bleeding occurs at more or less regular intervals, sometimes even peri-

odically; this regularity is in all probability due to periodic and regular fluctuations in the degree of fulness and distention of the hemorrhoidal varices; formerly this peculiar regularity led to the formulation of quite fantastic theories as to a relationship which was thought to exist between the occurrence of hemorrhoidal bleeding and certain phases of the moon, or with menstruation (vicarious menstruation). Lastly, in some patients bleeding persists for weeks, there being a daily loss of a certain quantity of blood. The color of the blood varies: sometimes it is bright, sometimes it is dark. The quantity of blood lost also varies—sometimes only a few drops or a few streaks of blood are found on the surface of the feces, while in other cases hemorrhage may be so profuse that the patient becomes greatly exhausted and may even faint after one of these acute gushes of blood from the rectum; between these different grades of severity we find all degrees of hemorrhage. Anemia is quite a common result, especially when the hemorrhage, though not excessive at any one time, occurs daily for a long period of time; this may lead to very grave secondary anemia with all its characteristic features. A description of the symptomatology of anemia is unnecessary here. Ewald has recently directed attention again to those cases in which, on account of a long-continued, progressive anemia, the diagnosis of pernicious anemia is made until a careful examination reveals hemorrhoidal bleeding. The blood may gush forth from the rectum pure and unmixed with other material, or it may be mixed with fecal matter. When the feces are formed, the blood is always on the external surface of the dejecta and never intimately mixed with the feces. If the hemorrhage is severe, so that a large quantity of blood is passed at once, the feces may, so to speak, float in the blood. Hemorrhages from piles either appear quite spontaneously without any apparent extrinsic provocation, or they are precipitated by some external cause—a ride on horseback, some excess in Baccho, etc. Small quantities of blood are frequently passed with the stools when the feces are hard and much straining is necessary in defecation; under these conditions some of the dilated blood-vessels in the lower part of the rectum may easily become slightly eroded. The majority of hemorrhoidal hemorrhages are due to capillary erosion and only rarely to rupture of a varicose vein. In the section on Intestinal Hemorrhage it was pointed out that in cases of intestinal bleeding of obscure origin it is important to consider the possibility of distended hemorrhoidal veins situated high up in the intestine and visible only with the Otis proctoscope.

The symptoms of hemorrhoids are frequently limited to these two phenomena—namely, the presence of dilated veins in the rectum and occasional hemorrhages from them. Subjective symptoms may in many cases be completely absent; a patient may not become aware of the existence of hemorrhoids until hemorrhage occurs from the rectum. As a rule, however, some subjective symptoms are in evidence. Generally speaking, these are confined to local sensory disturbances, such as tickling, itching, burning, pricking, a feeling of pressure and weight in

the rectum and anus, and occasionally the sensation as of a foreign body impacted in the rectum or the anus, with moderate tenesmus. All these symptoms may vary in intensity at different times ; the bleeding and the subjective signs may disappear altogether for a time or may increase greatly in severity ; all the factors that determine increased venous engorgement in the veins of the anal region or that produce arterial fluxion in this area exercise a marked influence on the severity of the manifestations of this disease. Patients with hemorrhoids are particularly apt to complain of disagreeable sensations of pressure in the sacral and the whole lumbar region. When the various distressing symptoms produced by hemorrhoids become greatly exaggerated, the resulting condition is that described by the older physicians under the name of hemorrhoidal colic. This collection of symptoms is characterized by a feeling of soreness over the whole of the lower part of the abdomen, pain in the sacral region, a feeling of pressure in the rectum, bladder, uterus, and vagina, while in addition pain radiates into the thighs. Not uncommonly such an exacerbation of subjective distress of this kind precedes a hemorrhage, after which the abnormal sensations disappear and the patient feels well for a shorter or a longer time.

The character of the stools in this simple form of hemorrhoidal disease varies. Even though the clumps of piles attain a considerable size, defecation may proceed quite regularly and the bowels may be open every day ; as a rule, however, the patients suffer from irregularity of the bowels ; this interference with the normal action of the bowels may be due to a variety of causes ; in one of the preceding paragraphs, when discussing the etiology of hemorrhoids, attention was called to the fact that habitual constipation must be considered one of the most prolific causes of hemorrhoids ; under these conditions constipation is primary ; but in other cases the hemorrhoids and constipation come on simultaneously, and they must be considered as concomitant results of the same primary cause. Occasionally, however, the presence of piles in the lower part of the rectum actually causes, or at least favors, the development of constipation, especially when the clumps of piles are irritated, inflamed, torn, or ruptured. These complications invariably make defecation painful, so that the patients (perhaps foolishly and certainly to their detriment) postpone the act of defecation as long as possible. It is true that in some cases the pain on defecation may become excruciatingly severe ; under certain conditions, which will be described presently, the pain may become so extremely severe that a number of alarming general symptoms, such as vomiting, palpitation, great anxiety, and even syncopal and convulsive seizures supervene.

In older medical works the so-called remote symptoms of hemorrhoids play an important rôle. All the manifestations ascribed to this sequence of events are identical with those described above under the heading of Chronic Constipation (compare p. 101). There is no doubt whatever that these symptoms must all be attributed not to the piles, but to constipation, the genesis of which has already been described in another section of this work where the production of these various

symptoms and their dependence on constipation were fully dealt with. It is, therefore, unnecessary to go over this ground again.

As an illustration of the way in which false conclusions are drawn, I cite the following case, observed the very day on which I write these lines : A man of fifty-seven, with organs otherwise normal, has quite well-developed external hemorrhoids, from which a daily, but insignificant, flow of blood takes place, always with stools, which are evacuated with entire regularity. He complains of dizziness for the last few days. He attributes this to the fact that for several days past the usual flow of blood from the hemorrhoids has not taken place. But at the same time constipation had existed several days, and the patient did not perceive that the absence of bleeding was due to freedom from the mechanical irritation of the passing fecal masses. The vertigo was most probably due to the constipation.

A number of *local*, in contradistinction to the remote, symptoms of hemorrhoids are, however, of considerable importance ; these local disturbances are manifested in the clumps of piles themselves and in the mucous membrane of the lower part of the rectum. The latter occasionally shows chronic catarrhal inflammation (proctitis chronica), as a result of which a mucopurulent secretion is poured out and passed in the motions either as pure mucus or mixed with fecal material or with blood—in this way a local condition, called by the older writers mucous hemorrhoids, results.

A variety of changes occur that may be studied in the piles themselves, one of the most frequent of which is prolapse. Here the internal clumps of piles are forced out through the anus, especially when the sphincter ani is relaxed, and may even happen each time the bowels are evacuated. Many of these patients have no difficulty in pressing the prolapsed piles back again, and indeed they often return spontaneously. Occasionally, however, in patients who are obliged to strain excessively to bring about an evacuation of the bowels, it may happen that an internal pile is unexpectedly pushed through the anus, and, should the patient be unable to return it himself, may become strangulated by the sphincter ani when this muscle becomes spasmodically contracted as a result of the local irritation. The pile now begins to swell and turn deep blue, and a serious condition of affairs rapidly develops. The patient suffers agonizing pain, which starts from the strangulated pile, which is exquisitely sensitive when touched ; and at the same time there are a very distressing tenesmus and frequently radiating pain ; occasionally vomiting, hiccup, meteorism, painful micturition and retention of urine, fainting fits, great general prostration, and febrile symptoms appear. The pile may become gangrenous unless it is returned in time, and sepsis has been known to follow gangrene. Inflammation of piles accompanied by fever but without strangulation has also been known to occur. Occasionally the inflammation stops spontaneously, and may even be followed by shrinking and atrophy of the pile ; this is the most favorable issue. In other cases inflammation goes on to suppuration. In the latter case there are fever, usually of high degree, tenesmus,

spasm of the sphincter with severe hemorrhages and often complications in the neighboring tissues, such as periproctitis and rectal fistula. Anal fistulæ very frequently accompany hemorrhoids.

In the differential diagnosis the following conditions must be considered : prolapse of the rectum, syphilitic condylomata, polypi, simple folds of mucous membrane. By careful local examination it will always be possible to make a differential diagnosis between hemorrhoids and all these conditions.

The prognosis of hemorrhoids must be made exclusively from the point of view that the disease is a local affection. When due to subacute causes, which are in themselves curable, a restitution to normal is possible. This refers to piles occurring during pregnancy. From consideration of the various possible causes enumerated above it can readily be understood why the disease does not, as a rule, terminate so favorably—in other words, why a spontaneous cure does not occur. Generally speaking, hemorrhoids, when fully developed, constitute an obstinate affection which causes the patient a great deal of distress. At the same time, the disease cannot be considered as grave or dangerous. Serious danger to the patient arises only when local complications appear, such as inflammation, gangrenous destruction, strangulation, and sloughing, with excessive bleeding.

Treatment.—It is, to a certain extent, possible to meet the causal indications for treatment by avoiding all the factors capable of producing venous engorgement of the hemorrhoidal plexus, and, conversely, by leaving no stone unturned to improve the condition of the general metabolism and of the circulation of the blood. The necessary lines of treatment can be summarized in the three following rules : Avoidance of excesses of all kinds ; regular and sufficient physical exercise ; regulation of the stools. The same indications must be met when hemorrhoids are already fully developed. As a matter of fact, the whole treatment of the " hemorrhoidal state " is limited to a fulfilment of these conditions, provided, of course, local changes in the piles do not call for special treatment.

The diet should be mixed. When the general condition of the patient is otherwise healthy and there are no other definite pathologic conditions requiring special dietetic regulations, he may, generally speaking, be allowed to eat anything and everything. We know empirically, however, that spices and irritating condiments, at least when taken in considerable quantities, should be avoided. The diet should be arranged to suit individual peculiarities, and should be made to fit any special conditions that may be present, such as anemia or plethora. The selection of the different articles of food should be made according to the well-recognized and established principles of the dietetic treatment of these various possible conditions. One very important point, which must not be neglected, is restriction of the quantity of food taken at each meal. It is always better to give frequent meals consisting of a small amount of food than large meals at long intervals.

Alcoholic liquors and beverages containing theobromin (tea, coffee)

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should be permitted only in very moderate quantities, any excess in this direction being carefully avoided. The amount of water ingested should also be limited, and free water-drinking interdicted. Excesses in venery are also considered injurious. It is absolutely necessary that sufferers from hemorrhoids should have plenty of physical exercise, and they should be strictly forbidden to sit down or to stand up for long periods at a time. The latter rule, of course, can be enforced only in those patients who are not prevented by the stern necessities of life from violating this fundamental rule. The best forms of bodily exercise are walking, gymnastic exercises, either at home or in a gymnasium, swimming, etc. Much horseback-riding, on the other hand, is injurious, and the same is probably true of bicycle riding. Oeder recommends supination with the anal region well elevated. This may be effected by means of a pillow or by elevation of the foot of the bed. He claims that this removes blood from the rectum better than any kind of exercise.

One of the fundamental rules in the treatment of hemorrhoids is careful supervision of the bowels. The bowels should be regularly open, and constipation must not be allowed. The same principles govern the treatment in this direction as those already discussed in the section on Habitual Constipation (see pp. 113–121). In order to avoid repetitions the reader should refer to that section for the details. But it is to be distinctly understood that the old-time aperients, like sulphur and rhubarb, and the so-called neutral salts, are particularly well adapted to these cases.

In cases of piles, a certain amount of attention must be given to the hygiene of the anus. Sitting on soft, warm cushions is harmful, and the patients should be advised to use cane and board seats or those made of horsehair or leather. After defecation the cleansing of the anus should not be performed with paper, however soft it may be, but the parts should be washed instead with a soft, moist sponge and then carefully dried. Cool sitz-baths and cold ablutions ought to be performed regularly. When the piles constantly prolapse during defecation, but do not undergo any other change, the patients are usually able to replace them themselves. The best method of doing this is to stoop over forward and to push the prolapsed pile slowly into the rectum with the fingers. It is well to lubricate the finger with oil or with boric vaselin. Wallis[1] approves of the treatment of prolapsing piles, which can be returned, by injection of a 10 per cent. solution of carbolic acid in glycerin into the substance of the pile. This is a prophylactic measure and leads to considerable diminution in the size of the pile, but is not a radical method of treatment.—ED.]

When there is severe local irritation, much tenesmus, exquisitely sensitive piles, or hyperesthesia of the mucous membrane of the lower part of the rectum, the external piles should be treated with lead or zinc ointment, or boric vaselin. It is also a good plan to apply cold compresses. The treatment of the internal piles is slightly different under these conditions. Here suppositories of belladonna, opium, or

[1] F. C. Wallis, *Clin. Jour.*, 1902.

morphin should be inserted into the rectum, or some lukewarm water of from 40° to 50° C., or olive oil should be injected. Rosenheim advises the injection of a few drops of a 0.5 to 1 pro mille solution of silver nitrate, and states that in many cases this injection relieves the pain.

For inflamed piles, absolute rest should be enforced and small packs of ice should be applied locally. Some patients prefer the application of warmth (poultices) and claim that this treatment is more grateful. The application of a few leeches to the rectum is often very advantageous, but leeches should never be applied directly to the piles themselves. In addition, anodyne suppositories may be employed. If a restitution to normal does not occur within a short time, surgical interference is indicated—either incision or removal of the hemorrhoids.

If the patient himself cannot replace the prolapsed piles after they have become strangulated, the medical man must attempt to return them. The method to be employed is as follows : At first ice should be applied to the parts and then gradually increasing pressure should be exerted on the pile. The pressure, however, should never be excessive. In this way it is often possible to first reduce the size of the pile and then to push it back into the rectum. Occasionally it is impossible to perform this manipulation without first placing the patient under chloroform anesthesia. [The application of a solution of adrenalin to irreducible piles when threatened with strangulation has recently been recommended (Bouchard,[1] Mossé).[2]—ED.] The subsequent treatment of these advanced cases of prolapse of hemorrhoids is really surgical. The same applies to the treatment of hemorrhoids that have already become gangrenous.

When the disease is complicated by proctitis, this condition should be treated according to the rules laid down in the section on this affection.

Slight and insignificant hemorrhages do not call for special treatment. When bleeding is severe, the hemorrhage should be stopped by the application of ice-bags or injection of small enemata of ice water ; or the patient should be instructed to take cold sitz-baths. In some cases the injection of so-called styptic solutions of tannin, alum, acetate of lead, or silver nitrate are useful. Recently the treatment of hemorrhage by the application of ointments containing chrysarobin (in external hemorrhoids), or of suppositories containing this drug (in internal piles), has been advised. In very violent hemorrhages tamponing or the injection of *liquor ferri perchloridi* should be employed. If all these measures are found to be without avail, *ferrum candens* should be used, or in extreme cases ligation of the bleeding artery performed.

Occasionally the radical removal of the piles becomes necessary. The indications for this treatment are the following : Excessive and unsupportable pain in the hemorrhoids, with or without fissures, ulceration, or a tendency to recurrent inflammation. Removal is especially indicated in cases complicated by exhausting hemorrhages. (For the

[1] Bouchard, *La Presse Médicale*, Dec. 31, 1902.
[2] Mossé, *Soc. Med. des Hôp.*, Feb., 1903.

details and the technic of this operation the reader should refer to text-books on surgery.)

NERVOUS DISEASES OF THE INTESTINE.

UNDER the heading of nervous diseases of the intestine I include those clinical conditions which are independent and due to perversions of the innervation of the intestine. This definition sharply isolates this group of diseases, and more especially differentiates it from all those symptom-complexes which, though also due to abnormal innervation of the intestine, are really nothing more than symptoms of definite anatomic lesions of the intestinal wall or of more distant organs. I can best illustrate my meaning by quoting the following example : Contraction of the intestine in acute catarrhal enteritis assumes the form of colic, whereas in hemorrhoids it appears as spasm of the rectum, and in cerebromeningitis as boat-shaped retraction of the abdominal muscles. These various forms of contraction, however, are all merely symptoms and cannot be considered independent nervous diseases. They are, in fact, analogous to spasm of the glottis in whooping-cough, or to tonic muscular contractions of the lower extremities in many forms of myelitis, and have just as little claim to be considered independent nervous diseases.

While it is easy to make this differential diagnosis in many cases, it may in others be difficult to draw a hard-and-fast line between independent nervous affection of the intestine and nervous manifestations dependent on definite anatomic lesions either in the bowel or in distant organs. In many cases the neurotic manifestations in the intestine are an independent manifestation of some general neurosis underlying the whole disease. This is shown, for instance, in many of the intestinal symptoms seen in cases of neurasthenia, hysteria, and hypochondriasis. In fact, it may fairly be said that the majority of so-called intestinal neuroses belong to this group.

There are, however, undoubtedly, some diseases of the intestines in which the symptoms are produced exclusively by some functional perversion of the intestinal nerves. The recorded cases of this character are, however, rather few and far between at present, especially those cases in which the clinical records are corroborated by the data of morbid anatomy obtained by autopsies.

It is hardly possible to give a systematic description of the nervous diseases of the intestines. I must confine myself to a clinical description of those various conditions which can be referred to anomalies in the innervation of the intestine. From this point of view it is usual to divide the subject into disturbances of the motor, the sensory, and the secretory vasomotor functions. At the same time special attention should be directed to the fact that it is comparatively rare to find perversion limited to one function alone. As a rule, two, and occasionally three, of the different nerve functions are abnormal at the same time.

Another factor which makes it exceedingly difficult to formulate a

correct judgment as regards the significance of nervous disorders of the intestine is the exceedingly complicated nervous mechanism of the bowels. It is, of course, impossible to give a detailed account here of the anatomy and physiology of the innervation of the intestine. At the same time attention must be called to the fact that the innervation of the intestine is even more complicated than that of the heart. The intestine has motor, sensory, and vasomotor secretory nerves. There are, in addition the nerve plexus of Auerbach and Billroth-Meissner in the intestinal wall, besides fibers from the vagus, the spinal nerves, and from the sympathetic ganglia, which all enter the intestinal walls from without. One page 73 of this work I have given a brief sketch of the exceedingly complicated nervous mechanism of the intestine. *A priori* a scheme could possibly be constructed to explain the different effects and consequently different clinical pictures occasionally seen in nervous diseases of the intestine. It might, for instance, be possible to calculate out the functional disturbances which would appear if the inhibitory or the excitory action of the vagus fibers was arrested; or, if the inhibitory or excitory action transmitted through fibers derived from the splanchnic nerve was stopped; or if the plexus in the intestinal wall itself was affected in some way and its function deranged, etc. I will refrain, however, from doing this because my aim is to avoid all theoretic discussion and to limit myself in this whole review to a description of those perversions only which are based on the solid foundations of clinical and anatomic observation. Unfortunately, the latter, namely, anatomic investigation of well-characterized disease pictures, seems to be very deficient in the case of this particular group of diseases.

It will be seen, therefore, that it is impossible to group the nervous diseases of the intestine from an anatomic or physiologic point of view. In fact, it is not even possible to classify the various nervous diseases of the intestine from a purely clinical standpoint, for many of the different disease pictures merge into one another and have one or several features in common. I shall, therefore, endeavor to arrange the material according to certain important traits which are specially characteristic of certain syndromes.

The following description of the nervous diseases of the intestine will be found to be somewhat disconnected, owing to the fact that some aspects of the nervous affections of the intestine have already been described in previous sections. In order to avoid unnecessary repetition, the reader should refer to the different sections for many of the details.

DISEASES OF THE MOTOR NERVES OF THE INTESTINES.

(a) Peristaltic Unrest (Tormina Intestinorum).—In the review of the peristaltic movements of the intestine given on pp. 70–78, the so-called "rolling movements" of the intestine were briefly mentioned. This peculiar form of motion consists in peristaltic movements of certain portions of the intestine, usually the small intestine, which travel with great rapidity from one end of a segment of the bowel to the other.

At the same time gas and fluid contents are carried along with this peristaltic wave ; in some instances the converse occurs, and the peristaltic wave is excited by the presence of gas and liquid material in the bowel lumen. This form of intestinal movement may occasionally assume the character of a neurosis. Peristaltic unrest of the bowels is sometimes seen in neuropathic subjects, particularly in those with hysteria and hypochondriasis. In these cases the peristaltic unrest of the intestine is one of the symptoms of hypochondriasis or of hysteria, and has the same significance as many other single symptoms of these diseases. Occasionally, however, peristaltic unrest of the bowels is an entirely independent affection, and is the sole pathologic phenomenon seen in subjects who are otherwise perfectly healthy. Nothing definite can be stated as to the prime cause of this condition when it occurs in healthy people. We can merely assume that it is due to increased irritability or increased functional activity of the nervous apparatus which governs the peristaltic movements of the bowel. Apart from the facts that all possible disturbances capable of producing peristaltic unrest are absent in these cases, the theory that I have advanced seems to be strengthened by certain experiments ; for, under " Movements of the Intestine," a number of my observations made in the course of experiments on animals were given which showed that extremely violent rolling movements of the bowels may occasionally be suddenly arrested, apparently by nervous inhibition.

Peristaltic unrest of the bowels is almost exclusively seen in the small intestine, the movements usually ceasing at the ileocecal valve. In peristaltic unrest of the bowels, moreover, evacuation of the intestinal contents rarely occurs, a fact which also seems to point to limitation of the abnormal motor activity to the small intestine, for if the large intestine was also involved, defecation would certainly occur.

Clinically, peristaltic unrest of the bowels is manifested by a variety of loud, rolling, gurgling, squelching, and squeaking noises in the abdomen, of varying intensity, which may be exceedingly loud at one time and faint at another. As a rule, these noises can be heard at some distance from the patient, so that everybody in his vicinity notices them. This is usually a source of great discomfort to the patient and may be so mortifying that sufferers from this disease are unable to attend to their business, even though the condition is essentially without danger. They rarely suffer any pain, and when they do, I am inclined to assume that it is not so much due to the presence of a mixed motor-sensory neurosis, but simply to the fact that the violent peristaltic movements of the bowels are associated with tetanic contractions of the muscular walls of the intestine, which in their turn cause the pain. The general and undefined sensations, on the other hand, which many of these patients complain of are undoubtedly due to an increase of the peristaltic movements of the bowel-wall. The latter movements when they remain within physiologic limits are never perceived by the patients themselves. When the abdominal walls are very thin, the movements of the intestines may occasionally become visible and palpable. Another

symptom that is occasionally noticed is eructation. This is specially common when there is peristaltic unrest of the stomach-wall in addition to peristaltic unrest of the bowel. Rosenheim mentions the fact that occasionally in peristaltic unrest of the bowel feculent vomiting occurs as a direct result of antiperistaltic movements. I am unable to corroborate or to deny this statement, as I have had no personal experience in the matter.

Tormina intestinorum either appear at quite irregular intervals, and last several minutes or longer, or they follow some distinct provoking cause, in particular emotional disturbances of various kinds, the ingestion of food, or menstruation. Occasionally peristaltic unrest of the bowels persists for many hours and may even appear during sleep.

The **diagnosis** can always be made without difficulty. Unless the patient is examined very carelessly, simple tormina intestinorum should never be confused with the increased peristaltic movements occasionally seen in stenosis of the bowels. A mistake of this kind might, of course, be fraught with dangerous consequences.

The **prognosis** is favorable, the affection being disagreeable, but not dangerous.

Treatment of the condition in neuropathic subjects should be directed toward the underlying disorder. If it is an entirely independent condition, treatment should also be directed toward improving the tone of the whole nervous system. At the same time, it is always well to look for incidental causes which may be responsible for the occurrence of individual attacks. Such treatment includes a general régime similar to the one employed in nervousness—viz., mild hydropathic procedures, a course of treatment in some watering-place where the patient can drink indifferent waters or bathe in them, electric baths, and general faradization. Internally, I advise the use of arsenic and possibly of bromids. During the night Priessnitz compresses may be applied to the abdomen. Symptomatically, carminatives as well as valerian, and in cases in which the peristaltic movements of the bowels are exceedingly violent, opium may be given internally.

[Boas [1] obtained good results in one case with codein and belladonna. Chloral hydrate, 15 grains, in gruel at night has been recommended by Rosenheim [2].—ED.]

(b) Nervous Diarrhea (Diarrhœa Nervosa).—Peristaltic unrest of the bowels is closely allied to another form of intestinal neurosis, namely, nervous diarrhea, which is relatively frequent and appears, as a rule, as an independent condition. In nervous diarrhea, in contradistinction to peristaltic unrest, the exaggerated peristaltic movements occur not only in the small intestine, but also in the large intestine ; as a matter of fact, to judge from the character of the dejecta, the peristaltic movements may be limited to the colon in some cases. In addition we must assume that in nervous diarrhea an increased transudation of fluid occurs into the lumen of the intestine ; and it is quite probable that

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[1] Boas, *Diseases of the Intestines*, American translation by S. Basch, 1901.
[2] Rosenheim, *Path. u. Therap. d. Krankh. d. Darms*, p. 492.

this transudation in its turn is due to nervous influences. In no other way is it possible to explain how an individual who is perfectly healthy may suddenly be seized with an attack of diarrhea after some mental shock, etc., and pass thin liquid stools very shortly afterward, and this when the motions were solid and formed before the psychic shock occurred.

As I have already given a description of nervous diarrhea both from the clinical and the therapeutic point of view on p. 126, I must refer to these portions of the book for the details. The reader should also refer to the case reported by Emminghaus, which will be described in the paragraph on Paralysis of the Intestine.

(c) **Spasm of the Intestine (Enterospasmus).**—Much remains to be explained as regards enterospasm ; it is clearly a difficult matter to clear up some of the obscure points, since there is very little postmortem material available for the study of this disease. In the first place it is not an easy matter to clearly define this condition. In general, enterospasm means tonic contractions of the muscular coats of the intestine, leading, as a result, to narrowing or complete occlusion of the intestinal lumen ; these contractions involve both the circular and the longitudinal muscle-fibers of the bowel-wall. This is the view adopted by the majority of clinicians, and is also adopted in text-books on intestinal diseases (Rosenheim, Fleischer, A. Pick, etc.) and in other writings.

Generally speaking, this definition is correct, and it may be well to adhere to it. At the same time we must distinguish several degrees of contraction of the intestinal musculature, for, as will be presently seen, the varying intensity of the contraction produces different clinical pictures. A few statements can be made as to the severity of the contractions of the bowel-wall. Our information in this respect has been gained in different ways. I was able to produce the most violent contractions in the course of my experiments when the mucosa of the intestine was irritated by a powerful stimulus ; this, for instance, occurred during the production of artificial acute enteritis, and was also produced when the serous covering of the intestine was artificially irritated by touching it with a crystal of salt (sodium chlorid). In all these experiments the lumen of the intestine disappeared completely and the bowel was converted into a hard, round, solid cord, pale in color. It is probable that the intestine of human subjects behaves in the same way in cases of acute and violent enteritis, and that the spastic contractions involve only certain parts of the intestine, and only temporarily ; it may also be assumed that similar conditions occur in enterostenosis ; in fact, in this disease palpation proves that the intestine is temporarily converted into a solid cord in some parts of its course. In all these cases we are dealing with contractions *ad maximum*, with the strongest degrees of tetanus. The same applies to the rigid form of intestinal contraction so frequent in lead colic, which, combined with tetanic contractions of the abdominal muscular walls, leads to the retracted and flattened condition of the abdomen. This form of enterospasm is also accompanied by violent colicky pains which are analogous to the pain experienced in tonic contractions of other muscles of the body—for example,

in the muscles of the calf in so-called cramps. This genuine colicky pain is the direct result of the muscular contractions, and is directly produced by compression of the sensory nerve-fibers contained in the muscle tissues that are undergoing contraction.

There is another and entirely different form of contraction which is seen in certain diseases of the brain, particularly in meningitis ; here, too, the contractions of the intestinal musculature are more or less diffuse and may be accompanied by boat-shaped retraction of the abdomen. When a case of this kind is examined postmortem, it will be found that the affected loops of intestine contain no air whatever, and that they are soft and flattened and occasionally pressed into triangular shapes by mutual pressure. This appearance is, of course, seen only when the retraction of the abdomen has persisted until death ; the flattening of the different loops of intestine into such characteristic forms can have occurred only during the life of the patient, and shows beyond doubt that *intra vitam* the different loops of the bowel were not contracted into solid, rigid strands as in the form described above, but were soft and flaccid, as seen after death. This view is borne out by the fact that this form of enterospasm, as seen, for instance, in meningitis, is never accompanied by pain, for sufferers from meningitis (unless, of course, certain complications exist) never complain of colicky pain in the abdomen.

Spastic contraction of the intestine, as I have already intimated, may be either local, and limited to some one or other portion of the bowel, or it may be general or at least involve large portions of the intestine. There are, in addition, certain special types of intestinal contraction, such as proctospasm, which, as a rule, are very strictly localized.

Analysis of the etiologic factors causing enterospasm shows that in the great majority of cases it is merely a symptom, a concomitant phenomenon, or at best the effect of local intestinal changes or occasionally of more remote causes involving organs other than the bowel. This applies to the great majority of cases of local as well as of general enterospasm. In order to avoid repetition, the reader should refer to the account given on pages 148–150 in the section on Colic of the Intestine, where attention was called to a large number of primary causes which may produce colic. What has been said there applies with equal force to enterospasm, since colic is in general merely the result of violent local tetanus of the intestine. The general condition of contraction of the bowels seen in cerebral meningitis is also merely one of the symptoms of the brain affection.

In lead colic enterospasm occupies a more independent position in the clinical picture. Since, however, enterospasm is not the prime nor the most important factor even in this condition, it will be referred to again below when considering neuralgia of the mesenteric plexus.

As a matter of fact, general observation shows that enterospasm probably never occurs as an independent neurosis nor as the only intestinal manifestation in subjects who are otherwise normal and healthy ; to judge from personal experience I should consider it very rare indeed,

as I have never seen a case. The nearest approach to this clinical form of enterospasm that we see occurs in neuropathic, hypochondriac, or hysteric subjects, where enterospasm occasionally occupies such a prominent place in the clinical picture and appears to be such an independent affection that it might be regarded as a primary neurosis of the intestine. Strictly speaking, however, enterospasm must be omitted from our category of independent morbid entities; for enterospasm is never, not even in hysteria, nervousness, neurasthenia, or hypochondriasis, so completely disassociated from the syndrome of the underlying neuroses that it deserves the dignity of an independent clinical entity; it may even be said that it is less independent in character than so-called nervous diarrhea, for the latter, though rarely, still occasionally does appear as an independent disease.

[While admitting that primary spastic conditions of the intestine are very rare, Hemmeter[1] has no doubt that they do occur, and quotes 3 cases under his care to support this contention.—ED.]

In view of the fact, however, that enterospasm occasionally and for a time at least may dominate the general clinical picture in some neuropathic persons, a short sketch of this affection may be given.

Ewald, in his *Diseases of the Intestines*, rightly remarks that the clinical picture of an apparently independent nervous enterospasm may be simulated when in reality we are confronted by a preataxic irritational manifestation of many years' duration in the course of a gradually developing tabes dorsalis. He cites a case in point.

Enterospasm in neuropathic subjects is, as a rule, or, in all probability always, local and limited to certain circumscribed portions of the intestine. To judge from the various clinical pictures which may be presented in this disease, the process may involve either the large or the small intestine. The direct effect of enterospasm is stasis of the bowel contents and constipation. When the process is confined to the lower part of the colon or the rectum, the dejecta have a peculiar form, and are flattened and ribbon-shaped or resemble sheep's dung, or are pencil-shaped; on page 81 and on page 369 these peculiar anomalies in the form of the feces have already been described under the heading of Stenosis Feces. In addition to producing constipation, local enterospasm may also cause strictly localized protrusions of the abdominal wall; the latter are caused by the accumulation of gas above the stenosed area of the bowel and by the condition of local meteorism above the spastic stenosis that develops as a result; occasionally, in fact, meteorism does not remain local but becomes diffuse. Cherchevsky reports a case of enterospasm in which the patient actually vomited fecal matter during the attack; he brought up a piece of formed fecal material 3 inches long; this author calls the state he has described *ileus nervosus*. Strauss details a case in a young man, twenty-nine, hysteric, on whom two operations had been performed for suspected organic stenosis of the intestine. He forms the following conclusions: "It is possible that one or another of the following factors dominated at various times: entero-

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[1] Hemmeter, *Diseases of the Intestine*, 1902, vol. ii.

spasm, enteroparalysis, paresis of the abdominal muscles, abnormal situation of the diaphragm, perhaps also deglutition of air with simultaneous incontinence of the pylorus." This is an instructive illustration of the great difficulties sometimes encountered in diagnosis. It also justifies the doubts expressed by many, particularly surgeons (*e. g.*, Kocher), that ileus occurs with mere spasmodic contractions of the intestine. Recent observations by Heidenhain and by Schloffer, among others, seem to demonstrate that enterospasm alone may cause the complete clinical picture of intestinal occlusion. Laparotomy showed either no changes whatever or else distended coils, which, without any mechanical obstruction, passed directly into contracted ones. See page 381 for the vomiting of formed feces that occurred in these cases.

Attacks of enterospasm are generally attended with pain. Their occurrence is variable—they come and go, lasting from minutes to hours. The location is equally variable, as is also the protuberance of the local meteoristic distention. Moreover, there may exist other neuropathic affections.

The following instructive case reported by Talma may be quoted here by way of illustration :

The patient was a man of twenty years, nervous by heredity, who had had repeated attacks of very severe colic, which presented the following features : The disagreeable sensation in the abdomen that he suffered from continuously gradually increased in severity in the course of a few days, while at the same time the bowels became very confined. The feeling of discomfort in the abdomen gradually increased until it became actual pain ; finally constipation became complete. At the same time the patient complained of a very disagreeable spasm in the urinary bladder and in the testicles. The surface of the abdomen was generally retracted, without any real contraction of the abdominal muscles. In the left hypochondrium, however, the surface of the abdomen projected above the level of the belly, and in this region there were a few tumor-like swellings, one of which was spherical, while the others were more sausage-shaped. These tumors continuously changed in consistence, so that at different times a different degree of resistance to the palpating fingers was noticed, but never became completely soft. The percussion-note over the tumors was tympanitic, loud, and somewhat low ; at the time of the greatest resistance in these tumors the percussion-sound became less loud, higher, and non-tympanitic. Through the length of the sausage-shaped tumor continuous peristaltic waves were seen to pass ; the swelling, therefore, was formed by loops of intestine, certain portions of which were occluded by spastic contractions of the bowel-wall ; between the different points of closure of the lumen contents of the bowel accumulated and formed the palpable masses, or, in other words, constituted the tumor. The pain increased and decreased in severity according to the spasm. Occasionally small quantities of urine were passed with a violent feeling of tension in the urinary bladder. Pressure over the aorta above the umbilicus always produced violent pain. In spite of free purgation it was impossible to get the bowels open ; while morphin did not relieve the pain and distress. After the administration of antipyrin, however, all the symptoms disappeared, and the bowels even acted. At the end of about a week the attack recurred and lasted for a number of days, again to disappear after the administration of antipyrin and of quinin ; in addition, the abdomen was packed in thick woollen blankets. The patient during all this time was given light and easily digestible food. The clinical picture persisted in this way for a number of months, gradually decreasing in severity. During all this time the abdominal sympathetic was tender on pressure, although it gradually decreased as the disease dragged its course through several months ; pressure on the sympathetic invariably precipitated an attack characterized by the old disagreeable sensations of which the patient had for so long complained. An attack of gastric spasm complicated by vomiting also appeared intercurrently.

Talma, relying on the experimental researches bearing on this subject, assumed that his patient was suffering from excessive irritability of the celiac ganglion, and advanced the theory that in all cases of this character there is excessive irritability of this ganglion. For it is known that slight irritation of this ganglion inhibits the normal movements of the muscular coats of the bowel, and that, on the other hand, severe irritation of the ganglion produces violent movements of the intestinal wall; the latter movements, however, appear in the form of incoördinate spasmodic contractions of the muscular coats, either locally, leading to the complete obliteration of the lumen of the bowel in certain circumscribed portions of the intestinal tract, or, more generally, leading to tonic contraction of two more or less remote portions of the bowel and anastaltic and catastaltic to-and-fro movements of the bowel contents arrested between these contracted portions of the intestine.

It may occasionally be found to be a difficult matter to arrive at a correct diagnosis of conditions of this character, since the clinical picture produced resembles organic stenosis of the bowel so strikingly; in making a diagnosis, therefore, it is particularly important to pay attention to the possibility that latent stenosis of the bowel may be present as the result of some past peritonitis which may have been overlooked or forgotten.

Enterospasm appearing in the course of lead-poisoning and lead colic presents quite a different local picture. Here tonic contractions of the bowel-wall occur throughout the course of the whole intestine, but particularly in the small intestine. As a result, the abdomen becomes flattened and retracted so as to form a boat-shaped surface, often combined with an additional rigid contraction of the external muscles of the abdominal wall. Another result of the tetanus of the bowels (perhaps also a result of certain other conditions, such as direct irritation of the inhibitory fibers of the splanchnic nerves) is obstinate constipation, which frequently appears in the course of this disease and usually accompanies attacks of lead colic; lastly, there is also violent colicky pain. In view of the fact, however, that the various factors concerned in producing attacks of lead colic are extremely numerous and complicated, and that it is quite possible that the pain may be due to neuralgia of the mesenteric plexus, this syndrome does not constitute a pure clinical picture of violent diffuse enterospasm.

There is a special form of enterospasm confined to one portion of the bowel alone which requires special mention—viz., proctospasm, or spasmodic contraction of the sphincter muscle of the anus. This spasm of the sphincter is never an independent nor a primary phenomenon, but is always reflex and, as a rule, is produced by some local disorder of the rectum or the urinary bladder, and in some instances it is one of the manifestations of disease of remote organs other than the bladder or rectum, or of some general disease. The diseases of the rectum which produce proctospasm are particularly hemorrhoids, anal fissure, proctitis, and ulceration of the rectum; also certain diseases of the prostate gland,

the urinary bladder, and the uterus. The general diseases which may set up proctospasm are tabes dorsalis, hysteria, and very advanced degrees of neurasthenia.

Proctospasm causes complete closure of the anal orifice, very violent pain, which may be so excruciating and overwhelming in severity that fainting occurs. In many cases the anus is kept so tightly closed that it is impossible even to insert the finger by force; the pain and sensitiveness may be so acute that merely touching the anal region may cause considerable pain. During an attack of proctospasm it is manifestly impossible for the patient to perform the act of defecation.

It is quite unnecessary to enter into a detailed discussion of the prognosis of enterospasm, as it is essentially dependent on the character and the duration of the primary disease responsible for this condition. The same applies to the treatment of enterospasm, for this coincides with the treatment of the causal factor or factors. The prognosis and treatment of enterospasm due to some neuropathic predisposition may be briefly referred to. Experience shows that in this form of the affection all measures which irritate the intestine or which, without directly irritating the bowel, increase its irritability to other stimuli must be avoided; for this reason such therapeutic procedures as massage, electrization of the abdomen, drastic drugs, cold, and irritating irrigations of the bowel must all be omitted from the treatment. Warmth in any form, on the other hand, is grateful and may be employed, as baths, warm compresses, abdominal binders, warm carminative infusions given internally, and warm irrigations of the bowel. The best remedies in this condition are opium, morphin, and belladonna, given internally, subcutaneously, or in the form of suppositories, while antipyrin, phenacetin, and quinin should also be tried. In cases complicated with very obstinate constipation irrigations of the rectum with oil are particularly useful; these irrigations should be given according to the method recommended by Kussmaul and Fleiner—viz.: injection of 400–500 grams of the best olive oil heated to 104° F. and allowed to flow into the rectum in the course of twenty minutes; the irrigations to be given daily and to be repeated until spontaneous evacuation of the bowel contents occurs.

(d) Paralysis of the Intestine (Paralysis Intestini).—In a subsequent section (pp. 585–593) paralysis of the intestine will be considered from a different point of view—namely, in connection with stenosis and occlusion of the bowel; for the details of paralysis of the intestine following these conditions the reader is expressly referred to that section. Here it need only be repeated that paralysis of the intestine ultimately leading to the development of the symptom-complex called ileus paralyticus may occur without any discoverable anatomic lesion of the bowel; in the section on Stenosis and Occlusion of the Bowel an explanation of how this can occur was given and it was shown that in all probability the condition is the result of severe reflex irritation of the inhibitory nerves of the muscular coats of the intestine, particularly when there is some injury or inflammation in the vicinity

of the intestine which does not necessarily involve the bowel itself. To the same class belong those forms of intestinal paralyses seen in hysteric subjects who suffer from meteorism (see p. 141); here the meteorism is in all probability due to sudden paresis of the muscular coat of the bowel and is only one of the symptoms of the protean symptom-complex met with in this disease.

There is an independent form of chronic atony of the bowels leading to habitual constipation which may be included in this category if so desired; this state has been described on pp. 102, 103. The disease is in a measure a subparetic condition, so that it might fitly be included under paralysis of the bowel; it is not customary, however, to do so.

Paralysis of the sphincter calls for special notice, although it is never an independent nor a primary disease of this portion of the intestinal tract. Paralysis of the sphincter occurs in some diseases of the brain which are accompanied by loss of consciousness and in a variety of other drowsy and comatose states; in all these conditions the paralysis of the sphincter muscle is one of the symptoms of the general disturbance of muscle innervation. It is also occasionally seen as one of the characteristic symptoms of certain spinal diseases. In addition, however, paralysis of the anal sphincter may be one of the symptoms of a variety of local affections of the rectum, as, for instance, fissure, hemorrhoids, and similar lesions involving the lower portion of the bowel. Other conditions, like proctitis or fecal accumulation in the rectum, may also lead to paralysis of the sphincter muscle, when they—and this applies also to the lesions mentioned above—produce violent and persistent tenesmus by reflex irritation, since long-continued tenesmus must ultimately lead to fatigue followed by paralysis of the sphincter. The symptoms of paralysis of the sphincter vary according to the degree and the severity of the paralysis. At first patients may still be able to retain the bowel contents at will; as soon, however, as the slightest pressure is exerted, the bowel contents, particularly gases and fluid fecal matter, are passed through the sphincter; whenever the patient coughs or sneezes or laughs or performs some other involuntary act of expiration, the motions may pass through the paretic sphincter without meeting any resistance to their exit. In some cases the motions may pass when the patient urinates or when he walks about. Lastly, when the sphincter muscle is completely paralyzed the contents of the bowel are passed at once directly they enter the lower part of the rectum, whether the patient makes any voluntary or involuntary effort at evacuation. In many of these cases the patients are not aware that they are emptying the bowels, for they are quite unconscious of the passage of feces through the anal orifice; this occurs when the rectal mucosa is anesthetic, while the sphincter is also paralyzed (" *sedes involuntariæ* "). Lastly, in very advanced degrees of paralysis of the sphincter ani the anus is widely open at all times, so that two or three fingers can be inserted into the rectum without meeting resistance; the outside of the anus is then frequently excoriated, owing to the irritation exerted upon the circumanal tissues by the bowel contents which are constantly

oozing from the patent sphincter. The treatment of paralysis of the sphincter ani consists primarily in keeping the parts as clean as possible ; the rectum itself should be kept perfectly clean and overdistention of this portion of the bowel carefully avoided (the treatment of paralysis of the bowel in general has already been discussed on p. 103). Treatment must also be directed to the cure, if possible, of the primary disease responsible for the local muscular paralysis. In addition, local treatment of the paresis of the sphincter muscle should be undertaken on the same lines as in the treatment of other forms of muscular paralysis ; the measures at our disposal for this purpose are essentially local and consist of electric treatment, massage, etc.

It would be possible to act with much more confidence in the treatment of motor diseases of the bowel if there were more detailed anatomic data as to the conditions present ; unfortunately, we are essentially limited to a number of clinical observations and to some physiologic experiments which are of use in explaining and interpreting the latter, while postmortem observations are scanty and insufficient to throw much light on this class of disorders. While there are numbers of clinical cases reported of motor paralysis of the bowel, very few of them are supplemented, as they should be, by the descriptions of postmortems. A beginning has, however, been made in this field ; two reports recently communicated by Emminghaus deserve special attention, as they supplement this long-felt want of combined clinical record and postmortem examination. The reports of Emminghaus are models of careful and methodic analyses of cases of this character and encourage the hope that soon our positive knowledge of this subject will be greatly amplified and that an anatomic basis for the proper nervous diseases of the intestine will be found. One of his cases was a woman who had suffered for many years from obstinate habitual constipation ; at the autopsy a dense mass of pleural adhesions was found in the thoracic cavity exactly over the origin of the right *nervus splanchnicus major ;* this adhesion was of long standing and was strictly confined to this area on the right side ; it was very thick and tough in consistence, shiny and white in color. The right splanchnic nerve was different in appearance from the left one, and contained a much smaller proportion of bundles of nerve-fibers ; on cross-section the left splanchnic nerve showed four large and fourteen small intact bundles of nerve-fibers, whereas the altered nerve on the right side showed four large, but only two smaller, fasciculi. In Emminghaus' other case the patient was also the subject of habitual constipation, which three weeks before death was succeeded by a mild form of diarrhea with two or three motions a day. These motions were pultaceous, light colored, and contained a considerable proportion of undigested food remnants. In this case also an encapsulated pleuritic exudate was found on the right side of the thorax, consisting of fibropurulent material and covering the origin of the right splanchnic nerve. In this case several bundles of the affected nerve were again found in a

condition of atrophy, some of them being in an advanced state of atrophy, others being totally atrophied.

Emminghaus explains the constipation in the first of his two cases as follows : Accepting the theory formulated by Basch and Ehrmann as to the innervation of the intestine, he argues that the constipation may have been due to obliteration of those fibers of the splanchnic nerve which stimulate the longitudinal muscular coat of the intestine ; these fibers of the splanchnic enforce the action of the fibers coming from the mesenteric plexus, for both supply the longitudinal fibers of the intestinal wall. In the second case reported by Emminghaus the occurrence of the diarrhea is difficult to explain ; it is possible that this diarrhea was merely an accidental and terminal complication ; it is also possible, however, that another variety of the fibers of the splanchnic nerve was degenerated in this case, leading to looseness of the bowel motions in some indirect way.

The reports and investigations as to anatomic changes occurring in the nerve-plexus of the intestinal wall are somewhat more numerous in cases of intestinal motor neurosis. Thus Jürgens, Blaschko, Sasaki, Scheimpflug, have all published researches which have already been considered. Unfortunately, even these data do not justify any far-reaching conclusions or any definite explanations as to the relations existing between degeneration of the plexus of Meissner and of Auerbach and changes in the motility of the intestine ; as a matter of fact, it is quite impossible at present to make any definite statements whatever on this point.

SENSORY NERVOUS DISORDERS OF THE INTESTINE.

Under normal conditions we are not in any way conscious of the processes going on in the intestine, as these processes produce no sensations. In the light of our present knowledge of the physiology of intestinal function it seems very probable, moreover, that the functions of secretion, absorption, and peristalsis are carried on without the participation in the process of any sensory nerves. Consequently reduced sensibility of the intestine, even complete anesthesia, may be present without attracting the patient's attention and without producing any clinical symptoms whatever. As a matter of fact, no clinical symptoms can be attributed to anesthesia or to a condition of diminished sensation of the intestine ; the condition of anesthesia of the intestine, therefore, cannot be diagnosed. It is, therefore, also manifestly impossible to prove positively that such a condition ever actually exists ; it is, however, not improbable *a priori* that such a state does occasionally appear, particularly in hysteric subjects.

Only one part of the intestine is an exception to these statements which apply to the rest of the bowel—viz., the lowermost portion of the rectum. The sensibility of this part of the bowel is of physiologic importance and plays a physiologic rôle in that the irritation exerted on the lower portion of the rectum by masses of accumulated fecal matter

in the rectal ampulla produces a sensation that is conveyed to the sensorium and gives notice that the bowel-contents must be evacuated ; at the same time the sensory impression transmitted starts the whole motor mechanism of defecation. When for any reason the sensory irritation transmitted from the rectum is repeatedly suppressed, hyperesthesia of the nerves of the rectum may develop, followed as a natural result by retention of feces. Further, some pathologic conditions, chiefly of the spinal cord, may produce anesthesia of the rectum. Since these patients are incapable of feeling the passage of fecal matter through the rectum and anus, and, further, as the reflex contraction of the sphincter muscle is inhibited, involuntary passage of feces occurs.

Hyperesthesia of the intestine is of far greater importance than the hyperesthetic conditions just described, and is much more frequent. Everything that has been said in the preceding paragraphs as regards the deficiency and incompleteness of our knowledge of the motor function of the intestinal nerves and the diseases of these motor nerves ; everything that has been said as to the scantiness of our knowledge of the physiology and anatomy of *these* nerves, applies with equal force to the diseases of the sensory nerves of the intestine and the condition of hyperesthesia of the intestine which is occasionally met with. It may be added that very little is known as regards the combination of hyperesthesia and other functional disorders involving other nerve-fiber tracts.

One point I wish to emphasize particularly : Among the so-called " sensory neuroses " of the intestine colic is usually included, and, in fact, occupies a leading position in this category. In my opinion this is wrong. Colic, in the first place, is not a simple sensory neurosis ; in the second place, it is not a primary neurosis ; and in the third place, it is not a sensory neurosis at all. On pp. 147, 148 of this work I have given my views on this subject, and have called attention to the fact that colic is always a secondary symptom, the pain in every case being produced by the tetanic contractions of the intestinal musculature. For this reason the pain of colic cannot be included among the sensory neuroses of the intestine ; we are here confronted with conditions that are perfectly analogous—for instance, to cramps in the calf ; in this affection the pain is not regarded as a sensory neurosis of the sciatic nerve and attributed primarily to an affection of this nerve, but the pain is recognized as secondary and due to the violent tetanic contractions of the muscles of the calf ; the pathogenetic relationship, therefore, between " cramps " in the calf and the pain of colic is apparent. In the following paragraphs " colic, colicky pain " will not be described at all ; the reader should refer to p. 147 for a description of this condition.

Even less is known of the pathogenesis of true sensory diseases of the intestine than of the motor affections ; the most that can be done is to give a general sketch of the clinical features characteristic of this class of disorders. In doing so I shall attempt to group the different forms of intestinal neuroses according to the same principles that governed the grouping and classification of the motor affections.

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(a) **Hyperesthesia of the Intestine.**—Digestion and all the processes connected with this function are carried on in normal individuals without producing any sensation whatever. This point has already been mentioned, as well as the fact that in many cases of neurasthenia, hypochondriasis, and hysteria the patient may be conscious of certain abnormal sensations in the intestine during the act of digestion. This abnormal state may be due either to certain changes in the brain or possibly to increased irritability and sensibility of the sensory nerves of the intestine. The disagreeable sensations experienced during digestion or occasionally spontaneously after some violent psychic emotion or shock are a feeling of fulness, burning, stabbing, tearing, and sensations as if the ingesta were moving about in the abdomen; lastly, in some, hypochondriacal subjects, delusions may develop.

Occasionally there is regional hyperesthesia manifesting itself as a feeling of soreness or even of direct painfulness limited to certain loops of intestine. The rectum and the region around the anus are special points of predilection (according to Peyer, especially in sexual neurasthenics) for the development of a great variety of abnormal sensations. Among these abnormal feelings I may mention the sensation of tenesmus and the feeling as though some foreign body were impacted in the rectum even when the rectum is anatomically normal and contains no fecal material whatever; other patients complain of a feeling of tickling and burning, stabbing and cutting, pulsating and throbbing, pressure and fatigue, often combined with voluptuous sensations.

(b) **Nervous Enteralgia (Neuralgia Plexus Mesenterici).**—This term is employed to describe pain in the abdomen or in the intestine which is secondary and not due to tetanus of the intestinal musculature, like colic, but due to a genuine neuralgic affection of the nerves of the intestine, and appears as a primary affection with all the characteristics of neuralgic pains. This pain is the direct manifestation of increased sensibility and irritability of the sensory nerves of the bowel. In a previous section, on pp. 153, 154, I called attention to this form of intestinal pain and placed it in juxtaposition to the pain of colic; in the same place I mentioned the fact that nothing definite is at present known as to the particular nerve-fiber tracts involved in this process; I may supplement the remarks made in the paragraphs mentioned by the following considerations: It is probable that nervous enteralgia may be localized in two different anatomic situations; while it is impossible to prove this theory absolutely and there are no cogent reasons to assume that this is actually the case, there is, nevertheless, much clinical evidence to show that nervous enteralgia may either involve the whole area of the mesenteric plexus or, in some cases, only isolated sections of the area of distribution of the nerves emanating from this plexus. Here there is an analogy, with neuralgia involving the leg, for a form of neuralgia involving the whole sciatic area can readily be differentiated from a more localized form of neuralgia of the lower extremity involving only a portion of the sciatic area, as, for instance, neuralgia plantaris or suralis. It is highly probable that in intestinal

neuralgia the fibers of the splanchnic nerves are chiefly involved in the process.

In discussing the etiology of nervous enteralgia I have mentioned that the disease may frequently appear in neurasthenic and hysteric subjects and hence appears to rest on a neuropathic basis, but that, on the other hand, it may also occasionally occur as an independent neurosis in the form of an independent idiopathic nervous intestinal pain. I have seen cases in which chronic nervous enteralgia appeared as an entirely independent affection in subjects who were otherwise perfectly healthy; it is probable that in such cases we are dealing with a continuous state of neuralgic hyperirritability; we must assume that the latter develops from some primary local cause which is located in the intestine, and that it persists even after this primary cause is removed; in this way the development of a continuous and chronic state of neuralgic sensitiveness without any demonstrable local cause in the intestine can be explained.

Many accidental causes may, in addition, favor the development of " enteralgia," the majority of them being localized in the bowel. It is probable that all these local causes correspond essentially with those enumerated on pp. 148, 149 among the possible local causes of intestinal colic. In other words, the same causes as produce colicky pain seem to precipitate enteralgic attacks. I believe that attacks of colic are pathogenetically in no way related to attacks of enteralgia, and that nervous enteralgia must be discussed quite separately from the former affection; consequently we may say that the same causes that precipitate attacks of colicky pain can also independently, under favorable conditions, precipitate an entirely different form of intestinal pain—viz., nervous enteralgia.

One of the most frequent, and I may say the most important, primary causes of neuralgia of the mesenteric plexus is lead-poisoning; the general clinical picture of lead-poisoning appears in practice as lead *colic* and represents the totality of clinical symptoms produced by saturnine toxicosis. It is impossible to give a complete description of this condition here, and I must limit myself to making a few remarks on that symptom of the disease with which this section deals—namely, the pain:

Many years ago Eulenberg and Guttmann advanced a theory and an explanation of the general disease picture observed in lead colic that I am able to indorse fully. I have had a large and varied experience in this disease, and have, in addition, studied the enormous mass of literature that has been written on the subject, and all my studies lead me to agree in all essential points with the views enunciated long ago by the two above-named authors. This view essentially amounts to this, that the pain of lead colic—*i. e.*, that symptom that dominates the clinical picture during the attacks—is in the main a genuine neuralgic pain; in other words, neuralgia of the mesenteric plexus. It is possible for true colicky pain—that is, pain produced by tetanic stiffening and contraction of certain loops of intestine—to appear at the same time as

the other neuralgic pain, but it is not absolutely necessary that this should be the case. It is a well-known fact that occasionally the abdomen is not retracted at all during the attack of pain, even though the pain be severe; and it may even be found that the intestinal walls are not only not contracted, but, on the contrary, somewhat distended. It will be seen, therefore, that in cases of this character the name "lead colic" is far from appropriate. As a rule, two factors are at work at the same time in attacks of lead colic, and are both actively concerned in the production of pain—viz., in the first place, hyperesthetic irritation of the mesenteric plexus; in the second place, tetanic contraction of the intestinal musculature with the colicky pain which is the ordinary consequence of such contractions. There are, in addition, cases in which the pain is undoubtedly produced by the neuralgic condition of the mesenteric plexus alone; on the other hand, it is quite impossible to prove the converse—viz., that attacks of saturnine "colic" ever occur without some purely nervous enteralgia. It may be considered as positively established that the tonic contractions of the intestinal walls are the result of the direct action of lead on the musculature of the parts. It is equally well established, on the other hand, that lead may act locally and directly on very many parts of the nervous system. While changes in the mesenteric plexus and in the splanchnic nerves have not been discovered in a sufficiently large number of cases of saturnine intoxication, and while those cases that have been found have not been studied with sufficient care to enable us to draw any far-reaching conclusions, I am, nevertheless, strongly inclined to the belief that in lead-poisoning direct alterations of the nervous system are present. From our present knowledge of the subject it may, I believe, be concluded that the nervous elements of the mesenteric plexus and the splanchnic nerves are changed by the direct action of lead. [As the result of giving acetate of lead hypodermically to animals, Mosse[1] produced symptoms corresponding to those of lead colic, and found marked changes in the ganglion-cells of the abdominal sympathetic ganglia.—ED.]

It is reported that genuine visceral neuralgia involving the area of distribution of the superior mesenteric plexus of nerves may also occur in the course of arthritis uratica; the very small number of cases of genuine gout in my practice prevent me from expressing any definite opinion on this question on the basis of my relatively limited experience in this field; I am not prepared either to deny or to corroborate this statement. [Hemmeter has satisfied himself on three occasions of the existence of gouty enteralgia, and suggests that it may be due to the action of uric acid salts on the sensory nerves of the intestine.—ED.]

In conclusion I may mention those forms of neuralgic pains of the intestine that occur in the course of certain spinal diseases, especially in patients with tabes dorsalis. These attacks of intestinal neuralgia in locomotor ataxia are known under the name of abdominal crises, "*crises abdominales.*" Incidentally, I may remark that I do not consider it

[1] Mosse, *Centralbl. f. innere Med.*, 1902, p. 281.

definitely settled that the abdominal crises always occur exclusively in the area of the sensory tracts; I believe that occasionally coördinating or reflex fibers are involved, and that the affection of these tracts may lead to enterospasm; it is impossible to determine in any given case whether this enterospasm is reflex in character or whether it is due to incoördination. A colleague of mine with locomotor ataxia told me, for instance, that he always passed spastic feces, which were thin and faceted, after each attack of abdominal pain, whereas at other times the dejecta were always formed in a perfectly normal manner.

The following description of hyperesthesia of the mesenteric plexus which is purely clinical was given by Romberg and is reproduced here verbatim out of respect to the memory of this old master of neuropathology : "The pain spreads from the umbilicus over the abdomen, appearing in paroxysms with intervals of quiescence and rest between. The pain is tearing, cutting, pressing, most frequently twisting, pinching, and is ushered in and accompanied by a peculiar feeling of soreness. The patient is restless and tries to get relief from his distress by changing his position and by compressing the abdomen; the feet, hands, and cheeks are cool; the expression of the face is tense, the eyebrows puckered, and the lips pressed tightly together; altogether the facies expresses pain. The pulse is small and of high tension. The abdominal walls are distended or retracted, and in any case tense. Vomiting, nausea, strangury, and a constant desire to urinate are frequent, and occasionally there is tenesmus. The patients are usually constipated during the attacks of abdominal crises; less often the bowels are open normally, and in some instances defecation may even be increased in frequency. An attack of this kind may last for a few minutes only or for several hours with intermissions. The attack stops suddenly as though "cut off"; as soon as it is over the patients feel quite well and there is a distinct sensation of euphoria. The course of this affection is periodic, though the attacks do not recur with the same regularity as in other forms of neuralgia."

A critical and unprejudiced observer will notice at once that the clinical picture described does not accurately correspond with the condition now under discussion—viz., that of nervous enteralgia : Romberg's description, given above, is only a classic sketch of an attack of genuine intestinal colic. Such a clinical picture of colic I have described on page 151, and it will be sufficient here to refer to this description.

It is difficult to give a description of pure neuralgia of the mesenteric plexus. It is apparent at once that lead colic cannot be considered to be the basis of this affection, for lead-poisoning involves many different parts of the body and is characterized by a number of anatomic changes and of functional disorders; consequently, the varied and complicated clinical picture of plumbism is very different from the syndrome presented by cases of neuralgia of the mesenteric plexus; in lead-poisoning, moreover, not only the plexus mesentericus, but also the muscular tissue of the intestine and the blood-vessels, are simultaneously involved. It is an equally difficult matter to arrive at a correct inter-

pretation of the complicated clinical pictures described by hysterical and neurasthenic sufferers from this disease. A case of the kind is described on page 153 ; in the same paragraph I have also described a case which I regarded as one of chronic nervous enteralgia.

In tabetic subjects neuralgia of the mesenteric plexus frequently occurs as a violent attack of pain accompanied by constipation and sometimes by diarrhea. In this form of mesenteric neuralgia there may also be a purely local form of neuralgic pain limited to the rectum, and therefore involving chiefly or exclusively the hypogastric plexus (so-called *crises anales*) ; these attacks are characterized by violent tenesmus, occasional paroxysms of furious pain, and a feeling as though a red-hot rod of iron were inserted into the rectum ; occasionally these attacks are complicated by diarrhea.

It is quite impossible to give a description which applies to all the cases of neuralgia of the mesenteric plexus. An essential point in arriving at the diagnosis is to exclude all other possible sources of the syndrome ; it is always necessary, therefore, to examine the patient thoroughly and to rule out any other explanation of the pain, and especially any organic lesion which can possibly account for the syndrome of mesenteric neuralgia. Any careful and conscientious physician will at once admit how difficult it may be in any given case to determine the presence or the absence of anatomic lesions which might possibly be responsible for the attacks of pain—for example, cords and adhesions due to past peritonitis. I go so far as to state that the diagnosis of neuralgia of the mesenteric plexus is never fully justified unless a laparotomy has been performed, and that the diagnosis cannot be made until such an exploratory measure is adopted ; a possible exception to this rule may be made for neuralgia of the mesenteric plexus in patients with a distinct and well-marked neuropathic disposition. [It may be extremely difficult to make a diagnosis between a gastric crisis and genuine intestinal obstruction in a patient who has tabes. Lead colic may be followed by appendicitis ; appendicitis may occur in a patient working in lead, and may prove fatal from perforation (de Havilland Hall).[1] Apert[2] has recently described cases of appendicitis and of lead colic in workers engaged in pearl setting which were extremely puzzling from the point of view of diagnosis. The presence of a marked leukocytosis would be in favor of appendicitis. Clifford Allbutt[3] describes enteralgia as perhaps the most terrible of all the neuralgias, and quotes the case of a patient who, after having had cervicobrachial neuralgia, insomnia, periodic coryza, and dyspnea, developed enteralgia ; great benefit resulted from a long rest and sea-baths. He insists on the importance of distinguishing between enteralgia and the wearing pain at the hepatic flexure of hypochondriacal patients, the latter being relieved by blue pill and senna, while enteralgia is made worse by this treatment. Arsenic is not of so much use in enteralgia as in gastralgia ;

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[1] De Havilland Hall, *Lancet*, 1899, vol. ii., p. 302.
[2] Apert, *Soc. Méd. des Hôp.*, Paris, February 27, 1903.
[3] Clifford Allbutt, *Allbutt's System of Medicine*, vol. iii., p. 479.

belladonna is of some use, but not in severe attacks. Opium, though it relieves enteralgia, must never be given, as the habit is so prone to be acquired.—ED.]

The treatment of colic and of neuralgia mesenterica has previously been discussed.

DISEASES OF THE SECRETORY-VASOMOTOR APPARATUS OF THE INTESTINE.

It is very probable that changes in the blood-contents of the intestinal vessels occur as a result of direct nervous influences, and that the amount of blood present in these vessels is dependent on changes in the nervous system; it is true that it is difficult to prove that this is actually the case in human subjects. Certain morbid conditions, however, almost force us to this conclusion, and we are justified in believing that certain nervous influences affecting the vasomotor nerves of the intestine may in one instance lead to the pouring-out of an abundant amount of fluid, in another of mucus. In all these cases, however, the condition is not one of a pure and uncomplicated secretory neurosis, for in nearly all the instances that have been observed there is also some evidence that the motor and sensory nerve-tracts share in the morbid process; occasionally both motor and sensory tracts are involved at the same time as the secretory fibers.

These clinical conditions have already been dealt with in the section on Nervous Diarrhea, and also on p. 223, where mucous colic is described.

DIVERTICULA OF THE INTESTINE (Diverticula intestini).

DIVERTICULA of the intestine, formed by bulging or protrusion of a portion of the intestinal wall, are usually subdivided into congenital and acquired forms. In the congenital forms the wall of the diverticulum is formed by the whole intestinal wall, and on this account they have also been called " true " diverticula, in contradistinction to " false " diverticula, in which the wall of the diverticulum is formed only by the mucosa and serosa, the former protruding through a space in the muscular coat. Acquired diverticula are, as a rule, constructed in the latter way, so that acquired and false diverticula are usually considered to be identical. This definition, however, is not quite correct, for occasionally the wall of an acquired diverticulum is composed of all the layers of the intestinal walls.

CONGENITAL—SO-CALLED MECKEL'S—DIVERTICULUM.

Among the congenital forms, " Meckel's diverticulum " is the diverticulum $\varkappa\alpha\tau$ $\grave{\epsilon}\xi o\chi\acute{\eta}\nu$. Other forms of congenital diverticula are so exceedingly rare that, from a practical point of view, they are quite unimportant (compare the summary of the literature by Leichtenstern in von Ziemssen's *Handbook* and in Edel).

A genuine, true congenital Meckel's diverticulum is due to the per-

sistence or incomplete obliteration of the omphalomesenteric duct, consequently Meckel's diverticulum is always single and always attached to the small intestine, and especially to the ileum. Generally it arises at a point from one-half to one meter above the ileocecal valve; in exceptional cases it is found only a few (5) centimeters above this valve, and in rare instances higher up in the small intestine than one meter (in one instance it was found in the jejunum and in another case at the junction of the jejunum and ileum). The diverticulum always arises from the convex, free margin of the intestine, opposite the attachment of the mesentery, and leaves the intestine either at a right angle or at an acute angle. It varies in length from 3 to 10 cm.; in extreme cases it may acquire a length of 25 cm.; in other cases again it may merely form a small, wart-like nodular protrusion on the wall of the ileum.

[Incidence of Meckel's diverticulum: It occurs in about 3 per cent. of all the bodies examined, and is much commoner in males. In 298 autopsies Kelynack[1] found 4 cases of Meckel's diverticulum, all in males, and in 337 cases examined at St. George's the editor[2] found 10 examples of Meckel's diverticulum, 9 of them in males. The statement that Meckel's diverticulum is always inserted into the convex border of the ileum is perhaps too dogmatic, for among the editor's 10 cases there were 2 instances in which it arose from the mesenteric attachment and lay between the peritoneal folds of the mesentery. These cases were in a male baby aged six weeks and in a youth sixteen years old, and there was no reason to regard them as acquired diverticula, which are attached along the concavity of the bowel.

In a case recorded by Pollard,[3] a diverticulum 36 inches long came off from the small intestine at a point 24 inches from the pylorus and 63 inches from the cecum; it passed out at the umbilicus and had a complete mesentery. Cases of this kind have been described as bifurcation of the intestine.—ED.]

Occasionally it has a small mesenteriolum.

In the most marked cases of this abnormality the omphalomesenteric duct persists as a tube whose walls possess the same histologic structure as those of the ileum, and with glands of Lieberkühn, and occasionally even Peyer's patches. Sometimes the lumen of Meckel's diverticulum establishes a free communication between the ileum and the external surface of the body, so that the stercoraceous contents of the ileum may escape at the umbilicus. In the great majority of cases the distal end of the tube—that is, the end away from the intestine—becomes obliterated, and subsequently becoming separated from its umbilical attachment, hangs free in the abdominal cavity. In this way a Meckel's diverticulum proper is formed. At its point of origin from the intestine the diverticulum is occasionally as broad as the intestine itself, but, as a rule, it is somewhat narrower. Occasionally it maintains the same width throughout; in the great majority of cases, however, it gradually becomes narrower, so that its free distal end is conical. Occasionally

[1] Kelynack, *Jour. Anat. and Physiol.*, vol. xxvi., p. 554.
[2] Rolleston, *ibid.*, vol. xxvi., p. 93.   [3] Pollard, *Trans. Path. Soc.*, vol. xlviii., p. 47.

the distal end again enlarges, so that a spheric, flask-shaped swelling is formed, due to a protrusion of the internal layer of the wall of the diverticulum through the atrophied muscular coat; this presents the appearance of a herniform protrusion. [Hudson [1] gives a good illustration of a hammer-shaped Meckel's diverticulum.—ED.] This ampullary enlargement of the free end of Meckel's diverticulum occasionally plays an important rôle in the pathogenesis of occlusion of the bowel.

In the great majority of cases, as I have already mentioned, the distal end of the diverticulum is free; occasionally, however, it may remain adherent to the umbilicus by a solid cord; more frequently secondary inflammatory adhesions are formed around the distal end. In cases of this kind the free end may become attached anywhere—most frequently to the mesentery. Cazin found it attached to the mesentery ten times in 23 cases; Treves seven times in 19 cases. It may also become attached to the umbilicus, the small intestine, the cecum, the colon, the inguinal ring, the omentum, the pelvic organs, or any portion of the abdominal parietes.

Meckel's diverticulum is of some importance in the pathology of internal herniform strangulation of the bowels, as has been shown under those headings.

[When Meckel's diverticulum is patent at the umbilicus, the condition is serious and the infant rarely survives. Hubbard [2] has been able to collect 9 cases in which a cure was obtained. Umbilical polypi may be due to remains of the omphalomesenteric duct. For an account of primary umbilical tumors Giannettasio's [3] article should be consulted. Dobson [4] has recently collected 13 cases of intussusception of Meckel's diverticulum, of which 9 proved fatal. Obliteration of Meckel's diverticulum was thought to be the explanation of an adenomyoma attached to the small intestine of a premature infant (Durante). [5]

Grawitz [6] described a congenital diverticulum of the colon, and Treves [7] quotes 5 cases of congenital diverticula of the rectum.—ED.]

ACQUIRED DIVERTICULA OF THE INTESTINE.

Acquired diverticula of the intestine, in contradistinction to the congenital form just described, are rare; as a rule, they are multiple, and are occasionally present in astonishing numbers. Wallmann found nine in the colon of one case. Some authors record much larger numbers, Hansemann even reporting "about 400" in a case. The size of the diverticula varies as much as their number: they may be as large as a millet-seed, a pea, a walnut, a pigeon's egg, a hen's egg, or an apple. Virchow and Edel have recorded the largest ones, some of them possess-

[1] L. Hudson, *Trans. Path. Soc.,* vol. xl., p. 98.
[2] J. C. Hubbard, *Annals of Surgery,* April, 1902, pt. cxii, p. 495.
[3] Giannettasio, *Arch. gén. de Méd.,* 1900, p. 52.
[4] Dobson, *Lancet,* 1903, vol. i., p. 1160.
[5] Durante, *Bull. Soc. Anat.,* Paris, 1901, p. 598.
[6] Grawitz, *Virchow's Archiv,* vol. xlviii., p. 506.
[7] Treves, *Intestinal Obstruction,* 1899, Ed. ii.

ing a volume equal to that of an apple. The form of these divertic-
ula may be hemispheric, cone-shaped, cylindric, or lobulated. They
may be found in any portion of the bowel from the duodenum down-
ward as far as the rectum, and when multiple, may occur in all parts of
the bowel. On an average they are more frequent in the colon than in
the small intestine, but occasionally the reverse holds good. In Hanse-
mann's case, for example, the diverticula were present in the largest
numbers in the jejunum, the upper part of the ileum, and in the sigmoid
flexure, where they were especially numerous. Roth describes a case in
which the diverticula were situated in the duodenum, and in which the
descending portion of this part of the bowel seemed to contain more
diverticula than the other portions. The diverticula were particularly
numerous around the area of the common bile and pancreatic duct.
Diverticula have also been occasionally seen in the vermiform appendix.
[M. H. Fischer [1] has recently described a case and discussed the mode
of formation.—Ed.]

All authors seem to agree more or less as to the exact portion of the
bowel that is occupied by these acquired diverticula, for they all state
that diverticula are nearly always found in the concave surface of the
intestine opposite the attachment of the mesentery. Here they are
usually situated between and covered by the diverging layers of the
mesentery.

Acquired diverticula in the great majority of cases are herniform
protrusions of the mucosa through the muscular coats; at the same time
the serous lining is pushed out by these hernias so as to cover the diver-
ticula, the muscular layer being usually absent. For these reasons
Rokitansky has designated these diverticula as hernias of the mucous
lining. A number of cases, however, are also on record in which all
the layers of the intestinal wall were well preserved and formed the
wall of diverticula.

Of late years special attention has been directed to the interesting
question of the *pathogenesis* of these diverticula. As these lesions are
chiefly of anatomic interest, and have very little clinical significance,
only a few general remarks will be made on this question. In the first
place, attention must be drawn to the fact that a distinction should be
drawn between *pulsion* and *traction diverticula*. This conception, how-
ever, by no means exhausts the mechanical explanations for the genesis
of diverticula.

As diverticula are relatively frequent in the colon, and as normally
small diverticula are found between the tenia of this part of the bowel,
the theory has been advanced that these diverticula were due to the
action of accumulated fecal matter in the small protrusions between the
tenia. This is the probable explanation. In cases of diverticula of
the bowel occurring after partial rupture of the intestinal wall, or after
gall-stone impaction in the intestine, or after stenosis of the bowel, the
same origin must be assumed. Conversely, Birch-Hirschfeld reports a
case in which the apex of the diverticulum was directly continuous with

[1] M. H. Fischer, *Jour. Exper. Med.*, 1901, vol. v., p. 833.

ribbon-shaped and contracted cords of mesentery. In the same way the formation of diverticula by traction has been seen in cases of atrophy of the pancreas and in the formation of cicatricial tissue in the intestine itself. We may assume at least that the traction exerted in these latter cases is the cause of the diverticulation of the bowel-wall.

Special attention has recently been directed to certain relations which seem to exist between diverticula situated on the concave side of the intestine and the entrance of the mesenteric vessels into the intestine. This relation was first pointed out by Klebs, and was subsequently studied more carefully by Hanan and Hansemann, and lastly specially emphasized by Grassberger. These writers all showed that these hernial diverticula consist exclusively of mucous membrane, and contain no muscular tissue; they appear by the side of the veins of the intestine, and penetrate through the sheaths of the vein into the mesentery. At this point a *locus minoris resistentiæ* must be imagined. [Fischer, who adopts this view, gives some useful figures to illustrate the method of formation of these diverticula.—ED.] It has even been shown experimentally that if the intra-intestinal pressure is greatly increased, small diverticula can be artificially produced in these positions, particularly in the intestine of senile persons.

A special kind of diverticula may be formed by the development of neoplasms in the intestinal wall. On page 449 I have reported a case of this kind that I observed myself.

[The diverticula on the descending colon may give rise to pericolitis sinitra, or local peritonitis, which imitates appendicitis, only it is on the left side.

False diverticula or pouches formed of the mucous coat only are not very uncommon in the duodenum; they nearly always occur in connection with the biliary papilla, and have been regarded as developmental in origin and due to weakening of the wall of the bowel at this point owing to the hepatic diverticulum. They may, however, be in reality acquired and be traction diverticula. Keith [1] states that they are present in all women who have evidence in their bodies of having worn tight corsets; the duodenum, which is depressed downward, as in enteroptosis, is held up at one spot by the bile-duct, which, being coated with a layer of tough connective tissue, will not yield; as a result the duodenal wall is pulled out into a pouch. The pouch is usually single, but may be bifid or double; Nattan-Larrier [2] and Rolleston and Fenton [3] have described bifid pouches. Duodenal pouches have also been seen in the position of the opening of Santorini's duct of the pancreas (Rolleston and Fenton).—ED.]

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[1] Keith, *London Hosp. Gaz.*, October, 1902; *Lancet*, 1903, vol. i., p. 640.
[2] Nattan-Larrier, *Bull. Soc. Anat.*, Paris, 1898, p. 4.
[3] Rolleston and Fenton, *Jour. Anat. and Physiol.*, vol. xxxv., p. 10.

ANOMALIES IN THE POSITION AND THE FORM OF THE INTESTINE AND SPLANCHNOPTOSIS.

THE relative position of the different portions of the bowel to one another and to the other abdominal organs is usually regarded as constant and fixed. Our diagnosis is based on this assumption, and the surgeon acts upon it. Generally speaking, this view is correct, but occasionally deviations from the normal occur. Such abnormalities may be extremely important, both in the diagnosis and treatment of intestinal diseases, and therefore call for a detailed description. Many of the abnormalities in the position and the form of the intestine have long been recognized. Esquirol was the first to call special attention to the displacement of the transverse colon, which was subsequently recognized by Fleischmann (1815) and Annesley. Rokitansky and Virchow investigated the displacements of the bowel due to peritoneal adhesions, and made us thoroughly acquainted with these anomalies of form and position. Other anomalies, such as changes in the position of the vermiform appendix, have only recently been studied, particularly during the modern operative epoch, and by physicians since Glénard's publication of his work. The first systematic investigations, however, into this subject from a clinical point of view were embodied in Curschmann's valuable work. He paid special attention to the anomalies of that part of the bowel which is chiefly important from the practical point of view, namely, the colon. A comprehensive study from an embryologic and comparative anatomic standpoint was made by Koch. The following description is largely based on Curschmann's researches :

Anomalies in the position of the intestine may be either acquired or congenital.

An anomalous position of the bowel may be acquired ; thus some portion of the bowel becomes overloaded, and in obedience to the law of gravity, becomes displaced downward. The overloading is almost always due to fecal accumulation. Tumors of the intestine are rarely sufficiently heavy to cause displacement by their weight. In the great majority of cases of displacement of the bowel associated with the presence of a tumor the bowel becomes overweighted by the accumulation of fecal material near the obstruction—in other words, by coprostasis. Portions of the bowel with a long mesentery are naturally most easily displaced. The reason why the small intestine is relatively rarely involved in this process is that the factors which tend to produce displacements, such as tumors and fecal accumulation, are very rare in this part of the bowel. These two factors are most frequently present in the large intestine, especially in the transverse colon and in the sigmoid flexure. Habitual overloading of the transverse colon with fecal material may lead to its displacement downward in the middle ; in this way the well-known M shape is created, with the middle portion of the colon nearly touching the symphysis pubis. Other factors which favor this form of displacement are the clothing, particularly in women, tight

lacing, excessive leanness, relaxed abdominal walls. The portions of the bowel which become displaced are, as a rule, also elongated.

In another form of acquired displacement a loop of intestine becomes fixed in some distant part of the abdomen by local peritonitis; if this occurs, for instance, during a period of meteoristic inflation of the bowel, the most remarkable and bizarre forms of fixation may result; *e. g.*, the sigmoid flexure may become fixed in the region of the cecum. The different possibilities of this form of displacement are, of course, almost innumerable, and can hardly be arranged in a schematic way.

Congenital anomalies in the form, position, and size of the intestine in the great majority of cases involve the large intestine, and are of more importance when occurring there than in the small intestine. Leichtenstern has shown that certain fetal conditions may determine the development of anomalies of the large intestine. In a relatively large number of cases the congenital abnormal position of the large intestine is due to the fact that the cecum, which in the fourth and fifth months of fetal life is still in the right hypochondriac region underneath the liver, occasionally fails to descend completely into the right iliac fossa, and thus remains behind the liver. A second factor which may produce congenital displacement of the colon is incomplete development of the muscular bands of the colon. When this occurs, the colon does not become shortened on its longitudinal axis, and as a result it remains too long. A third factor must be sought in a want of proportion which occasionally exists between the fetal development of the abdominal cavity, on the one hand, and of the longitudinal growth of the colon, on the other. It occasionally happens that the abdominal cavity develops too slowly, while the colon grows too rapidly. Lastly, there is a fourth reason—*i. e.*, the development of abnormally long mesenteries; under these conditions the colon acquires an abnormal degree of mobility, and abnormal kinks are consequently formed. The great majority of anomalies in the position of the intestine must be attributed to these four causes, described by Leichtenstern. In all of them the large intestine, contrary to the well-known diagrammatic representation of the relationship of the two parts of the bowels, does not encircle the small intestine, but forms entirely abnormal and occasionally very remarkable configurations.

The great majority of these different variations have been described in the endless number of publications dealing with occlusion of the bowel, especially axial rotation. Curschmann describes the following variations:

THE CECUM AND THE ASCENDING COLON.

The cecum is in its normal position, but the ascending colon, owing to the excessive development of its mesocolon, forms veritable loops which may lead to volvulus.

Cases in which the position of the cecum is abnormal are more frequent. A number of different displacements and distortions can be imagined, such

as elongation or enlargement of the cecum, this enlargement and elongation being either true or merely apparent (it may be apparent when the first portion of the ascending colon possesses an abnormally long mesocolon, and consequently is abnormally movable together with the cecum). Both forms of elongation and enlargement of the large intestine, the true and the apparent, may produce torsion of the bowel around the longitudinal axis of the cecum with occlusion of the intestinal lumen. Curschmann also describes cases of kinking and bending of the cecum, occurring in such a way that the cecum becomes twisted upward with its fundus directed toward the diaphragm, and in this way covers a corresponding portion of the ascending colon. In cases of this kind the vermiform appendix is also naturally displaced, so that its free end may touch the margin of the liver or even lie underneath it. It need hardly be mentioned that a knowledge of this anomaly is important in diagnosing the position of the appendix in inflammation of this organ.

Another form of postural anomaly is met with when the cecum does not descend during fetal life in the normal manner, but, together with the vermiform appendix, remains below or behind the liver, so that the ascending colon is either very much shortened or practically absent, while the transverse and the descending colon are normally developed. Here, again, remarkable errors in diagnosis in connection with appendicitis may be made (compare also the section on Perityphlitis).

THE TRANSVERSE COLON AND THE TWO FLEXURES.

One or both flexures of the colon may be congenitally absent. If both flexures are absent, the whole colon may be abnormally short. In cases of this kind the ascending colon runs directly from the lower and outer portion of the abdomen upward to the middle of the liver or behind it; then comes a short transverse colon, and finally the bowel dips immediately downward to the left and outward, forming the descending limb of the colon. In other cases in which only one of the flexures is absent the branch of colon which is abnormal in this sense usually rises upward from the iliac fossa and passes transversely across the abdomen to the region of the other flexure.

In cases in which the flexures are absent or shortened, and in which, nevertheless, the length of the colon is increased, the bowel may form a large loop, the two limbs of which lie in close proximity to each other. This loop takes the place of the transverse colon and is usually situated in front of the liver above, covering the whole of its anterior surface. On autopsy a groove is usually found on the anterior surface of the liver, showing that this loop of colon was permanently in this position. The serous lining over this grooved portion of the liver is usually opaque and thickened. Physical examination of a case of this kind *intra vitam* shows that the liver dulness is replaced by a resonant tympanitic note in the corresponding portion of the anterior abdominal wall. The liver dulness, however, is present in the axilla. Such anomalies in the position of the colon may, of course, occasionally lead

to mistakes in diagnosis, such as confusion with subphrenic abscess or with peritonitis ; it is also possible that, owing to this peculiar position of the colon, the diagnosis of pathologic decrease in the size of the liver may be made.

When the transverse colon is congenitally abnormally long, or if it becomes elongated later in life in the manner already described, abnormal loops of colon may readily be formed ; the apex of these loops often reaches far into the pelvis, where it is either freely movable or becomes fixed by peritoneal adhesions.

If the splenic flexure is enlarged and forms abnormal loops, the splenic dulness may be permanently obliterated.

THE DESCENDING COLON AND THE SIGMOID FLEXURE.

These two portions of the large intestine are the most frequent seat of abnormalities. In early childhood the sigmoid flexure is proportionately much larger than in adults. In many persons, however, these abnormal dimensions persist until late in life. Curschmann examined 233 subjects postmortem, and found great enlargement of the sigmoid flexure, leading to the formation of enormous loops, in 15 of the cases. In all these 15 cases he also found that the whole intestine was abnormally long. Occasionally the sigmoid flexure is double.

Curschmann lays special stress on one abnormality, as it is of great importance from a practical point of view, namely, the occurrence of an abnormally large loop between the starting-point of the lower limb of the sigmoid flexure and the beginning of the rectum. This abnormal loop and the cecum—that is, the beginning and the end of the large intestine—may thus be quite close to each other in the right iliac fossa. It can readily be understood that under these conditions serious diagnostic and surgical errors may be committed : when, for instance, an operation is undertaken to make an artificial anus in the right iliac fossa and the lowermost portion of the colon is opened, the opening would be below the occlusion.

Abnormally large loops of the sigmoid are generally found in the center of the abdomen, their longitudinal axes usually running parallel to the linea alba. In cases of this kind the loop may be situated in front of the stomach, the left lobe of the liver, and a portion of the right lobe of the liver, and may project upward under the convexity of the diaphragm. It is particularly interesting to note that the base of this loop and the lower portions of the descending colon are under these circumstances usually covered by convolutions of small intestine. From a diagnostic point of view this is a very important point to keep in mind, because under some pathologic conditions, such as volvulus of the sigmoid flexure, the whole abdomen may be occupied by dilated and meteoristic loops of the sigmoid flexure, whereas the lowest and most external portions of the left lower quadrant of the abdomen give no meteoristic note, since the loops of small intestine are in front of the sigmoid flexure in this situation.

Curschmann denies that the abnormal size of the loops of sigmoid is due to fecal accumulation and the resulting overweighting of this portion of the bowel (a view which is held by many authors), and believes that it is a congenital condition.

The vermiform appendix also offers an important series of congenital abnormalities for our consideration, particularly with regard to its position. (For a description of these anomalies I must refer the reader to the section on Appendicitis and Perityphlitis.)

Many of these anomalies undoubtedly remain unnoticed and produce no symptoms whatever during the life of the subject. It may be considered established that no impediment to the bowel passage is offered in many cases in which the colon is found enormously enlarged and convoluted after death. In other cases again abnormal length of the colon and looping of this portion of the bowel may produce a series of dangerous sequelæ, as axial rotation and knotting of the bowel. In other cases it may lead to serious but unavoidable errors in diagnosis. It might be possible to recognize some of these conditions in healthy subjects, as, for instance, permanent interposition of the colon between the liver and the abdominal wall. In order to do this the subject would have to be repeatedly examined by careful percussion. Abnormalities in the ascending colon might possibly be recognized by inflation of the bowel from the rectum. But how often, let me ask, is it possible to examine perfectly healthy subjects in this way, and even if these abnormalities are detected, to make use of them afterward in diagnosing some disease in the same individual? Practically, therefore, the advantage of a knowledge of these anomalies consists in putting the physician or the surgeon on his guard, the former in arriving at a diagnosis in very unusual and obscure conditions and signs, the latter in performing laparotomies. It is always well to remember that such conditions exist, and that they may often make a diagnosis or an operation complicated and difficult. Further than this we can probably hardly ever go, as the diagnosis can be made only in exceptional circumstances and when all conditions are particularly favorable.

We may include here a condition which has been described for the past fifty years, in isolated cases, chiefly under the name "dilatation (and hypertrophy) of the colon." It has only recently gained general attention and clinical importance.

This condition appears to be exclusively congenital. The clinical manifestations begin in the first days of life. Records of onset later in life are doubtless due to a lack of data concerning the early postnatal days. The new-born child passes no feces. The abdomen is often enormously distended. At times markedly distended coils of intestine are discernible through the abdominal wall. There are dyspeptic symptoms, malnutrition, and colicky pains. Frequently purgatives are effective; at other times they are valueless. But practically always—and this is characteristic—passage of feces and gases, with subsequent reduction of the distended abdomen, will be secured by inserting a

finger, a catheter, or a drainage-tube into the rectum. Roser has said : "The introduced finger reveals a considerable narrowing, but above this a large, sacciform distention." Göppert writes : "The finger passes through a normal rectum to a point 6 or 7 cm. above the anus, somewhat under the promontory (case, child of five weeks) ; on slight flexion the first phalanx of the inserted finger passes into a large hollow space. The finger is extended and withdrawn when a passage of gas and feces occurs, with great ructus, and the abdomen collapses."

Many children in this condition die early, with obstinate constipation and in an extreme condition of emaciation, often with symptoms of intestinal occlusion, which at times are frequently repeated. Others attain greater age, some reaching the third decade ; but evacuations must always be induced artificially, either by purgatives or by the introduction of a sound. Faradization of the abdomen and irrigation of the intestine are useful aids.

Sections have revealed certain points with great uniformity : enormous distention of the colon from the sigmoid at its entrance into the rectum up to the cecum ; thickening of the intestinal wall ; hypertrophy of the muscularis. The mucosa is usually hyperemic, inflamed, and generally studded with ulcerations. It is very remarkable that no stricture is found.

Various views of the pathogenesis of this condition have been advanced. Hirschsprung believes it to be a congenital primary dilatation and hypertrophy of the large intestine. Marfan thinks it is secondary, the primary factor being a congenital anomaly in the form of the sigmoid flexure. Roser recognizes "a valve mechanism. It shows itself unmistakably by the fact that the intestinal contents are disgorged when a tube or half cylinder is introduced, but as soon as the tube is removed the passage ceases." Göppert argues from his case that there is a kinking of the intestine at the point of transition from the sigmoid flexure to the rectum, with secondary dilatation and hypertrophy of the large bowel, the deciding and primary factor being the presence of a valve mechanism in Roser's sense.

In my opinion the anatomic condition of the colonic wall (muscular hypertrophy and catarrhal ulcerative affection of the mucosa) shows all the signs of secondary dilatation, as developed in stenosis of the intestine. We can readily conceive the production of this condition by such a mechanism as is described by Roser and Göppert. Yet this itself forces us to presuppose a congenital anomaly of the sigmoid flexure whose anatomic form and mechanical action must lead to a kinking.

[IDIOPATHIC DILATATION OF THE COLON.

The term "idiopathic" definitely implies that there is no stricture in the bowel or mechanical obstruction to the passage of feces through the colon. The condition may be : (1) Congenital ; (2) develop within a few months of birth ; (3) come on some years after birth ; or (4) be acquired in adult life (C. F. Martin).[1] Most writers are content with

[1] C. F. Martin, *Montreal Med. Jour.*, March, 1897.

two classes—(1) those occurring in very early life and usually in males ; (2) those which develop in later life, usually after fifty and also more often in males.

Besides the theories of **causation** before noted, nervous derangements of the colon, the effects of habitual constipation, the weakening of the bowel by chronic colitis, and other possible factors have been discussed by various authors. Probably the cause is different in individual cases.

Morbid Anatomy.—The colon, and especially its lower part, is greatly distended, but in most instances the rectum is not dilated, the distention gradually ending at the lower end of the sigmoid flexure. The sigmoid flexure is sometimes the only part affected, and the dilatation is always more marked there and spreads upward into the colon. The distention may be enormous ; the sigmoid flexure has been found to have a circumference of two feet. The colon shows hypertrophy of the muscular coats ; in some instances it is expressly stated that both the coats were affected. The mucous membrane may be normal or may be ulcerated ; the latter condition is secondary. The records of the postmortem examination of 18 fatal cases in children do not support the view that the underlying cause is in reality obstruction (Crozer Griffith).[1]

Clinical Features.—The abdomen is greatly distended so that the name " balloon man " (Formad) [2] may be justified. On percussion the abdomen is tympanitic, and from upward displacement, the liver and splenic dulness disappear. The patients are naturally unwieldy and suffer from shortness of breath and occasionally from edema of the legs. There is usually constipation, but the motions are liquid, rather than scybalous or hard. Attacks of vomiting and hiccup may occur.

The prognosis is very bad, as nearly all the cases die. Osler's,[3] Treves',[4] Cheadle's,[5] and Gwynne's [6] cases recovered.

Treatment.—The bowels should be kept open by aperients. Enemata have been much employed, but they may increase the distention ; irrigation of the colon and the passage of a long tube may be employed to empty the distended colon. Massage is not without danger, as if the colon be ulcerated, rupture might occur.

Operative Measures.—Tapping the colon is dangerous, and might easily lead to leakage and so to peritonitis, though Cheadle's case recovered after the transverse colon was punctured. Osler's and Gwynne's cases recovered after an artificial anus had been made. Parts of the colon have been excised ; thus Treves excised the descending colon, sigmoid flexure, and rectum, and sutured the transverse colon to the anus, with recovery. M. H. Richardson [7] excised the sigmoid flex-

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[1] J. P. Crozer Griffith, *Amer. Jour. Med. Sci.*, vol. cxviii., p. 283.
[2] Formad, *University Med. Mag.*, June, 1892.
[3] Osler, *Arch. Pediatrics*, 1893, vol. x., p. 113.
[4] Treves, *Lancet*, 1898, vol. i. ; *Intestinal Obstruction*, ed. 1899.
[5] Cheadle, *Lancet*, 1898, vol. i., p. 399.
[6] Gwynne, *Reports of the Study of Disease in Children*, vol. ii. ; *Quarterly Med. Jour.*, 1902.
[7] M. H. Richardson, *Boston Med. and Surg. Jour.*, February 14, 1901.

ure, but in fifteen months' time a new sigmoid flexure was formed which filled the lower part of the abdomen.—ED.]

Finally, we may best discuss here a condition that, since Glénard's first contributions on *enteroptosis* (1885), has caused an ever-increasing volume of work on the subject. Among numerous other investigators we need mention only Ewald, Meinert, Weisker, Kelling, Meltzing, Tuffier, Schwerdt, Stiller, Kraus, and Einhorn. The affection involves various organs besides the intestines, and, therefore, the term splanchnoptosis would be more fitting than enteroptosis, yet the latter—the original name—obliges us to give the condition some space in a work on intestinal diseases.

To Glénard belongs the credit for having collected under a general head a series of functional disturbances that had up to that time been considered individually. These were gastric and intestinal symptoms and nervous manifestations on the one hand, and, on the other, malpositions, especially dropping of various abdominal organs, such as the kidneys, stomach, and colon. It is impossible here to retrace step by step the course of discoveries in this field during the past eighteen years, and we must confine ourselves to a few remarks sketching briefly our present knowledge.

First, we shall draw a brief clinical picture of the disease, which may be divided essentially into functional disturbances and anatomic abnormalities.

The functional symptoms concern principally the stomach and intestine. In the stomach they appear as nervous dyspepsia, with all its abnormal subjective sensations—belching and vomiting, with hyperchlorhydria or subacidity, or normal secretion of gastric juices. In the intestine there are distention, wide-spread unpleasant sensations in the abdomen, colic, and constipation, sometimes of spastic, at others of atonic, form, while occasionally there is diarrhea. The patients frequently present numerous subjective symptoms, such as those of the neurasthenic—headache, vertigo, and depression; or the reverse ones—ready excitability, adynamia, paresthesia, pains in various parts of the body, etc.

The anatomic changes, which are in part the consequences of the functional changes, manifest themselves chiefly as displacement and dropping of the abdominal viscera. The stomach becomes lower and more nearly vertical, its musculature extensible, in consequence of which a certain amount of dilatation exists, although the latter is not comparable to that in pyloric stenosis. Besides gastroptosis there is coloptosis. The transverse colon may be sunk low down, but all parts of the large bowel are involved. Ptosis of the small bowel is not demonstrable, yet it is quite possible.

[Prolapse of the mesentery and small intestines was investigated by C. B. Lockwood,[1] independently of Glénard's work; he showed that it was antecedent to and necessary for the production of hernia.

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[1] C. B. Lockwood, *Hunterian Lectures on the Morbid Anatomy, Pathology, and Treatment of Hernia*, 1889, Lewis.

By means of the *x*-rays the existence and position of abnormally situated portions of the intestine can be determined when capsules containing bismuth or when foods mixed with bismuth have been given. This method was suggested by Boas[1] and was employed by Cannon in his investigations on Antiperistalsis (*vide* p. 78).—ED.]

Palpation and percussion and the form of the feces show sometimes atonic dilatation, and sometimes spastic contraction of individual parts of the large intestine. At other times conditions may be normal.

Glénard's view concerning the transverse colon has elicited the sharpest criticism. He supposes the transverse colon to be fastened to the pyloric end of the stomach by a band which he terms the "pylorocolic ligament," while the parts of the colon to the front and the back of the point of attachment (the shorter to the right, the longer to the left) remain free. It is this attachment and the consequent sharp flexion that constitute the chief hindrances to the progress of the intestinal contents and causes the constipation. He further claims that from this point toward the stomach the ascending and transverse colons are filled with fecal matter and gases, and that toward the anus the transverse colon forms a rather hard, diagonal convolution (*corde colique transverse*), as do also the descending colon and sigmoid flexure (*corde colique gauche*).

[This, however, Ewald and other observers regard as the pancreas, which it was, indeed, shown to be by laparotomy in a case of W. F. Hamilton's.[2]—ED.]

A particularly frequent and well-marked appearance is nephroptosis, involving mainly the right kidney, sometimes both. Floating kidney may occasion a secondary series of disturbances. Dropping of the spleen and liver is rarely noted.

A third series of symptoms in splanchnoptosis is also of anatomic nature and concerns the general physical development. Affected subjects generally have a soft skeleton, scant, lax musculature, particularly of the abdomen, sparse cushions of fat, and are often anemic. Stiller has added another characteristic symptom : that the tenth rib has no cartilaginous attachment with the sternum (in very severe cases this is true also of the ninth), but is freely movable (*costa decima fluctuans*). In his experience this has so frequently formed a feature of the symptom-complex that he considers it a veritable *stigma enteroptoticum*.

We cannot here enter upon a lengthy discussion of the foregoing elementary clinical sketch. Nor can we here undertake any further discussion of the nature and pathogenesis of the disease. Be it briefly stated then that, judging from the present state of knowledge of the condition, and from my own experience, I coincide with the opinion expressed by other authors, such as Schwerdt, Tuffier, and Stiller. The last-named authority expresses his opinion exceptionally well in saying that the condition is to be regarded as a congenital one *sui generis*, and suggests the name *asthenia universalis congenita*. The characteristic

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[1] Boas and Levy-Dorn, *Deutsch. med. Wochenschr.*, 1898, No. 2.
[2] W. F. Hamilton, *Montreal Med. Jour.*, September, 1889, p. 698.

of the condition is that congenital anomalies causing fixation of the abdominal viscera exist, and that there are subsequent abnormalities developed in the organism affecting particularly the musculature (smooth and striated alike) and the central nervous system.

From the mass of material, which unfortunately cannot be exhaustively analyzed here, we shall consider briefly one more point of special interest in enteroptosis, not only in the form covered by Glénard's clinical picture, but other forms as well. What factors cause the dropping of the various organs of the abdominal cavity? A number of authors have of recent years been engaged upon this question. Wolkow and Delitzin, in an admirable monograph, have contributed a most thorough report, based on nephroptosis, and have given also a comprehensive review of the literature on the subject. Kraus's recent work is rather a clinical study.

It was formerly believed that bands and folds of the peritoneum (hepatic suspensory ligaments, mesentery, etc.) were the exclusive or chief supports of the abdominal viscera. This view proved untenable, and the belief that the abdominal pressure was of importance in this particular became prominent. Since the time of Braune and Schatz numerous studies of the so-called intra-abdominal pressure have been made (Weisker, Schwerdt, Repreff, Kelling, Meltzing, Wolkow and Delitzin, etc.). It is a very complex phenomenon, numerous factors being involved in its origin and in its variations: Atmospheric and hydrostatic pressure; varying pressure through voluntary or reflex contraction of the abdominal walls; direct compression of the abdominal viscera by certain postures of the body or external pressure; the pressure in stomach, intestines, and blood-vessels; pathologically increased intra-abdominal pressure from a collection of exudates. Wolkow and Delitzin have made a particularly exhaustive study of the equilibrium of the contents of the abdominal cavity and the significance of the chemistry of the latter.

The abdominal viscera are fixed in their normal positions by anatomic and physiologic means when abnormalities of the anatomic structure or changes in the physiologic processes occur, and especially when both occur together splanchnoptosis may result. The anatomic anomalies are mainly congenital. Occasionally they may cause ptosis unaided, but general functional factors act with them. The latter may be the sole cause of dislocation. From this we may see that ptosis of any individual abdominal organ may occur alone.

One of the most important factors for securing static equilibrium in the abdominal cavity is the musculature of the abdomen—"the outer muscle-balloon bounding the general abdominal cavity" (Wolkow and Delitzin). To this belong not only the muscles of the anterior wall, but also the long dorsal and lumbar muscles, the diaphragm, and the muscles of the pelvic floor. Anything that reduces the tone of the outer abdominal musculature favors splanchnoptosis. Thus we can understand its occurrence when general (nutritional disturbances from any cause) or local weakening factors have reduced the strength of the

abdominal muscles. The most frequent local weakening cause is over-distention—and this is the reason for the relative frequency of splanch-noptosis in women who have borne children (in pendulous abdomen, prolapse of the rectum, etc.). Another factor, acting from without, that frequently causes descent of the viscera is the pressure of the bodice and other kinds of tight lacing and belting. A number of authors emphasize this fact.

[Keith,[1] who has recently investigated the anatomy of Glénard's disease, regards it as a result of a vitiated method of respiration, and lays stress on the importance of tight lacing as a causal factor.—Ed.]

Of the remaining factors, we shall consider only one, which, as already stated, is specially noted by Kraus. There are some individuals who, despite a well-developed, strong abdominal wall, have more or less general splanchnoptosis. In such cases the most important physiologic factor in the pathogenesis is lacking, there is no insufficiency of the abdominal musculature, and the general requirements for intra-abdominal balance are not changed. In them Kraus believes, and we can only agree with him, that the pathogenesis is to be sought in congenital constitutional defects, particularly in an abnormal shape of the thorax. This causes narrowness of the lower aperture of the breast, and, of course, also a narrowing of the upper part of the abdominal cavity.

The possibilities for the treatment of splanchnoptosis due to congenital defect are, of course, negative. But the functional factors which first develop into a pathologic condition, the anatomic changes, are to a certain extent susceptible to treatment. This is particularly so in insufficiency of the abdominal musculature, which, when once well developed, can best be compensated for by abdominal binders. The form of binder must, of course, be adapted accurately to the requirements of each case. For the rest we must try everything that will tend to strengthen the muscles in general, and those of the abdomen in particular. Therefore, we prescribe (of course, with due regard to the idiosyncrasies of each patient) exercise in the open air, passive and active muscle exercises (of such nature as not to increase the ptosis), local faradization, massage, and suitable hydropathic procedures. We must also advise good and plentiful nourishment. In doing this we must be careful that the food does not increase distention and descent of the intestines by overproduction of feces and of fermentation.

There are no other important guides for the treatment of the clinical picture as given by Glénard. Treatment must be symptomatic and directed against the underlying cause, as well as against any further development of the causal factors. The disturbances of gastric digestion, the spastic or atonic constipation, the general neurotic state, and the anemia are all treated in accordance with established rules. As a general thing the treatment of this condition is unsatisfactory. Complete cure is impossible : only more or less improvement is attainable, and even this is sooner or later nullified by a recurrence.

[1] Keith, *Lancet*, 1903, vol. i. ; *London Hosp. Gaz.*, October, 1902.

STENOSIS AND OCCLUSION OF THE INTESTINE IN GENERAL (Stenosis et Occlusio Intestini).

I have not altered from the first edition the sequence and grouping of the material in the succeeding pages, as I could not get it into more convenient form. I have endeavored to incorporate with the matter of the first edition all that the copious literature and my own experience have brought to light on this exceedingly important subject.

First, a few words concerning the nomenclature, which here again shows a regretable lack of uniformity. In conjunction with most authors, I mean by—

Stenosis, any narrowing of the lumen of the intestine, regardless of etiology and anatomy.

Occlusion, any complete closure of the intestinal lumen.

Stricture, narrowing of the lumen from diseases that involve circular areas of the intestinal wall.

Constriction, narrowing of the lumen by constriction from without.

Obturation, a stoppage from obstructions in the lumen of the intestine.

Compression and *incarceration* are self-explanatory.

Strangulation is that form of closure in which a strong excitation of the nerves in the intestinal wall occurs, and at the same time the circulation in the intestinal wall and the mesentery is so affected that severe venous hyperemia ensues.

By stenosis of the intestinal lumen it is implied that the occlusion of the bowel is incomplete, whereas occlusion signifies that the lumen is completely obliterated. In stenosis the onward passage of the intestinal contents is rendered very difficult; in occlusion the bowel contents are arrested above the lesion and their onward passage is consequently entirely prevented. These morbid conditions may either have an insidious onset or may appear suddenly; they may attack the patient gradually, or they may appear suddenly and endanger his life in a very short time. I may say that the conditions created by occlusion or stenosis of the intestine must always be considered exceedingly dangerous, and that a patient suffering from stenosis or occlusion of the bowel is always in an exceedingly precarious position. Medical men of all generations have paid particular attention to this condition chiefly because the life of the sufferer is frequently in immediate danger, and because the group of symptoms presented is exceedingly distressing both to the patient and to those who are forced to witness his suffering.

It is very difficult to describe this subject in a clear, succinct, and comprehensive manner. The study of the subject and its description can be approached from various points of view. I consider it my primary object in this work to discuss diseases from the clinical point of view, and to proceed in my descriptions according to the rules that the medical man would observe in dealing with a given case. Special prominence will, therefore, be given to those points of view which

are essentially clinical, and which are of paramount importance to the medical practitioner in actual bedside work. The most important information, therefore, to be imparted in regard to stenosis and occlusion of the intestine is the following : In the first place, it must be shown how occlusion and stenosis of the intestine are to be diagnosed ; this presupposes an exact and exhaustive knowledge of the symptomatology of these diseases ; in the second place, how to determine the exact situation of the lesion or lesions in the intestine ; in the third place, to elucidate and explain the anatomic character of the lesions and the causes which lead to their production ; and, lastly, to find the chief therapeutic indications, and to describe the means and measures at our disposal to meet these indications in the treatment of occlusion and stenosis of the intestine.

Acting on these lines, I will first describe the general anatomy of occlusion and stenosis of the intestine, and then describe those features of the disease that command particular clinical interest and are clinically related to one another, namely, the symptom-complex presented by this condition and the pathology of occlusion and narrowing of the intestinal lumen. An attempt will be made to give all this information in a connected and comprehensive form. My reason for dealing with the symptom-complex and the pathology of occlusion and stenosis together is that these conditions, whatever the underlying causes may be, may produce the same consequences and lead to the same sequelæ ; in other words, the anatomic, physiologic, and the clinical features of any form of stenosis or occlusion of the intestine are more or less alike, or at least resemble each other in many of the essential points. The description of the causes that produce certain special forms of occlusion and stenosis of the bowel, and of the anatomy of these special forms, such as carcinoma, stricture, invagination, obstruction, incarceration, axial rotation, etc., of the bowel, will also be discussed separately. After this a special section will deal with paralysis of the bowels, a condition which is anatomically entirely different from the other stenotic lesions enumerated, but which functionally and clinically produces results like those produced by organic occlusion and narrowing of the bowel lumen. Finally, the general diagnostic points of view and the general principles governing the treatment of all these primary conditions and of all the consequences of stenosis and occlusion of the bowel will be considered in a special section.

A review of the literature of intestinal occlusion and stenosis shows an almost unlimited amount of material. The literature is full of the reports of cases illustrating this condition. It is hardly necessary to lay stress on the fact that I have utilized all the most important and essential features of these numerous descriptions in the following account. I do not consider, however, that a complete review of all this material, or even a review of the majority of the case-records furnished by so many authors properly comes within the scope of a hand-book such as this one. My chief aim in writing this work has been to give as clear and as comprehensive a description of disease as possible. The

book is intended, in the first place, to give the medical practitioner a clear conception of the nature and the peculiar features of this disease and a general outline of the treatment. The information imparted has been largely gleaned from my own experience, but also in great part from the experience of many other authors and investigators.

ANATOMY.

Whenever the intestine becomes narrowed or occluded, the stenosis or occlusion *per se* produces certain anatomic changes in the intestine. The cause of the stenosis or the occlusion and the nature of the primary processes that lead to the development of this narrowing of the intestinal lumen may vary greatly, but whatever the origin of the stenosis or occlusion, the structural changes produced are so uniform that they can be included in a common description. The anatomic condition of the portion of the bowel that is primarily diseased varies, of course, greatly according to the nature of the primary disease. These differences will be described in the account of the various forms of occlusion and stenosis of the intestine—*i. e.*, under the heading of Carcinoma, Intussusception, Volvulus, etc., of the Bowel. All these primary conditions, however, have this factor in common, that they cause narrowing of the intestinal lumen, and this narrowing or occlusion of the intestine is followed by the same structural consequences in all cases. Qualitatively, then, the results of occlusion and stenosis of the intestine are the same. Certain differences become apparent, however, according to the length of time during which the stenosis is in process of development ; in other words, the morbid appearances produced by stenosis or occlusion of the bowel will be different if the narrowing of the lumen occurs gradually and occupies a long time ; or if it develops suddenly, produces acute occlusion of the bowel, and rapidly leads to death of the patient. In describing the anatomic features of this disease, therefore, a distinction must be made between acute and chronic narrowing of the intestine and occlusion. The same distinction, as will appear later, must be made in the clinical account of stenosis and occlusion of the bowel.

Chronic Stenosis of the Intestine.—In any form of chronic narrowing of the intestine, whatever the anatomic process that produced the morbid condition, the segment of the intestine that is situated below the stenotic area is always found empty and contracted ; in other words, it presents the picture seen during inanition. It may be fitly called " hunger intestine " (Hungerdarm). In all other respects the piece of intestine immediately below the stenosis is normal. The appearance of the bowel immediately above the stenosis is entirely different, for the intestine is dilated, occasionally enormously, so that extensive areas of the intestine are distended and may even form a sac-like pouch above the stenosis. In the majority of cases, however, the distention and the sacculation of the intestine are not very great and involve only a short piece of the intestine above the stenosis. In acute occlusion of the in-

testine enormous degrees of tympanitic dilatation are frequently seen; in chronic forms of enterostenosis, however, such a condition is hardly ever seen. It may, however, occur exceptionally, chiefly in cases in which complete occlusion of the intestine occurred immediately before the fatal issue. This acute occlusion following a chronic process of course prevents the passage even of the gases that form in the intestine, so that in this way severe degrees of intestinal distention may develop.

Examination of the wall of the intestine above the location of the chronic stenosis shows a series of changes, some of them of the most severe character. These structural changes of the intestinal wall are produced by the stagnation of intestinal contents, which naturally accumulate in this portion of the bowel because their passage through the stenotic area is so difficult. The stagnating bowel-contents may exercise a deleterious effect on the structures of the intestinal wall above the area of stenosis in two ways: in the first place, they may cause mechanical distention of this portion of the intestine, and consequently stimulate peristaltic actions of the muscular coat of the intestine throughout the whole area of stasis; in the second place, the bowel-contents may act as a direct mechanical and chemic irritant for the mucous lining of this portion of the intestine.

As soon as the bowel-contents stagnate in the portion of the intestine situated immediately above the stenosis, this part of the bowel becomes slightly distended, and as a result the musculature of the intestinal wall is stimulated to increased activity. The direct result of this increased peristaltic action of the musculature in this area is hypertrophy of the muscularis above the stenosis (I refrain from entering into a detailed description of the physiologic factors that are operative in this process). Hypertrophy of the musculature thus produced can be clearly recognized even by macroscopic inspection of the intestine. If the disease is of protracted duration, the muscular hypertrophy may reach very considerable degrees; it may involve short or long portions of the bowel; in stenosis of the hepatic flexure of the colon, for instance, it may involve all the large intestine as far as the ileocecal valve and portions of the ileum; in stenosis of the sigmoid flexure it may involve the whole of the large intestine as far as the ileocecal valve. It will be found, however, that the hypertrophy of the muscular coat of the intestine is always most pronounced in the immediate vicinity of the obstruction. The histologic features of this muscular hypertrophy are the following: The muscle-fibers are broader than normal, but are not increased in number; in other words, there is no hyperplasia of muscle-fibers, but a true muscular hypertrophy, in the sense of Virchow. I have been able to determine the character of the change of the muscular coat by careful histologic examination of the human intestine taken from cases of stenosis or occlusion; and one of my pupils, Herczel, has performed a number of experiments in animals and has carefully investigated the condition of the muscular coat of the intestine in experimental occlusion of the bowel. He found that the first signs of this peculiar hypertrophy of the muscular coat became

manifest histologically as early as the fourth or fifth day after the stenosis of the bowel is brought about, and that on the ninth day after the experimental stenosis the hypertrophy of the muscular coat is distinctly pronounced and easily recognizable. He never succeeded in demonstrating an increase or proliferation of the connective-tissue structures of the bowel-wall. He found, however, that the smaller blood-vessels and the capillaries running through the muscular tissues of the intestinal wall situated above the stenosis underwent considerable dilatation. It seems clear, therefore, that in these instances we are dealing with a genuine functional hypertrophy caused directly by the increased work which is thrown upon the muscle-fibers of the intestinal wall immediately above the stenotic area. This hypertrophy of the muscular coat enables the intestine to overcome the obstacle presented by the stenosis to the onward passage of the intestinal contents. The increased muscular exertion followed by hypertrophy of the muscular coat, and consequently greater power of the musculature in the intestinal wall immediately above the stenosis, may aid in propelling the intestinal contents through the stenotic area for some time, and in this way a compensatory activity is inaugurated that neutralizes, for a time, at least, the effect of the stenosis.

[Patel,[1] in a discussion on the factors which determine whether or not hypertrophy of the muscular coat occurs above an intestinal stricture, points out that in many cases in which the lumen of the intestine is narrowed—*e. g.*, by pressure from without or by innocent tumors, such as multiple polypi—the bowel above the point of stricture is dilated, but not hypertrophied unless there is ulceration. He considers that ulceration and not obstruction is the essential factor determining hypertrophy. Analogy with the heart would suggest that obstruction is the essential factor, but the heart is continually contracting, while the intestine does so only when stimulated. When ulceration is present, the muscular fibers of the intestine are continually being stimulated to contract. This theory would explain hypertrophy in cases of intestinal ulceration without any stenosis—for example, in congenital dilatation of the colon.—Ed.]

In addition to hypertrophy of the muscularis of the intestine, changes in the mucosa and the submucosa are very frequently met with. These lesions are more pronounced in the large intestine than in the small intestine, and consist in inflammation and ulceration of the mucosa and submucosa. As these lesions are never present below the stenotic area, but are usually most marked immediately above the stenotic area, the intestine presents a very peculiar and quite characteristic appearance, the mucous membrane above the stenosis forming a marked contrast to the mucous membrane below the stenosis. These changes in the mucosa and submucosa are brought about, in the first place, by the mechanical pressure exerted by the stagnating intestinal contents, feces as well as gases. They are due either to chemic or bacterial irritants present in the bowel-contents. As a result of all these factors an intense catarrhal

[1] Patel, *Lyon médical*, vol. xciii., p. 73.

affection of the mucosa is developed ; later most pronounced ulcerative destruction of the mucosa and the submucosa supervenes, leading to the form of ulceration of the intestine that is described under the name of stercoral or decubital ulcers of the bowel (see p. 255). Kocher has suggested for them the term "distention ulcers" (Dehnungs-geschwüre). He remarks that the stagnating masses are by no means always hard and fixed above the stenosis, and therefore we cannot speak of decubital or stercoral ulcers. He claims that the only constant factor accompanying this ulceration is overdistention of the intestine, which considerably affects the circulation in the walls and disturbs their nutrition. This disturbance of circulation alone is sufficient to cause circumscribed necrosis, whose development into ulcers is further favored by the chemic and bacterial irritants. When this condition of ulceration develops, the appearance is very striking. The intestine is usually dilated and sacculated in certain areas, so that it is shaped like a flask. The mucous membrane in the dilated portion is seen to be covered with a surprising number of decubital ulcers. If the stenosis or the stricture of the intestine is the result of some carcinomatous growth or follows cicatricial contraction of tuberculous or dysenteric ulceration of the bowel, great care is required in interpreting the anatomic findings, for it is very easy to confuse the ulcers due to the primary disease— *i. e.*, the carcinoma, the tuberculous or dysenteric process—with the stercoral or decubital ulcers that develop as secondary results of the stenosis produced by these primary processes. Decubital or stercoral ulcers above a stenosis of the intestine in their turn may lead to further complications ; the most notable of these are perforation of the bowel and secondary adhesive peritonitis. Perforation may either occur into the general abdominal cavity, or, if a sufficient number of peritoneal adhesions form before perforation occurs, may lead to the development of a circumscribed fecal abscess which is separated from the general abdominal cavity by adhesions. Occasionally perforation of the bowel under these conditions may lead to the development of a gangrenous phlegmon.

Another very interesting and important sequel of stenosis of the bowel, which is specially seen in the chronic form of enterostenosis, is an elongation of the intestine above the stenosis. The elongation is particularly noticeable in the colon. It may become so elongated that it follows a serpentine, winding course across the abdomen. Herczel was able to produce elongation of the colon in some of his experimental investigations. In a few of the animals that he operated on the elongation of the bowel occurred within a very short time. In addition to these secondary changes Sklodowski points out that cicatricial striations are occasionally observed in the mesentery, and are caused by mechanical stretching of the mesentery.

Acute Occlusion of the Bowel.—We have seen that chronic stenosis of the bowel, whatever its primary cause, almost without exception leads to the development of the same pathologic changes. In the preceding paragraphs an account has been given of the structural changes met

with in chronic stenosis and of the secondary changes in the portion of the bowel above the stricture. In acute occlusion of the bowel the changes are entirely different. The immediate consequences of acute occlusion of the intestine may vary greatly ; they are certainly different from the immediate sequelæ of chronic occlusion. Acute occlusion of the bowel may be due to such a variety of etiologic factors that the secondary symptoms and the secondary anatomic lesions produced by this condition assume a great variety of forms.

The thickening of the intestinal wall seen in chronic occlusion of the bowel is completely absent in acute occlusion. It has been seen in the preceding paragraphs that this thickening of the intestinal wall is due to a considerable hypertrophy of the muscular coat of the intestine. The catarrhal and ulcerative changes of the mucous lining above the obstacle in the chronic form are completely absent in the acute occlusion. This is chiefly due to the fact that acute occlusion develops so rapidly that no time is given for the production of the changes in the muscular, mucous, and submucous coats that supervene in the chronic form of stenosis.

In describing the pathologic changes presented by the intestine in the chronic form of stenosis attention was directed to the marked contrast that exists between the state of the mucous membrane above and below the stenosis. In acute occlusion of the bowels there is also a great difference in the appearance of these two portions of the mucosa ; in fact, in the acute form the contrast may be still greater, so that the mucous membrane covering the loops above and below the stenotic area may vary still more in appearance than in the chronic form. The whole intestine below the stenosis is empty and contracted, whereas the loops of intestine above the area of acute occlusion are frequently dilated and distended to a much greater degree even than in the chronic form. The walls of these distended loops of intestine may either be very much thinner than normal or very much thicker. The condition of the wall will depend on the nature of the primary organic process producing the stenosis. If the distention is great and sudden, the walls of the intestine will be pale and thin ; in other instances the wall may, as has been said, be thick (but not hypertrophied) and at the same time of a dark, blood-red color.

Generally speaking, the most characteristic feature of acute occlusion of the intestine is the fact that the bowel becomes distended and dilated immediately above the occluded spot. In individual cases, however, certain peculiar conditions may be observed on close study in regard to the anatomic arrangement of this disordered area and in regard to the method in which the dilatation and distention of the bowel immediately above the stenotic area occur. Until recently the great majority of authors were inclined to the view that the dilatation of the intestine in this portion was, in general, due to meteorism ; this applied both to the dilatation seen during life and observed after death. It was believed that this flatulent distention was the direct result of stasis. Another explanation that has been advanced, and that applies to certain cases, is

that the dilatation and distention of the intestine are due to peritonitic processes that complicate acute occlusion of the bowels and appear in the course of the disease ; in other words, the opinion was generally prevalent that this form of meteorism was *diffuse,* in the sense, namely, that it was due to the stasis of bowel-contents above the area of obstruction in the intestine. Quite recently a new theory has been advanced, and modern authors speak of a *local* form of meteorism. Some time ago a number of different investigators described this form of meteorism quite clearly. Among them, Förster may be mentioned. Until quite recently, however, medical men and investigators in general paid little attention to these older descriptions and did not accept the views advanced by Förster and others with much favor. It remained for von Wahl to recognize the importance of local meteorism, and he deserves credit for his careful studies of this condition. He was the first to recognize the fundamental importance of this affection, and to observe that it constitutes one of the most frequent symptoms in certain forms of acute occlusion of the bowel. He also found that it occurs almost constantly in this condition, and that it is of the greatest importance in the pathology and the diagnosis of acute occlusion of the intestine. After von Wahl, Zoege von Manteuffel advanced some views and advocated a theory in regard to the origin and the significance of this local meteorism that was identical with the views advanced by von Wahl. Quite recently Kader has investigated another important question that is related to local meteorism, namely, the regularity and periodicity of its appearance and its mode of origin. He has published an excellent monograph on this subject and has described a number of experiments on animals. I am personally convinced that this investigation of Kader's into the mode of appearance and the origin of local meteorism solves the interesting problem in a most satisfactory and a very conclusive manner.

In one series of cases the dilatation of the intestine is actually the result of the stasis of gaseous and solid, or possibly liquid, bowel contents above the obstruction. This is, as we have said, the generally accepted view and explanation of this phenomenon, and, as in the preceding paragraph, we will designate this condition as meteorism from stasis. This form of meteorism, followed by dilatation and distention of the intestine, is seen in all those cases of occlusion of the bowel in which there is simply occlusion of the intestinal lumen without at the same time any interference with the circulation in the walls of the intestine or in its mesentery. The distention of the bowel is, of course, most pronounced in the immediate vicinity of the obstacle that causes the occlusion of the intestine, and is most intense in this area in the early stages of the disease. This has been demonstrated very conclusively by experimental occlusion of the intestine in animals ; similar conditions have been observed in a number of cases in human subjects in which the patients rapidly succumbed to the disease and in which an autopsy was made soon after death. Under the latter conditions—that is, in acute cases that die rapidly and are examined very soon after

death—the distention of the bowel may be limited, for instance, to the colon in occlusion of the large intestine—that is, in this case the bowel may be distended from the obstruction upward and backward as far as the ileocecal valve or beyond it. When this occurs, the ileum is not distended and performs its functions, particularly the propulsion of the intestinal contents, in a perfectly normal manner. In this way it becomes possible for the contents of the ileum to enter the colon through the ileocecal valve, while at the same time the contents of the ileum, after having once passed through the ileocecal valve into the colon, cannot regurgitate into the ileum. When the occlusion of the colon is complete or nearly complete, pressure in those portions of the colon above the obstruction which have become distended and filled with the contents of the ileum becomes very high, and there may be regurgitation of this material backward through the ileocecal valve into the ileum under certain conditions. If this occurs, the distention of the bowel extends to the small intestine, and occasionally the whole small intestine, sometimes even the duodenum and the stomach itself, are seen to be greatly distended and dilated. If the obstacle that causes occlusion of the intestinal lumen is situated high up in the bowel, distention of the duodenum and the stomach will, of course, occur more readily than if the occlusion is situated in some portion of the bowel far removed from the duodenum or the stomach. Stress need hardly be laid on the fact that the distention of the bowels can occur only within certain limits. In the first place, the tension of the abdominal wall offers a certain resistance to the distention of the intestine, and all the other tissues and organs which form the boundaries of the abdominal cavity act in the same way, so that as soon as the intra-abdominal pressure and tension become too great, these opposing forces come into play and prevent excessive distention of the bowel. The loops of intestine themselves exercise a similar effect, and there is no doubt that by pressing upon one another when distended, they prevent extreme degrees of dilatation and distention of the intestine.

This mode of dilatation and distention of the intestine is observed particularly in obstruction of the intestine by foreign bodies, gall-stones, and masses of hardened fecal matter. It is also occasionally seen in certain cases of invagination of the bowel ; further, in those cases of chronic stenosis of the intestine in which the nature of the stenosis is such that the lumen of the intestine gradually becomes smaller and smaller and is ultimately completely occluded. The nature of the chronic process that produces this progressive stenosis does not affect the result. In general terms it may be stated that this variety of meteorism from stasis is liable to occur in all cases of occlusion of the intestine in which the lumen of the bowel is simply occluded, but in which, as has already been mentioned, there is no interference with the normal circulation of the blood and lymph in the wall of the intestine and in the mesentery. Another variety of occlusion in which this form of meteorism may be observed is the " bileptic " form of occlusion. This is seen in certain cases of volvulus, kinking, and internal strangulation of the intestine.

In such cases meteorism, of course, appears only in certain loops of intestine, namely, not in those that are situated above any one of the several occlusions, but only in those above the highest occlusion, for here only can an accumulation of fecal matter coming from above occur; in other words, this meteorism from stasis in bileptic occlusion is chiefly marked in that loop of intestine which is situated nearest to the stomach and immediately above one of the occluded spots.

The contents of loops of intestine that are distended and dilated by meteorism from stasis consist of liquid matter and pultaceous masses of fecal material and of gas. Talma has investigated the constitution of the contents of the dilated loops of intestine in this condition in animals, and on the basis of his experiments on animals comes to the conclusion that the contents of the loops have approximately the following composition: The amount of gas present is comparatively small, and, as a matter of fact, the meteoristic distention of the loops of intestine is only to a slight degree due to an accumulation of gas and the pressure thus exerted. The greater portion of the bowel contents in these loops consists of liquid material that in reality represent an accumulation of the secretions that are poured into the bowel by the glands of the stomach and the intestinal wall. Talma also found that the amount of liquid present in the intestinal contents within these loops varies greatly according to the exact situation of the occlusion. He found that there is very much more fluid in the intestinal contents above the occluded area when the lesion is present in the small intestine than when it is present in the large intestine, for in occlusion of the colon a great proportion of the fluid that accumulates above the occlusion is absorbed again—certainly more than in the small intestine; this is due to the fact that in the colon the absorption of water from the bowel contents is very much more active than in other portions of the intestine. While admitting that these views are, generally speaking, correct, I do not believe that the results obtained from animal experiments can be applied to human beings, at least not in their totality. We know clinically, for instance, and from autopsy findings, that the statements made by Talma are not of universal application in human subjects, for it is a well-known fact that the accumulation of gas is often very considerable in cases of occlusion of the bowel with meteorism from stasis in man.

In contradistinction to the variety of meteorism considered in the preceding paragraphs, another form of meteorism, which I propose to call local meteorism, develops in another series of cases of acute occlusion of the intestine. This local meteorism seems to appear in all those forms of acute occlusion of the bowel in which certain complications supervene. For the production of local meteorism the lumen of the intestine must, in the first place, as in the other form, be completely occluded, and, secondly, there must be an additional complication, namely, serious interference with the circulation in the wall of the intestine and the mesentery belonging to the affected portions of the bowel. The disturbance of circulation usually assumes the character of venous

stasis ; in fact, this condition is necessary for the production of local meteorism. All these conditions are fulfilled, for instance, in volvulus and in kinking of the bowel, and these affections may be considered prototypes of the condition under discussion. The same applies to those rare forms of incarceration of the bowel that are occasionally seen to assume the character of internal herniform incarceration, a condition that naturally leads to disturbances of the circulation in the intestinal wall and the mesentery. Local meteorism does not necessarily produce a constant dilatation of the intestine above the occlusion, nor does it necessarily produce a gradually increasing degree of distention of the proximal loops of intestine (*i. e.*, toward the stomach) above the occlusion ; in other words, the appearances of local meteorism are not so uniform as those of diffuse meteorism from stagnation of the bowel contents. Occasionally, of course, this regularity may be observed within the bowel ; it may, in fact, be uniformly dilated above the area of occlusion, and the degree of distention may increase in an upward direction from the point of occlusion toward the stomach. In cases of local meteorism a peculiar appearance is presented : above and below the points of occlusion the intestine is seen to be perfectly empty, and only those loops that are situated between the two points of occlusion are dilated and dark blood-red. In general, the walls of this portion of the intestine are found to be thickened ; the bowel contents of the loops between the points of occlusion consist of gas, blood, and a mass of liquid material. A condition of this kind, when it is typical, is called local meteorism, and the description I have given is more valuable than an attempt to define the condition more exhaustively. The distention of the incarcerated loop at times reaches enormous proportions. In volvulus of the sigmoid a single loop may fill the greater part of the abdominal cavity. If it is very small and, in addition, lies hidden in the true pelvis, it may be impossible to detect any outward sign of it, and the abdomen need not show any distention at all (Zeidler). It can readily be understood why a great variety of different appearances may be presented in cases of complicated kinking of the bowel, for we can imagine how coils of intestine in a condition of so-called local meteorism alternate with unaffected coils. In order further to illustrate my meaning I will append the report of an autopsy by Küttner :

"The abdominal cavity of the subject was very much distended. The body was that of a man who died thirty-six hours after the appearance of the first symptoms of occlusion of the bowels, the proximate cause of death being collapse. When the abdominal cavity was opened, it was at once noticed that the two halves of the abdominal contents were not of the same color. There was meteorism which was not uniform, but was unevenly distributed over different portions of the intestine. The intestinal convolutions situated in the right hypochondriac region presented a normal color ; in fact, the color was somewhat paler than normal. The thickness of the intestine in this area was also normal. In the left hypochondriac region an entirely different condition was found, for here the small intestine was dark bluish red, enormously distended, and very

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meteoristic. The two masses of small intestine were distinctly differentiated, chiefly by their color, but also by the presence of meteorism in one mass of convolutions and its absence in another. The intestines were drawn apart and the following picture was seen in the deeper portions of the abdomen that were exposed to view. In the region of the left sacro-iliac synchondrosis a large mass was found, consisting of four pieces of intestine that were quite flat, folded into a number of fine ridges, and perfectly white in color. The mass was made up of two pieces of the large intestine twisted around each other in a spiral manner, and coming from the left side of the abdomen. They were crossed by two other pieces of the small intestine coming from the right. The portion of the large intestine that was included in this mass was the base of the sigmoid flexure; pieces of the large intestine entered the mass from above and passed behind the pieces of the small intestine. They reappeared in the region of the cecum as the blood-stained, distended coils of the sigmoid flexure in a condition of meteorism. The two pieces of the small intestine came from the right, passed below and behind the two coils of the sigmoid flexure that were rotated around their own axis, and reappeared in the left hypochondriac region as red and meteoristic coils of small intestine. It was found, therefore, that the mass consisted both of portions of the sigmoid flexure and of portions of the ileum (chiefly from its lower part). This could be demonstrated by the fact that the lower flattened piece of small intestine was buried in the colon for a distance of over five inches."

The subject of local meteorism of the bowel in occlusion is so important that I do not hesitate to quote the results of Kader's experimental work (already referred to above) at some length. The following is a summary of Kader's clear description of the pathogenesis of local meteorism, and of his arguments and conclusions:

When coils of intestine are strangulated to a moderate degree,—that is, if ligatures are placed around such loops of intestine and their mesentery, and if these ligatures are tied sufficiently tight to permit the entrance of a certain amount of arterial blood into the arteries and arterioles of the area of intestine and mesentery that is ligated off, but at the same time not so tightly as to prevent the outflow of venous blood from the venules and veins of this area,—the strangulated coil of the intestine and mesentery immediately assumes a dark reddish-blue color. This experiment is particularly valuable in that it artificially creates precisely the same circulatory conditions in the intestine and the mesentery as are actually seen in the majority of cases met with in pathology. By this partial experimental strangulation venous stasis occurs. As a result, extravasation of serum takes place into the wall of the intestine, which in this way becomes thickened, frequently to a considerable degree; the walls may become twice as thick as normal, and from the presence of extravasated blood, they may become very hard. At first only serum exudes from the blood-vessels; later, both blood-corpuscles and serum are forced out through the vessel-walls as a result of the severe degree of venous stasis that soon develops. At first, as

we have said, the blood enters only the wall of the intestine, but after a time it passes out of the wall of the intestine in both directions—that is, it enters both the lumen of the affected coil of intestine, on the one hand, and the general cavity of the abdomen, on the other. Within a very short time, often in the course of a few hours, the loop of intestine that is in this condition of venous hyperemia becomes completely paralyzed, so that we see that strangulation of the bowel in addition to producing venous hyperemia soon produces complete muscular paralysis. Within the affected loop of intestine itself gas soon begins to develop ; it seems to make no difference in this respect what portion of the intestinal canal is strangulated ; it also seems to be a matter of indifference whether the strangulated loop is long or short, whether considerable quantities of bowel-contents are present in the intestine, or whether the bowel is completely empty when the occlusion of its lumen is brought about. In all instances the loop of intestine situated above the strangulation became distended and infiltrated with blood, so that its walls became very tense and the circumference of the bowel is increased to many times its normal size. The tension within the strangulated loop of intestine may become so great that considerable portions of normal intestine situated above and below the strangulation are drawn into the strangulated area together with a certain part of the mesentery that belongs to the intestine situated outside of the constricting area. This, of course, can happen only when the tension is very extreme and when the constricting ring is sufficiently wide to permit the passage of some of the intestine and mesentery situated above or below it. All these changes—namely, venous hyperemia of a loop of intestine, thickening of the bowel-wall, paralysis of the intestinal musculature in the strangulated area, and local meteorism—may all develop completely within four to eight hours after strangulation of the bowel is brought about. Other sequelæ of experimental strangulation of the intestine that follow the events just enumerated are thrombosis of the vessels of the mesentery and later gangrene of the tissues supplied by the occluded blood-vessels. If gangrene supervenes, perforation is apt to occur. Kader has called particular attention to a capillary form of perforation of the bowel that may occur in these cases. While in many instances the hole produced by the perforation is fairly large and distinctly visible, the capillary form of perforation that may occur in this condition is frequently barely visible to the naked eye ; nevertheless, the opening produced suffices to allow an escape of the gas accumulated within the loop of intestine. As soon as this occurs, the distention and dilatation disappear and the loop collapses.

Kader performed a large number of control experiments in order to discover the exact cause of all these different results. He found that the development of gas in the coil of intestine situated between the two occlusions as well as most of the other phenomena were chiefly and primarily due to the disturbance in the venous circulation of the wall of the affected loop. It would be impossible to describe all the different experiments he performed in order to prove this proposition, and I

will limit myself to describing only the following experiment : If artificial stenosis of the intestine alone is produced in some one part of the bowel, so that the mesentery belonging to this portion of the intestine is not included in the stricture, but the mesentery belonging to the loop of intestine situated immediately below the point of stricture is ligated, meteorism from stasis will be discovered both in that portion of the bowel that is situated above the ligature and in the loop of intestine that is situated below this ligature. In the latter loop the passage is occluded above but remains patent below, and yet local meteorism of the kind already described occurs as soon as the mesentery is ligated. The reader is referred to the discussion in the section on the Pathogenesis of Meteorism (p. 138) for details as to the exact manner in which meteorism develops in the intestine under these conditions. Generally speaking, the finer mechanism of the process is approximately the following : The relative proportion of gas that is developed in the intestine and the relative proportion absorbed by the blood circulating through the intestinal wall is disturbed. Kader, as has been said, argues that interference with the venous circulation of the intestinal wall can also be regarded as responsible for the paresis of the intestinal musculature that is usually seen in local meteorism of a coil of intestine. I do not think, however, that he has definitely excluded a direct effect of the strangulation upon the nerves in the mesentery supplying the affected coil of intestine, for it can well be imagined that the pressure exercised by the ligature might cause constriction of these nerves and in this way lead to paralysis of the muscular coat of the intestine. Personally, I am very strongly inclined to the belief that the latter event may occur.

Certain writers, for instance von Leichtenstern and other leading authorities, do not agree with Kader and others with regard to the constant occurrence of local meteorism under the conditions described above. It appears, however, that this phenomenon, namely, local meteorism, does, as a matter of fact, appear with a certain regularity in all the cases in which the conditions are favorable for its development. I may say that it has been seen and positively recognized in the majority of cases in which the conditions are favorable. At the same time there is no doubt whatever that it may occasionally be absent in strangulation of the bowel ; but the absence of local meteorism must be considered the exception ; its presence, the rule. If it is absent, moreover, we should always look for special conditions or complications of the primary lesion which can be made responsible for the absence of this significant phenomenon. Kader himself has called attention to one factor of this character which, under certain conditions, may be made responsible for the absence of local meteorism, namely, the occurrence of capillary perforation of the affected loop of intestine. It has already been shown that as soon as capillary perforation occurs, the gas can escape from the distended and dilated loop of bowel, so that the intestine consequently collapses. In a case of this character, then, local meteorism would be absent. There is another possibility, however, that may produce the

same effect—*i. e.*, that may prevent local meteorism from appearing or may allow it to disappear after it has once developed in a strangulated loop of intestine. This second possible factor will be described below in the section on Internal Incarceration of the Intestine. The case that I will quote to illustrate my meaning, and to demonstrate that meteoristic distention of a strangulated loop of intestine does not necessarily always occur, was one in which a loop of the ileum was incarcerated. In this particular instance the bowel contents contained in the loop could pass downward without difficulty, as the passage from the ileum toward the cecum was perfectly free.

The condition of the peritoneum in strangulation of the intestine varies greatly. Very much will depend on the anatomic nature of the primary process causing the occlusion of the bowel, and still more will depend on the rapidity with which the occlusion develops. If the course of the disease is extremely acute and the various symptoms appear in rapid sequence, all inflammatory changes may be absent. Under certain circumstances, even in this form of development, there may be a mild form of local peritonitis that is strictly circumscribed and limited to the area corresponding to that portion of the bowel that is occluded. In exceptional cases the same findings in regard to peritonitis may be observed, even when the disease is more or less chronic and its course protracted. In general, however, I think the following rule can be formulated : the longer the occlusion of the intestine persists, the more active, severe, and extensive the peritonitis that follows. The inflammation of the peritoneum seen in these cases of prolonged occlusion of the bowel may either be serofibrinous in character or may furnish a purulent, a sanious, or a hemorrhagic exudation. It is impossible to discuss the details of the morbid appearances presented by these different forms of peritonitis in this disease. (The reader is referred for all these features, as well as for a discussion of the different etiologic factors that can produce these different forms of peritonitis, to the section on Peritonitis, in which all these matters are described in detail.)

I merely, however, wish to mention briefly here that recent investigations by a number of competent authorities seem to point to the fact that the form of peritonitis that develops in acute occlusion of the bowel must be studied from a special point of view and differs materially from all other forms of peritonitis. This new point of view is the following : It can be shown that the wall of the intestine, particularly of those portions of the strangulated loop of bowel in which the muscular coat becomes paralyzed, and in which meteorism from strangulation develops, becomes permeable to bacteria present in the bowel contents. As soon as micro-organisms from the bowel contents penetrate the bowel-wall and come in contact with the serosa, peritonitis naturally develops. In some instances a severe general bacterial infection may supervene before local peritonitis can develop. Both these consequences of the penetration of the intestinal wall by bacteria are occasionally seen. Kocher emphasizes particularly that deterioration of the intestinal wall,

allowing micro-organisms to pass through it, occurs not only in strangulation occlusion, but also in obstructions that do not directly affect the mesenteric vessels. It occurs, in fact, in consequence of distention above the occlusion.

No description will be given here of the anatomic changes seen in numerous other organs of the body in occlusion of the bowel. So many factors play a part in producing the great variety of lesions of other organs that are occasionally seen as complications of occlusion of the bowel, that it is impossible to do justice to all of them and to study them from a common point of view.

CLINICAL FEATURES.

Symptoms.—Stenosis of the bowel almost without exception develops slowly and gradually. This statement applies to all forms of occlusion of the bowel, the organic nature of the lesion being immaterial. In very rare cases the condition develops in the course of a few days; in the overwhelming majority of cases, however, several weeks or even months must elapse before appreciable narrowing of the bowel lumen occurs. In the majority of cases, therefore, the course of the disease is slow and gradually progressive. It may occur, for instance, that an exudation forms within the abdominal cavity that increases with great rapidity. In an event of this kind certain loops of intestine may become anchored and fixed and their lumen may become narrowed within the course of a very few days; or, again, some foreign body may enter the intestine, act as an obstruction, and in this way suddenly cause narrowing of the lumen and interfere seriously with the passage of the intestinal contents. But, as said, the overwhelming majority of cases of stenosis of the bowel are due to some slowly developing, progressive process. Among these processes I may mention stricture of the intestine as a result of cicatricial contraction, malignant, or very rarely benign, tumors which compress the intestine or narrow its lumen from within, peritonitic fixation of the bowel, compression by tumors from without.

When we consider that in the majority of instances the stenosis of the bowel develops very slowly and progressively, we might also expect that the symptoms produced by this condition would be slow and insidious in their onset. As a matter of fact, this is often the case, but there are many instances in which the reverse seems to be true. It is an important fact that in many cases of stenosis of the bowel that develop very slowly and gradually, serious disturbances of the function of the bowel may be completely absent for a remarkably long time. This state of affairs may persist for a certain time, when suddenly and unexpectedly a variety of distressing symptoms appear. This may be particularly startling, as the absence of all functional or other disorders for a long time leads the patient and the medical man to the belief that the individual was perfectly healthy up to the time when the first serious symptoms appeared.

The primary cause of all the fundamental symptoms of narrowing of the bowel lumen is interference with the onward passage of the intestinal contents. Directly the passage of the bowel-contents through the stenosed area becomes difficult, symptoms appear. The severity of the symptoms and the time of their appearance will largely depend on the character of the bowel-contents: when thin or liquid, they will, of course, be able to pass through portions of the bowels that are in a condition of advanced stenosis; when, on the other hand, the bowel contents are habitually solid, symptoms will develop much sooner, for the passage of material of this kind through a stenotic area is naturally rendered difficult much sooner. For this reason stenosis of the small intestine *cœteris paribus* remains latent for a much longer time than stenosis of the large intestine. Stenosis of the colon below the sigmoid flexure, where the bowel contents, owing to the increased absorption of water in this area, are usually solid, produces symptoms sooner than stenosis in any other portion of the intestine.

In stenosis of the large intestine and of the rectum one of the first symptoms, as a rule, is constipation. If the patients are inclined to pay attention to their health, and as far as possible to regulate their bodily functions themselves, they will attempt to relieve this condition of constipation within a few days after it occurs; in other words, they will use artificial means to get rid of the accumulated bowel contents and in this way prevent the appearance of those symptoms that naturally follow stagnation of fecal matter in the large intestine. Gradually the condition of constipation increases in obstinacy and becomes more or less intractable. The patients complain of a feeling of distention and swelling in the abdomen; there is loss of appetite, and occasionally the patients suffer from attacks of nausea. They are gradually obliged to use powerful evacuants, and finally are forced to have recourse to the most energetic purgatives. Sometimes we see that even during this period of obstinate constipation an attack of diarrhea occurs spontaneously and in this way relieves the stasis of bowel contents and aids in the evacuation of the accumulated fecal material. Such attacks of diarrhea occurring in the period of constipation may last for one or several days; as a rule, very large, even enormous, masses of fecal matter are evacuated during these attacks. The material passed is frequently extremely offensive in character; at the same time very large quantities of gas are generally expelled from the bowel during these attacks. After an attack of diarrhea of this kind, the patients usually feel very much relieved, and frequently believe that they are perfectly well again. After a time, however, constipation returns, the same symptoms develop, the patients suffer great distress, and are finally again relieved by an attack of spontaneous diarrhea. Attacks of serious constipation may alternate with attacks of spontaneous diarrhea for a long time, the different attacks occurring at long or short intervals. After a time a new symptom develops: the patients suffer paroxysmal attacks of colic; these I will describe presently. As soon as colic appears, the situation may be considered comparatively grave; at this

period, at all events, both the medical man and the patient possibly realize for the first time that the disease is a serious one. In some instances, of course, both the medical man and patient come to this conclusion at an earlier stage of the affection and before the attacks of colic begin to dominate the scene.

I have already mentioned that constipation is more common in stenosis of the large intestine than in stenosis of the small intestine. It may even be said that narrowing of the lumen of the small intestine in the great majority of cases produces no constipation proper whatever. The explanation of this difference between stenosis of the small and of the large intestine has already been given—it is due to the difference in the character of the intestinal contents in the two parts of the intestine : in the small intestine it is thin and watery, whereas in the large intestine it is more solid. It may happen, therefore, that quite severe degrees of narrowing of the lumen of the ileum or jejunum produce no symptoms for a long time, and may thus be quite undetected.

It must not be imagined from what has been said that constipation is a necessary sequel or symptom of stenosis of the large intestine, for occasionally cases of most pronounced stenosis of the colon are observed in which constipation is absent and in which the character of the stools is either normal or only shows insignificant changes. The reader is, therefore, expressly warned of the danger of attempting to make a differential diagnosis between stenosis of the small intestine and stenosis of the large intestine on any diagrammatic scheme, and from diagnosing the latter condition if constipation is present ; the former, if it is absent. Constipation may be absent in stenosis of the large intestine, particularly in certain forms of slowly progressive stenosis of the region of the ileocecal valve, or in gradually progressive stenosis of the cecum or even of some of the first portions of the ascending colon. In all these conditions the character of the stools may vary greatly. I have seen cases of stenosis of the large intestine in which the patients suffered from a most obstinate form of constipation ; and, on the other hand, I have seen cases of the same affection in which a daily evacuation of the bowel contents occurred with the greatest regularity and in which the first sign that pointed to the existence of anything pathologic whatever in the bowel was a paroxysm of colic. In fact, I remember seeing cases of very advanced stricture of the sigmoid flexure in which a daily evacuation of the bowels occurred regularly. I recall the case of one patient in particular, a woman of fifty-eight years, who gave the following history : Up to February 24, 1886, the bowels acted daily ; the material deposited was quite normal in appearance and constitution. The patient up to this date felt perfectly well in all respects and was able, without difficulty, to perform the heavy manual labor that her occupation as a washerwoman imposed. On February 25th she suffered from an attack of colic. The paroxysm appeared suddenly and without warning, and was very violent. In the course of the next few days she suffered repeated attacks of this kind. She consulted a medical man for the first time on March 3d, who examined her and found

the syndrome of occlusion of the bowel. On March 6th—that is, on the tenth day of the disease—the woman suddenly died in collapse. An autopsy was performed, and the following conditions were found: At the lowest end of the sigmoid flexure, just where this portion of the bowel merges into the rectum, there was a hard carcinoma. The growth was about 2 cm. broad, very hard, and was ulcerated. The neoplasm caused stricture of the intestinal lumen; the stenosis of the bowel was so far advanced that only the point of the little finger could be passed through the stenosed passage. Notwithstanding this advanced degree of stenosis the patient had had a regular daily and normal evacuation of the bowels up to ten days before her death. In order to explain this peculiar phenomenon the musculature of the intestine above the stricture was examined, and it was discovered that in this instance an enormous degree of muscular hypertrophy of the bowel-wall had developed that extended throughout the whole large intestine as far as the ileocecal valve. A case of this kind is very instructive and throws much light upon the clinical picture of stenosis of the bowel; at the same time it teaches us much with regard to the symptomatology and the pathology of carcinoma of the intestine: it shows, especially, that enormous difficulties are frequently met with in making a diagnosis in these conditions.

In the preceding paragraphs the attacks of colic that occur in stenosis of the bowel have frequently been referred to. This symptom is a very important one and occupies a prominent position among the signs and symptoms of stenosis of the bowel. As a matter of fact, it may be regarded as one of the most conclusive symptoms of this disease. When attacks of colic appear, they indicate that the morbid process is well advanced, as they occur only when the stenosis has progressed to a certain degree; occasionally, as we have seen, they constitute the first symptom of stenosis. In the latter case attacks of colic are much more significant; they are also more striking and surprising to the patients themselves, for many subjects with stenosis of the bowel suffer from no symptoms or functional perversions whatever until the first attack of colic suddenly and unexpectedly appears. In looking over my notes of cases I find a large number of reports of this kind. Examples are frequent, therefore, of patients with stenosis of the bowel who consider that they were perfectly healthy until the first attack of colic appeared.

The attacks of pain that these patients suffer are frequently exceedingly severe, and may become so excruciating that the sufferers scream out, twist and turn in their agony, and scratch the walls with their fingernails; the pain, however, is usually endurable, even though severe. In other cases again the attacks of pain are not so severe—may, in fact, be quite mild. Sometimes the pain is experienced only in one small portion of the abdomen; in other words, it remains strictly localized. The site of the pain usually corresponds exactly to that of the stenosis. In other instances the pain is more diffuse and radiates all over the abdomen or even upward toward the thorax. When this occurs, a

feeling of oppression and of dyspnea may be complained of by the patient. In exceptional cases the pain seems to originate in some other portion of the bowel than the stenosis. In stenosis of the cecum, for instance, the pain may seem to begin in and radiate from the left side of the abdomen. A paroxysm of pain in stenosis of the intestine is frequently accompanied by vomiting ; these attacks of vomiting may occasionally be exceedingly severe. The vomited material usually consists of the contents of the stomach, of bile from the intestine, and of mucus. In all cases, without exception, there is constipation—that is, none of the bowel contents is evacuated during the paroxysm. This symptom is, of course, usually overlooked, since it is not conspicuous unless the paroxysm of colic persists for a relatively long time. Very frequently gurgling and rolling sounds and noises are heard in the bowels ; at the same time there is usually meteorism, so that the abdomen is seen to be distended either over its whole extent or in certain limited regions.

There is nothing particularly characteristic of stenosis of the bowel in the paroxysms of pain as we have described them. There is one sign, however, that is frequently seen during these attacks of intestinal colic due to stenotic narrowing of the intestinal lumen that may be considered absolutely typical and in a sense pathognomonic for this lesion ; I refer to the appearance of visible peristaltic movements of the bowel. If the abdomen of a patient with stenosis of the bowel is examined during the attack of colic, tonic contractions of the coils of intestine and increased peristaltic action of the intestine in general can usually be seen through the abdominal wall. At the same time some of the loops of intestine are seen to stiffen and to relax alternately (the reader is referred to the illustrations of this condition in the plates appended to this volume). No attack of colic can be considered due to enterostenosis unless this sign appears, and the discovery of this phenomenon, namely, visible peristaltic movements and tonic contractions of loops of intestine, must be considered almost pathognomonic of the disease. These paroxysmal pains are never observed without rigidity of the intestine or increased peristalsis. Their intensity is in accord with the intensity of the motor activity of the bowel—both manifestations hang together, clinically and pathogenetically. It may occur that in less marked contraction of the intestine sensory excitation is so slight as not to be termed pain. This peculiar stiffening of the intestine and the increased peristalsis of the bowels will be referred to again, and these phenomena will be considered from a variety of points of view in subsequent sections. The sign is of such paramount diagnostic importance that I consider it necessary to enter here somewhat minutely into its pathogenesis.

In the general part of this work (compare p. 70) attention has been directed to the fact that the physiologic peristaltic movements (of the small intestine) may occasionally become visible under perfectly normal conditions—that is, in patients whose abdominal walls are very flaccid and very thin (compare Plates I.–III.). The physiologic move-

ments of the bowel that are occasionally visible through the abdominal walls are entirely different, however, from the pathologic contractions and pathologic peristaltic movements now under consideration; the former are characterized by slow, sluggish movements, whereas the latter are highly energetic and vigorous. No one who has ever seen the increased peristaltic movements of the bowel and the stiffening of the coils of intestine that occur in stenosis of the bowel will confound this sight with the appearance presented by sluggish physiologic peristalsis of the bowels. I have also called attention to the fact that the peristaltic movements of the bowels may be pathologically increased by a great variety of different causes. While this is undoubtedly the case, I must add that this physiologic peristalsis only rarely becomes sufficiently intense and vigorous to be visible through the normal abdominal walls in other diseases of the intestine than those that are complicated by stenosis of the bowel. Such vigorous peristaltic movements of the bowel are particularly apt to occur in forms of stenosis of the intestine which develop slowly and progressively. This can readily be understood when we remember that in slow and progressive stenosis, hypertrophy of the muscular coat of the intestinal wall always occurs, so that the contractions of the bowel-wall are particularly energetic and forcible under these conditions. We must always, however, be guarded and careful in our diagnosis when confronted with a case in which the peristaltic movements of the bowels are visible through the abdominal walls. Special care is necessary when peristaltic movements only are seen and the stiffening of the bowel due to tonic contractions is not visible at the same time. Quite frequently peristaltic movements of the bowel may be visible through the abdominal wall, even though no stenosis of the intestine is present (compare p. 75). Whenever tetanic contractions of the intestine become visible through the abdominal wall, we can be more positive in our diagnosis. These tetanic contractions appear in paroxysms, and are characterized by a stiffening of the loops of intestine, so that the outline of the stiffened intestine becomes distinctly visible and forms a picture in relief on the abdominal wall. It is true that tonic contractions of the intestine *per se* may occur in other diseases, may even be so severe that the lumen of the intestine is completely obliterated. Contractions of this kind, however, which are not due to stenosis of the bowel, never become visible through the abdominal walls in tetanus of the bowels in violent acute enteritis, lead colic, and meningitis; it is a well-known fact that in none of these conditions are intestinal contractions seen on the abdominal wall. In stenosis, on the other hand, the picture presented on inspection of the abdomen is perfectly typical—viz., coils of intestine stiff from tetanic contraction which rise above the level of the abdomen. This phenomenon is usually developed when the loop of intestine that is undergoing tetanic contraction is filled with gas and liquid or pultaceous material, or when the walls of the piece of intestine that is greatly distended by gas or fluid or pultaceous material are in a state of rigid contraction. I feel justified in making the statement from my personal

experience that a phenomenon of this kind is seen only in those diseases that are characterized by occlusion of the intestinal passage, for only in this condition is there a combination of the factors which favor the development of this sign. These conditions, as has already been shown, are the accumulation of gas and of liquid material in some portion of the intestine, and the existence of some obstruction in the terminal portion of this loop of intestine which prevents the passage of the accumulated gas and of the liquid material. When these conditions obtain, the ordinary peristaltic movements of the bowel are insufficient to drive the stagnating material onward ; a compensatory process is then inaugurated—namely, increased contractions of the intestinal wall. As soon as these contractile efforts become excessive, the musculature of the intestinal wall reacts by tetanic contractions, and, further, as the muscular coat of the bowel-wall in most of these cases is hypertrophied, this tetanus become exceptionally energetic. The last-named point, namely, the excessively energetic character of the tetanic contractions of the intestinal muscular coats, is particularly important and deserves special attention, for we know that in no other affection of the bowel does muscular hypertrophy develop to such an advanced degree as in chronic stenosis of the intestine ; consequently, the peristaltic movements of the bowel and the tetanic stiffening of the coils of intestine are never so marked as in this condition. When all these factors that lead to the development of excessively energetic peristaltic movements and of vigorous tetanic contractions of the intestine in chronic stenosis are considered, it will be understood why in this condition particularly the pathologic movements of the intestine become clearly visible through the abdominal walls and constitute so important a symptom of chronic narrowing of the bowel lumen.

In general, peristaltic and tetanic movements of the intestine appear together in attacks of colic that are due to stenosis of the lumen of the bowel. In the majority of instances the two forms of movement of the bowel alternate. If the abdomen is carefully inspected, certain stiff coils of intestine will become clearly visible that look like convolutions of resting snakes ; between these masses are seen other loops of intestine that are performing winding and twisting movements. At the same time these loops of intestine become contracted first in one place, then in another ; they are also seen to change their situation either very rapidly or quite gradually ; at the same time loud gurgling and rolling noises that are occasionally accompanied by metallic sounds may be heard over the intestine. A very striking picture is presented when the paroxysm occurs in the small intestine, and the appearance of the abdomen under these conditions is very interesting. A great variety of contractions and other forms of movements are seen. In one place sausage-shaped ridges may suddenly appear, with the formation of deep depressions in their immediate vicinity. In the course of a few seconds the swollen ridges disappear from one part of the abdomen only to reappear in another. The disappearance of these sausage-shaped ridges of stiffened intestine is usually accompanied by loud gurgling sounds.

One of the most characteristic features of these visible movements, and one that I wish to call particular attention to, is the fact that these stiffened coils of intestine never remain visible in the same place for any length of time. The sudden conversion of a coil of intestine that is possibly distended and inflated, but is perfectly soft, into a tense tube with stiff walls, which projects considerably above the level of the abdominal wall, can often be distinctly seen and felt. A coil of intestine, when thus contracted and stiffened into a tube with hard walls, can be grasped between the fingers and almost taken up into the hand. As a rule, as I have said, this contraction and stiffening only last for a short time, usually the former state of elasticity being regained within a very few moments. After the expiration of a few minutes, however, the same process is repeated, the intestine stiffens and is again converted into a rigid tube, and again after a few seconds regains its elasticity. During this whole process the patients suffer the most violent distress and pain; this is quite natural, of course, for the paroxysms of colic are directly caused by the tetanic contractions of the intestine (see p. 148).

The duration of a complete paroxysm of colic varies greatly. In stenosis of the bowel the attacks of colic may last for a long or for a very short time; they may even persist for several hours with short interruptions. Relief is usually obtained by the expulsion of gas, but the paroxysms never cease completely until liquid, or in very rare instances solid, feces are evacuated. In some instances it may happen that this initial paroxysm does not disappear at all, but passes directly into the symptom-complex characteristic of complete intestinal obstruction.

The attacks of colic may recur after a few days or not for many weeks. The determining factors are the cause of the colic and the exact conditions existing in the bowel. If the attack is started, for instance, by the ingestion of indigestible material which leaves a large residue and thus causes occlusion of the bowel, an attack does not necessarily occur for many weeks. In the interval between these attacks a patient with stenosis of the bowel may enjoy perfectly good health, provided proper care is taken to keep the bowels regularly open. In cases of advanced stenosis attacks of colic usually recur at intervals of four, three, or only two days. During the intervals between the attacks these patients also suffer great distress, and thus differ from those patients in whom the stenosis is not so far advanced; for the latter patients, as already mentioned, may feel perfectly well between the different paroxysms. When the stenosis is far advanced and the attacks of colic recur with great frequency, the abdomen remains permanently distended, so that among other things the breathing is seriously interfered with, and there is consequently dyspnea. The patients suffer from loss of appetite and from frequent attacks of vomiting. Finally, as the stenosis progresses, attacks of colic occur every day, until ultimately the symptom-complex of complete occlusion of the bowel develops.

We have seen that the attacks of intestinal contraction do not occur

uninterruptedly : they may disappear only to reappear at some later time. Sklodowski emphasizes the division into painful periods and single attacks. This phenomenon can readily be explained. Two factors are chiefly responsible for producing these attacks of alternating contraction and relaxation. In the first place, we must remember that the muscular coat of the intestine when it undergoes tetanic contraction performs an amount of work that is in excess of its ordinary functions ; consequently it soon undergoes fatigue and naturally relaxes. In the second place, the tetanic contraction of the intestinal wall removes the primary cause of this contraction, for it forces the accumulated mass of gaseous or semisolid bowel contents onward. In this way the irritant is transferred from one portion of the bowel into some other portion situated further down. Here the gaseous and semisolid material again irritates the intestinal mucosa and again produces tetanic contraction of the wall of the intestine. In this way we can readily explain why the spasm relaxes in one portion of the bowel only to appear in another, and why, after a time, the relaxation again gives way to tetanic rigidity. Attention must also be directed to another very important point which must always be borne in mind in the examination of a patient suffering from stenosis of the bowel and attacks of colic. This point is, that contraction of the intestine may be produced by some external stimulation, especially tapping the abdomen or slapping the abdominal wall with a cloth wrung out of cold water. As a matter of fact, contractions of the bowel may be started in subjects with thin abdominal walls by the mere irritation exerted by a draft of cold air, so that occasionally an attack of tetanic contraction of the bowel—in other words, a paroxysm of colic—may be brought on when the bedclothes are removed and the abdomen is exposed for examination.

What causes these conditions that are characterized by pain and tetanic rigidity of the intestine with increased peristalsis? In the first edition of this work I was chiefly concerned to give prominence to the heretofore somewhat neglected great clinical importance of this syndrome in enterostenosis, and therefore expressed myself briefly concerning the pathogenesis to the effect that it appears when insufficiency of the hypertrophied muscular coat has set in and the fecal current has consequently stopped ; or when the stenosis has advanced so far that even the ordinary contents may momentarily block the narrowed orifice ; or, if the lumen is still sufficiently patent, when voluminous ingesta (asparagus, fruit kernels, lentils, and the like) have been taken. In the last case the condition supervenes suddenly in apparently perfect health. Since then Sklodowsky has considered the phenomenon exhaustively and arrives at the following conclusion : " The hypothesis which explains the colicky attacks exclusively on primary occlusion is theoretically entirely unnecessary, and we can dispense with it also from a clinical standpoint. The attacks and periods of painful rigidity may occur in relative, as well as absolute, occlusion. It is very likely that a closure of the lumen in relative stenosis may occur secondarily at the height of an attack in consequence of an unusual increase in pressure

or distention of the different sections. All factors that directly or indirectly cause motor activity of the intestine can occasion colicky attacks, because every acceleration of peristalsis in the upper loops causes an accumulation of contents and increased pressure in the adjacent intestine. A *circulus vitiosus* results: the peristalsis increases the stasis above the obstacle, and the stasis excites the peristalsis. It is for this reason that even weak irritation, that in a healthy intestine would be insignificant, may here lead to severe and protracted increase of motor activity of the intestine, causing painful attacks of tetanus." The statements of Sklodowski are convincing and seem to be correct. Only two points need be added. There appears to be a contradiction in the hypothesis I have accepted that attacks of colic can arise from insufficiency of the hypertrophic musculature, as each excludes the other—a paralyzed musculature cannot become tetanically rigid and cause colicky pains. This contradiction is only apparent, as experiment proves beyond doubt that the tetanic contraction, while not developing in a paretic part of the intestine, will nevertheless develop above such a part. I must maintain most decidedly that the severe pains are actual colicky pains—*i. e.*, are called forth by tonic contraction of the intestine. Simple pain from distention never attains such intensity—at least this has been my experience. But the salient point in Sklodowski's description, to wit, that characteristic attacks may occur, even during patency of the stenosis, is no doubt correct.

Another symptom of enterostenosis that we have mentioned is meteorism. This, however, is less important in the diagnosis than the attacks of colic. Meteorism does not occur so readily in stenosis of the bowel, for it can easily be understood that the gases developed in the intestinal contents and accumulating in the bowel can pass the stenotic area without difficulty provided the stenosis is not too extreme. Escape of gas may even occur in cases where the contents of the bowel are more or less solid and can no longer pass the obstruction. Even in cases of complete occlusion of the bowel meteorism may be very slight, provided the absorption of gas through the blood-vessels of the intestinal wall is sufficiently active to provide for the gas that develops in the intestine; the appearance of meteorism in stenosis or occlusion of the bowel is dependent primarily on this factor, and as long as the absorption proceeds *pari passu* with the formation of gas, meteorism does not develop. If the development of gas, however, becomes excessive, severe degrees of meteorism may be witnessed. This occurs chiefly when the intestinal contents contain much fermentable material and many fermentation excitants. Meteorism may, as has been shown in the preceding paragraphs, be either diffuse or local in character. The development of the one or the other form of meteorism will depend on two factors: in the first place, the actual quantity of gas that accumulates in the bowel; in the second place, the exact site of the stenosis; this point will be referred to again in considering the local diagnosis of stenosis and occlusion of the intestine. Even when meteorism is fairly constant in a case suspected to be one of stenosis of the bowel, its value

is of secondary importance in the diagnosis. This is chiefly due to the fact that meteorism may be due to so many different causes.

I should like to add a few words in regard to the character and constitution of the feces in these cases. Constipation is the rule, and is more pronounced the lower down in the intestine the stenosis is situated. Attention has repeatedly been drawn to the fact that the fluids of the intestinal contents are chiefly absorbed in the colon, and that the nearer the rectum is approached, the thicker the fecal material; consequently we may expect to find constipation more pronounced when stenosis of the bowel occurs near the rectum than when it occurs further up, for in the former instance the fecal material is normally more solid than in the latter. In stenosis of the small intestine, on the other hand, defecation may be perfectly normal for a long time or throughout the whole course of the disease. I have already called attention to this fact, and have also mentioned that the same condition may be observed even in cases of stenosis of the cecum. Another interesting complication of this constipation is the occurrence of attacks of diarrhea. There is another class of cases of stenosis of the bowel in which the patients suffer from a chronic form of diarrhea which may persist for many months. The appearance of chronic diarrhea in stenosis of the bowel is one of the most misleading symptoms, and frequently leads to serious errors in diagnosis. Some authors have advanced the view that this chronic diarrhea is always due to the existence of ulceration of the intestine, but Leichtenstern, who opposes this view, states that ulceration of the intestine is by no means always present in those cases of stenosis of the bowel that are complicated by diarrhea. Personally I agree with his view.

These attacks of chronic diarrhea are due to the same factors that cause those intercurrent attacks that are common in stenosis of the bowel. Both forms of diarrhea are due to certain catarrhal changes in the mucous membrane of the bowel above the stricture. These catarrhal changes of the mucosa are characterized by a hyperabundant secretion of intestinal fluids, which produce great dilution of the intestinal contents stagnating in these areas. The two forms of diarrhea, both the chronic and the intercurrent forms, are also due in part to stagnation of fecal masses and accumulation of gas in the portions of the intestine situated immediately above the stricture, for, as has been shown elsewhere, this stagnation alone is capable of stimulating the peristaltic action of the bowel (compare, for the mechanism of this process, p. 74). Very frequently the feces passed in the course of one of these attacks of diarrhea contain a considerable quantity of mucus. The presence of this mucus is also directly dependent on the existence of catarrhal conditions of the mucous membrane of the intestine above the stricture. The lower down the situation of the stenosis in the intestine, the more marked is the amount of mucus in the feces.

Occasionally blood and pus are also found in the dejecta. The admixture of these elements, however, with the bowel contents is less characteristic of chronic stenosis of the intestine, and can hardly be con-

sidered a symptom proper of this condition. Blood and pus in the feces may, however, be considered a sign of the existence of certain definite anatomic varieties of stenosis of the intestine; in other words, may be due to certain special factors and causes at work in certain forms of chronic enterostenosis. If the stenosis of the intestine is due to a carcinomatous stricture or to intussusception of the bowel, blood is often passed with the feces. Another condition in which blood is evacuated is stenosis of the bowel by very vascular adenomatous growths; finally, blood may be evacuated if the enterostenosis is due to the presence of progressive tuberculous or dysenteric ulcerations of the muscularis, or rather to the development of constricting cicatrices from healed ulcers of this character. In most instances cicatrized ulcers and rapidly progressive ulcers, of the dysenteric or tuberculous variety, will be found together. In a few instances the stenosis itself may possibly be the cause of a slight degree of enterorrhagia. Blood may be passed in the stools in uncomplicated stenosis when bleeding occurs from one of the distention or decubital ulcers that may develop in this condition immediately above the stricture. Another possibility is bleeding from hemorrhoids in simple stricture of the rectum, for hemorrhoids frequently develop in this form of stricture and must be considered the direct result of the stagnation of fecal material in the hemorrhoidal area.

Many authorities are inclined to attribute an almost pathognomonic significance in the diagnosis of stenosis of the intestine to certain peculiarities in the form of the solid dejecta passed. Statements of this character have been made so frequently that every medical man involuntarily thinks of stenosis of the bowel when he observes that the feces leave the bowel in certain peculiar shapes. It is not easy to define the exact shape of the fecal mass leaving the anus that is considered characteristic of stenosis. Different forms must be included under the name "stenosis feces." Occasionally small isolated and separated balls or nodules of fecal matter resembling sheep's dung are passed. The surface of each little sphere is irregular and is frequently scored with one or two furrows. In other instances thin, round, pencil-shaped, elongated columns of fecal material which may either be furrowed or have a smooth surface are passed. In still other instances the fecal material is flattened and is passed in long or short pieces, or one surface of the cylinder of fecal material may be flat and broad, the other sharp and wedge-shaped.

It is undoubtedly true that in stenosis of the intestine the fecal material frequently presents these abnormal shapes. It is established, moreover, that these changes in the fecal cylinder may be the immediate result of an intestinal stenosis. If fecal matter of this kind is continuously and permanently passed, the diagnosis of enterostenosis should always be considered, even when all other symptoms of narrowing of the bowel lumen are absent. The suspicion will be corroborated if fecal material of normal shape and normal bulk is never passed in the interim, and it will be still more strengthened if the feces frequently contain a certain quantity of mucus or in particular blood or pus.

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Notwithstanding all this, the diagnostic significance of ribbon-shaped, pencil-shaped, ball-shaped fecal material is very limited, and we should be exceedingly careful and circumspect in drawing any conclusions from the presence or absence of such abnormally shaped fecal masses in the diagnosis of narrowing of the bowel. In the first place, it is a well-established fact that in very advanced degrees of enterostenosis the form and the bulk of the fecal material passed may be perfectly normal, and no ribbon-shaped or otherwise distorted cylinders of fecal matter may ever be passed. If the stricture is situated in the small intestine, the cecum, or the ascending colon, it can readily be understood why the column of feces should be normal in outline and in bulk. Even if the stenosis is situated as far down as the junction between the sigmoid flexure and the rectum, the form and bulk of the feces may be normal. I know from personal experience that in cases of this kind daily and regular evacuations of normal feces may occur up to a few days before the death of the patient. As a matter of fact, it is by no means necessary that the form and the bulk of the feces should be abnormal even in those cases of occlusion of the bowel in which the stenosis is situated far down toward the anal end of the intestine. I need only to refer to the remarks made above with regard to the passage of pultaceous stools and even of chronic diarrhea in these conditions. A still more important fact to remember is the following: that dejecta may occasionally be passed which resemble in every respect the dejecta which are considered so characteristic of stenosis of the bowel, even though there is no enterostenosis. I have repeatedly observed the passage of so-called "stenosis feces" during life, and have then failed to find any anatomic evidence of narrowing or of any change whatever in the bowel after death. In simple habitual constipation, for instance, small ball-shaped masses of fecal matter are frequently evacuated. The individual fecal balls may be as small as hazel-nuts; they may be flattened on one or the other surface, or scored by one or two furrows. These ball-shaped masses are molded into this shape in the haustra of the colon; later, as they pass over the teniæ of the colon, their surface becomes indented and the peculiar furrow-shaped depressions are produced. If ball-shaped masses of fecal matter are evacuated in an individual who is poorly nourished, as not infrequently occurs in the neurasthenic or cachectic, for instance, as the result of a carcinoma of the stomach, and if all the fecal matter passed consists exclusively of such ball-shaped masses, and if this kind of feces is passed during the whole time the patient is under observation,—and I have repeatedly seen cases of this kind,—the medical man may easily be led to make the diagnosis of a stenosis of the colon, or will, at least, be very much inclined to consider such a diagnosis. I have myself been led repeatedly, in cases of this kind, to commit this error, and have then found in the further course in the former, on section in the latter, that no evidence whatever of stenosis of the large intestine could be discovered. What has been said with regard to the passage of these ball-shaped masses of fecal material applies with equal force to the passage of

elongated, pencil-shaped, flattened or rounded columns of fecal matter, for these, too, may be passed without any organic lesion of the intestine ; as a matter of fact, stools of this kind are frequently passed by patients who are essentially healthy, but who suffer from some slight functional disorder. I have seen such stools, for instance, in subjects who were very nervous, so that the bowel was in a condition of nervous spastic contraction. Again, I have seen them during inanition ; in other words, when the intestine was in a condition that has been called " hunger intestine." In the majority of these cases, of course, the existence of some organic form of stenosis of the bowel will be pre-supposed, but subsequent postmortem examination often shows that no such lesion exists.

In the preceding paragraphs all the essential symptoms of stenosis of the intestine have been described ; all the other symptoms that are occasionally observed in this disease are of entirely subordinate im-portance. The external aspect of the abdomen may, for instance, vary greatly ; in the first place, the seat of the stenosis exercises a great influence on the shape of the abdomen, and may distort it in different ways ; in the second place, the nature of the stenosis may vary and consequently produce various changes in the external appearance of the abdomen, according to the primary cause of the stricture. Finally, the appearance of the abdomen may vary in different phases of stricture of the intestine : if, for instance, the bowel lumen is occluded in the region of the hepatic flexure of the colon, the surface of the abdomen may, for a time, be perfectly normal in every respect, so that neither by inspection nor palpation can any deviation from the normal be discovered ; at some other phase of cases of stricture of the hepatic flexure of the colon, the bowel may be distended with gas, so that pro-trusions corresponding to the meteoristic loops of intestine can be dis-covered on inspection, and can usually be felt on palpating the surface of the abdomen ; again, at other times, there may be meteoristic protru-sions, rigidity and stiffening of certain intestinal coils, and the peculiar wave-like movements of the abdominal surface which we recognize as characteristic of increased peristalsis of the bowel. Occasionally, on brief inspection, a stiff, rigid loop of intestine may be mistaken for a tumor of the abdomen ; an error of this kind, however, is rapidly cor-rected, for within a few seconds, or at best minutes, the phantom tumor becomes softened and disappears from view, and palpation of the area of the abdomen in which the tumor appeared shows that it had disappeared.

In cases of stenosis of the bowel there is frequently a feeling of fluctuation on palpation, and occasionally distinct succussion-sounds can be produced, particularly above the stenosis. The presence of these two signs is quite natural, for fluctuation and succussion-sounds can always be produced in a dilated loop of intestine filled with a mixture of gas and liquid. Loops containing material of this kind, and, more-over, dilated, are frequently found in cases of stenosis of the intestine, particularly if the course of the disease is chronic. Occasionally the

stagnation of bowel contents becomes so considerable above the stricture and the mass of material accumulated in the loops of intestine is so enormous that the intestine above the stenosis becomes enormously dilated, and becomes so heavy that the dilated and distended coils sink downward to the posterior lateral parts of the abdomen when the patient occupies a horizontal position. If in a case of this kind the lateral aspects of the abdomen are percussed, a dull percussion-note will be obtained ; if now the patient is instructed to change his position, the heavy loops of intestine which are filled with fecal material will also change their position, so that the percussion-note in that portion of the abdomen that was occupied by these portions of intestine becomes clear. It will be seen that this change in the percussion-note following a change of the position of the patient, in the sense, namely, that percussion of the lateral aspects of the abdomen gives a dull note when the patient is lying on his back, and a clear note when he turns over on his side, resembles in every respect the percutory phenomena observed in cases of slight ascites, or in all cases in which a certain amount of fluid is present and freely movable in the peritoneal cavity. I have repeatedly found conditions of this kind in cases of stenosis of the sigmoid flexure and of the ileum, and we learn from this that loops of the large intestine as well as loops of the small intestine may under these conditions sink from their weight and occupy a position in the flanks of the patient. The differentiation between this condition and ascites or the accumulation of fluid in the peritoneal cavity is comparatively simple in many of these cases ; the following manipulation will usually enable us to arrive at a decision. The lateral aspects of the abdomen when the patient is lying on his back, or the other dull regions when the patient is lying on his side, should be tapped with short rapid blows directed vertically to the region in which the percussion-note is dull. If this short, rapid, vertical method of percussion elicits succussion-sounds, we may be certain that the fluid is contained inside of the lumen of the intestinal tube. In cases of stenosis of the bowel no definite and reliable information can be gained by percussion. This method of examination furnishes very uncertain results ; this is due chiefly to the fact that the percutory phenomena that may be produced are so manifold, and are dependent on so many different factors—such as, for instance, the degree of distention of the intestine, the proportion of gaseous, liquid, and solid matter that is present, the actual quantity of bowel contents present in a loop of intestine above the point of stenosis, etc. It is impossible, therefore, to formulate any definite rules with regard to the percutory phenomena observed in stenosis of the bowel. The information that can be obtained with regard to the clinical conditions existing is too general to be of value. Only one point is of diagnostic importance : it is that when local meteorism, strictly circumscribed to some one area of the bowel, can be discovered by percussion, we are justified in assuming that the stenosis is in this region ; whenever this condition is found, all our efforts should be directed toward discovering other signs of constriction of the bowel in this particular area. Local-

ized meteorism is of value only in the diagnosis of narrowing of the bowel *per se* when combined with other symptoms that point in the same direction.

In stenosis of the bowel a number of sequelæ are observed : in the first place, the structural changes of the bowel in the region of constriction produce certain definite consequences ; in the second place, the etiology of the disease usually leads to definite sequelæ ; in the third place, the stenosis *per se,* particularly if it persists for a long time, produces certain consequences which are deleterious to the general organism. This is, of course, self-evident. Sequelæ of a more general character are seen particularly in cases of narrowing of the small intestine. A lesion of this kind, as has repeatedly been mentioned, produces serious catarrhal changes in the mucous membrane of the intestine that may involve wide areas of the bowel, particularly of that portion of the intestinal canal that is situated above the stenosis. In addition to this catarrh ulceration may occasionally occur. Catarrh and ulceration of the bowel naturally pervert the normal functions of the intestinal secretions and the absorptive mechanism of the bowel-wall, and consequently seriously interfere with general digestion and assimilation. One of the chief symptoms of this impairment of the bowel function is the appearance of indican and of conjugate sulphates in the urine. In many of these cases the patients begin to emaciate greatly. These points will be referred to again in the consideration of the localization of stenosis and occlusion of the bowel.

PROGNOSIS AND COURSE.

Narrowing of the bowel lumen is always a serious matter, and a grave prognosis should always be given. The primary etiologic factors that are, in the first place, responsible for the development of enterostenosis, of course determine the exact degree of gravity of the prognosis ; in other words, the prognosis will vary in each individual case according to the etiology of the enterostenosis. As a matter of fact, however, there are certain definite forms of narrowing of the bowel which may undergo a spontaneous cure, so that in these cases the prognosis is very favorable ; these cases are, to give some examples, enterostenosis brought about by the presence of some foreign body or by the development of some polypoid benign neoplasm in the intestinal lumen. In both of these instances the case may recover. Then there are certain cases of invagination of the bowel producing stenosis that, under particularly favorable circumstances, may also recover, namely, when the invaginated portion of bowel is sloughed off and in this way patency of the intestinal lumen is again established. Other possible conditions that may lead to stenosis of the bowel, but may also recover spontaneously, are the development of a large exudate in the abdominal cavity, which compresses the intestine from without and in this way produces stenosis ; such an exudate may be absorbed, and in this manner the pressure on the intestine and the stenosis be relieved. Or, again, pres-

sure on the intestine may be exerted by a displaced uterus; here the organ may be replaced, the pressure relieved, and the stenosis permanently cured. The same good effects naturally follow artificial removal or cure of other conditions that cause compression of the intestine from without; I refer, for instance, to the operative removal of tumors of certain abdominal organs which exert pressure upon the intestine—the surgical or manipulative separation of peritoneal adhesions producing stricture of the bowel. Finally, in other instances, surgical interference may bring about a cure of enterostenosis by removing obstacles that are situated within the intestine itself and cause narrowing of its lumen. These considerations encroach on the subject of treatment and no further possibilities will be dealt with here, as all these points will be considered systematically in a later section on the Treatment of Enterostenosis.

If enterostenosis is allowed to run its course without any interference, the termination of the disease, in the great majority of cases, is unfavorable. The duration of the condition will vary in different cases, and a great many factors will determine the course of the disease and the length of time that must elapse before death occurs. The primary etiologic factors that produce the stenosis must, of course, be considered in predicting the course and in estimating the duration of the disease. Later, in discussing the different anatomic forms of enterostenosis, we shall describe the course and duration of each individual form (I may mention, for instance, that a patient suffering from simple cicatricial stenosis of the intestine will, *ceteris paribus,* live longer than a patient suffering from a carcinomatous stricture). Apart from the factors enumerated, which may be called primary, certain secondary factors also determine the course and termination of a case of enterostenosis—namely, those secondary developments that follow stenosis *per se.* Finally, unexpected complications and sequelæ must always be included in the calculation. One of the most frequent accidents of this kind which will be mentioned in order to illustrate my meaning is the following : Cases of stenosis are frequently seen which run a favorable course and are perfectly free from any serious or threatening symptoms whatever, when suddenly complete occlusion of the bowel occurs. In investigating the causes of the sudden turn for the worse,—viz., the sudden occlusion of the bowel,—it is found that some solid body that was contained in the intestinal contents (a fruit-stone or something of that character), or even a particularly hard piece of fecal material, has become engaged in the stricture and completely occluded it. In this way obstruction may occur that is frequently insurmountable. Another serious complication that may occur is the following : When the loops of intestine are very heavy from accumulated fecal material and sink down from their own weight, volvulus or kinking of the bowel may occur and produce complete occlusion of the lumen. This accident occurs with particular frequency in stenosis of the intestine situated at the junction of the sigmoid flexure and the rectum. In many of these cases the history of the patient is not very clear, and some of the

details of the course of the disease up to the time of this accident may not be known. If this is the case, the diagnosis of acute occlusion of the bowel without any chronic form of stenosis may be made. Occasionally the true state of affairs may, however, be recognized—that is, the existence of a gradually progressing stenosis of the intestine may be discovered, particularly if energetic peristaltic movements of the intestine and tetanic stiffening of the bowel are seen as soon as complete occlusion occurs. The appearance of the latter phenomenon justifies the conclusion that the muscular coat of the intestinal wall is hypertrophied, and in order to explain this hypertrophy, some obstacle to the onward passage of the intestinal contents must have existed for some time previous to the occurrence of the acute occlusion.

When none of these sudden complications appear and the ordinary course of the disease is not changed by any intercurrent complications or untoward events, the course of the disease is the following (this description applies only to those cases of stricture of the bowel that are of benign character and develop slowly and without complications): In the early stages hypertrophy of the muscular coat develops; this compensatory hypertrophy is usually sufficient to overcome the increased resistance to the propulsion of the bowel contents; consequently the passage of the bowel contents through the stricture proceeds in a normal manner for a time, and no stagnation of fecal matter occurs. The patients, as a result, feel fairly well during this period. Gradually, however, this compensatory process becomes insufficient in a manner that is analogous to insufficiency of all other forms of compensatory activities in any other organ or tissue. As soon as the hypertrophied muscular coat of the intestinal wall becomes insufficient, stagnation of fecal matter occurs, with all its results. It would lead us too far to discuss here the pathogenesis of this secondary muscular insufficiency; it is sufficient to remember that with the occurrence of this insufficiency and with the accumulation of fecal matter above the stricture, the intestinal wall in this portion of the intestine becomes distended. As a result, the muscular tissues within the intestinal wall also become distended and stretched; this, of course, interferes with their functional activity, for stretching of fatigued and degenerated muscular substance must necessarily be detrimental to its contractile power. In this way, then, a vicious circle of events develops, each one of which acts harmfully on the other. At an early period of this condition artificial stimulation of the peristaltic action of the bowel-wall may aid in the propulsion of the bowel contents, and in this way supplement the compensatory efforts of the muscular coat. As soon as this means fails and it becomes impossible to stimulate peristalsis and drive the intestinal contents through the stricture, the unavoidable alternatives are either death or, in rare exceptions, relief by operative interference. In another class of cases the lesions of the mucous membrane of the intestine that develop above the stenotic area, especially the ulcerations, determine the subsequent course of the disease. Sometimes decubital or stercoral ulcers perforate through the

bowel-wall and give rise to acute diffuse peritonitis, usually leading to the death of the patient. Again, in other instances, an adhesive form of peritonitis slowly develops before the occurrence of perforation of the bowel. When the latter event finally occurs, a sacculated fecal mass or a sanious phlegmon is formed in the abdominal cavity and produces a typical disease-picture. Many other septic manifestations and perforations into other organs may occur. These serious conditions are all the distressing results of peritonitis following perforation of the bowel. Sufferers from stenosis of the bowel that runs this course usually die from perforative peritonitis or one of the other consequences of perforation of the bowel.

SYMPTOMS OF INTESTINAL OBSTRUCTION.

Complete intestinal obstruction is a most horrible disease, in which the suffering of the patient is excruciating. This condition has been called ileus (*s. miserere s. passio iliaca*). Leichtenstern has written an excellent monograph on the historic development of the doctrine of ileus, which is characteristic of this author's thoroughness, for it is written with the greatest care and reviews almost the whole literature of the subject. The reader should refer for the changes in our views with regard to the clinical significance of this disease to his excellent work. It will be found that this disease has been known for many centuries, and that in the course of time the clinical conception of the older physicians has undergone many changes and modifications.

No uniform definition of the term " ileus " exists. In general, ileus means the collection of symptoms (symptom-complex) produced when the passage of fecal matter through the bowel is completely interfered with. The arrest of the passage of bowel contents may be due to a great many different conditions and may produce a variety of symptoms that differ greatly in individual cases ; thus, in one instance, the symptoms are merely those of occlusion ; in another there are, in addition, symptoms of strangulation ; as, furthermore, the anatomic basis of all these symptoms may vary greatly ; and, lastly, as stenosis of the bowel or occlusion of the bowel may be completely absent in ileus, medical men of the present day—and this works best in practice—employ this term only in those cases of arrest of the passage of feces which are accompanied by fecal vomiting.

Personally I believe that no harm would be done if the term ileus was entirely eliminated from the nomenclature of disease. The term is essentially useless, and is a survival of the past when the clinical conceptions as to the nature of this disease were radically different from our modern views. I do not wish to advocate a complete revision of our nomenclature, and am the last to advise throwing overboard ancient and thoroughly familiar clinical conceptions without due necessity. So long as these older terms designate distinct and clearly defined conditions, or so long as they signify a true clinical entity, they may be retained with profit, but when they fail to do this,—if, in other words,

they are obsolete,—why encumber our nomenclature with these old and useless terms? The question arises, for instance, whether the term ileus should be applied only to those cases of occlusion of the bowel in which feculent vomiting occurs, or whether the term should also be employed for those cases in which the serious symptom-complex of intestinal occlusion is present without fecal vomiting.

We know that fecal vomiting may occasionally occur without anatomic occlusion of the bowel, and, on the other hand, that the symptom-complex of intestinal obstruction may be present in a number of conditions other than occlusion.

[Sir W. H. Bennett,[1] under the title of "Feculent Vomiting which is Sometimes Curative," describes a number of cases in which feculent vomiting occurred and was followed by recovery. He further quotes a well-known physician to the effect that 10 per cent. of patients who have feculent vomiting recover without radical treatment.—Ed.]

Conversely, finally, total occlusion of the bowel may lead to the death of the patient without causing the development of the symptoms that are ordinarily grouped under the name of ileus. For all these reasons I believe that it is best to eliminate this term altogether from our present clinical nomenclature. Let me summarize my reasons for this again. In the first place, the word does not in all instances indicate that the same anatomic lesions are present; in the second place, it does not definitely indicate that the same functional perversions exist; in the third place, too much time is wasted nowadays in trying to define this term accurately and to determine exactly what it means; in the fourth place, there is always danger that the employment of this word in the practice of medicine may give the impression to those who do not care to think for themselves that it indicates a definite uniform pathologic state: in other words, it tends to degenerate into a meaningless term.

If we decide to give up the term ileus altogether, we are confronted with the necessity of devising new terms to designate the various artificial differences that have been formulated; this is necessary in order to include all those clinical features that may be met with clinically in the morbid phenomena of so-called ileus. It is especially necessary, for instance, to differentiate between a "mechanical" and a "dynamic, paralytic, or spastic" form of ileus. To the first group belong all those cases in which the symptom-complex under discussion is produced by some kind of mechanical occlusion of the intestinal lumen; in the second group of "dynamic" ileus are those cases in which the syndrome of occlusion of the bowel is presented, especially fecal vomiting, but in which, nevertheless, the lumen of the intestine remains patent. Cases of the latter kind develop whenever the muscular coat of the intestine becomes paralyzed, paretic, or spastically stenosed for one reason or another.

These terms, "mechanical" and "dynamic and paralytic" ileus, are useful and perfectly correct so long as the term "ileus" is retained. I doubt, however, whether the literature of this subject is rendered more

[1] *Brit. Med. Jour.*, 1900, vol. i., p. 691.

lucid by retaining this term, and whether the nature of the processes that
"ileus" is intended to designate will become clearer and more compre-
hensive if the use of this word is continued. Personally I am inclined
to the belief that this is not the case, and consequently have decided to
give up the term "ileus" for the reason chiefly that the group of morbid
phenomena which it is intended to delineate is indefinite, and also be-
cause the significance of the word itself varies. I have decided to adopt
another plan in describing the different syndromes that are usually in-
cluded under the name of "ileus," namely, to describe the different
phenomena presented, simply as symptoms of those primary conditions
that produce them. Such primary conditions are, in the first place,
paralysis of the intestine, which will be described in detail in a separate
section, and, in the second place, occlusion of the intestinal lumen, which
is now under consideration, and, further, enterospasm, the doubtful rela-
tion of which to ileus has already been touched upon. In addition,
there are a few isolated conditions which may possibly lead to the
development of what has been called ileus—for instance, a communica-
tion between the stomach and the colon.

Complete occlusion of the intestinal lumen may either develop slowly
and gradually as the result of a steadily progressive and chronic steno-
sis of the bowel, or it may appear suddenly and at once.

The symptom-complex that follows this accident may either be a
congeries of symptoms produced by the interruption of the passage of
fecal material alone, or it may be a combination of these symptoms with
others, produced by a variety of complications which may supervene
and are the direct result of other factors, as, for instance, impairment
of the circulation of the intestinal wall, perversions of the nervous func-
tions of these parts, and general infection or intoxication.

The symptoms usually grouped under the name of occlusion of
the bowel, and due to complete stoppage of the passage of bowel con-
tents in some area of the intestine, are hardly ever produced unless
the occlusion develops slowly—that is, unless it is the direct result of
a gradually progressing stenosis of the intestinal lumen. Very excep-
tionally all these symptoms may arise when the occlusion occurs sud-
denly—that is, when the intestine, previously absolutely free from any
obstruction, becomes acutely obstructed.

It is very important and of the greatest practical significance to
remember that the morbid phenomena in acute intestinal obstruction are
not, as a rule, produced by simple interruption of the passage of feces
through the occluded area, for this accident alone does not furnish a
satisfactory explanation for the development of the syndrome seen.
Another factor must be taken into consideration, a factor which I have
frequently mentioned, but which has only recently secured its full
measure of recognition from physicians in the study of these processes.
After von Wahl and his students (Zoege von Manteuffel and others)
established clearly the principles of "strangulation and obturation
ileus," they have doubtless found general acceptance among all sur-

geons. I refer to the circulatory disturbances in the intestinal wall and certain portions of the mesentery, and to certain perversions of the function of the nervous structures contained in these parts; complications of this kind develop in the majority of the cases of acute occlusion of the bowel which lead to an interruption of the passage of fecal material. Interference with the passage of feces; interference with the circulation of blood; and interference with the normal performance of nervous function may be considered the symptoms of *strangulation* of the bowel. In cases of this kind it may be said that symptoms of occlusion and of strangulation are combined. It is very important to recognize the true significance of all these conditions, for only by so doing can we understand the pathogenesis of the disease and the resulting pathologic processes. At the same time a thorough knowledge of all these points is necessary in order to treat this disease in a rational manner. The choice of methods and the therapeutic indications in general are primarily and essentially dependent on a grasp of the pathologic basis of the disease.

This account will, in the first instance, deal with the symptom-complex of simple uncomplicated occlusion of the bowel, and in particular, of that form of uncomplicated enterostenosis which comes on gradually and is the result of a slowly progressive stricture of the bowel. The reader should refer to the clinical description of stenosis of the bowel furnished in a preceding paragraph, as a knowledge of the symptoms of this condition is necessary to understand the symptoms described in this section. Complete occlusion of the bowel may develop from stricture or stenosis in a variety of ways. The primary process that produces stenosis of the bowel may be progressive in character and continue to develop until it finally leads to complete occlusion of the bowel. Or, again, complete occlusion of the bowel may occur at an earlier stage in the process of stenosis from the impaction of some foreign body accidentally present in the intestinal contents in the stricture. Or, again, stagnation of the bowel contents may occur above the stricture and cause the descent of this heavy portion of the bowel by gravity, and, finally, kinking of this dilated and heavy portion of the intestine that is situated immediately above the stricture, or the increased peristalsis and influx of copious contents from above, causes closure of the lumen at the stenotic point in consequence of unusually increased pressure and distention in the afferent section. Finally, the musculature in the intestinal wall of the loop of intestine situated immediately above the stricture may become insufficient, and in this way the onward passage of the bowel contents be rendered impossible. Whatever the exact process which leads to absolute occlusion of the bowel may be, the development of the typical syndrome of the complete clinical picture is usually preceded by a series of events which must be learned from the history of the case and have been described at length in the considerations on the symptomatology of stenosis of the bowel. Even in those cases referred to in the section on Stenosis of the Bowel, in

which the chronic progressive process of stenosis remains quite latent for a long period of time and then suddenly changes and presents the picture of occlusion of the bowel, the symptoms still differ materially from those which develop in cases of acute occlusion of the bowel occurring in a perfectly normal and healthy intestine. The symptoms of occlusion *per se*, it is true, are the same in both cases, but in the latter instance certain symptoms of *strangulation* are presented in addition, while all the phenomena that are due to the existence of hypertrophy of the intestinal muscular coat, and that are considered so pathognomonic of occlusion following chronic stenosis, are absent.

In entering on this description, I shall first deal with signs and symptoms presented by a simple cicatricial or carcinomatous stricture of the intestine leading to complete occlusion of the bowel.

The contents of the bowel above the stricture stagnate. The passage of the accumulated material, the gaseous, solid, and liquid constituents, is inhibited; in other words, there is absolute and complete constipation. At first flatus or fecal material that may have been present in portions of the bowel situated below the occlusion may be passed, either spontaneously or after the administration of enemata. Soon, however, nothing more can be evacuated in this way; occasionally small and isolated particles of fecal matter may be washed out by repeated irrigation of the colon and may raise false hopes in an inexperienced observer. As a matter of fact, these small particles are merely remnants of feces and are washed out of the haustra coli.

In the mean time attacks of colic begin. These are always more or less severe and always very painful. We have already described these paroxysms of colic at some length in the section on Narrowing of the Intestinal Lumen, and have called attention to the fact that they are chiefly and particularly characterized by the appearance of visible energetic peristaltic movements of the intestine, and occasionally by marked stiffening of certain loops of the intestine. Attention has also been directed to the fact that these abnormal movements of the bowel can usually be seen through the abdominal walls in these cases. The appearance of energetic peristalsis and stiffening of the bowel characterizes that form of occlusion supervening on chronic stenosis of the bowel, for in these cases in particular there is an advanced degree of hypertrophy of the muscular coat of the intestine above the stenosis, whereas in acute occlusion of the bowel occurring in an intestine that was normal up to the time of the accident no such hypertrophy of the muscular coat is present; consequently visible peristalsis and stiffening of certain loops of intestine are never seen in the latter condition (compare below the discussion on Acute Occlusion of the Bowel). In the great majority of cases in which the muscular activity of the intestine is found to be excessive and in which, at the same time, symptoms of occlusion of the bowel become manifest, we can be reasonably certain that the primary cause both of the occlusion and of the increased muscular activity is some structural change which has developed slowly; in other words, that we are dealing with a case of chronic narrowing of the intestinal

lumen that has gradually led to complete occlusion of the bowel. It is particularly important to remember this sign in making a diagnosis of those cases in which the stricture remains latent for a long time and in which the occlusion of the intestine apparently occurs while the bowel is perfectly normal and its lumen patent. In rare instances increased peristaltic movements and stiffening and rigidity of certain loops of the intestine may be absent even when occlusion follows a gradually progressive stenosis. This may occur under two conditions, namely, in the first place, when the powers of the muscular coat of the intestinal wall have become insufficient from overexertion or overdistention of the bowel-wall, or, in the second place, when accurate inspection of the abdomen is impossible, owing to the presence of an advanced degree of meteorism.

As soon as occlusion of the bowel occurs, the appetite is lost; in many instances it is lost before complete occlusion of the bowel takes place. Soon after the lumen of the bowel becomes obstructed the patients begin to belch and complain of nausea. The gas belched gradually becomes very offensive, and in the later stages of the disease, when occlusion has persisted for some time, acquires a somewhat feculent odor. The attacks of nausea persist until vomiting finally occurs. At first the contents of the stomach are vomited, mixed possibly with a little bile; finally, fecal vomiting occurs. The latter symptom, namely, fecal vomiting, has always attracted a great deal of attention; for over two centuries medical men have discussed and argued about the significance of this symptom, and so much has been written on this subject that it is impossible to avoid discussing the matter. I will limit myself, however, to a few general remarks on fecal vomiting. While discussing this symptom in this place—*i. e.*, under the heading of intestinal obstruction following stenotic processes—it will be as well, in order to avoid unnecessary repetition, to include in these remarks the feculent vomiting of acute intestinal obstruction in cases where the bowel was previously healthy.

Attention need hardly be directed to the number of highly disagreeable and disgusting features of fecal vomiting. The vomited material may be so profuse that the malodorous masses pour out of the mouth and even out of the nose of the patient in a veritable stream. The details of this description will not be further elaborated.

The feculent character of the vomit can easily be determined by its odor. So far as I know, no chemic investigations of the vomited material have as yet been undertaken as regards the presence of so-called fecal constituents, such as skatol, etc. The color of the material varies greatly: it may be a dirty yellow, brownish-gray, or yellowish-green.

The vomit is almost without exception liquid, and resembles in all respects a watery diarrheic stool. Personally, at least, I have never seen fecal vomit of a different appearance, and to my knowledge there has been no description of the vomiting of fecal masses in a positive case of organic intestinal obstruction.

Formerly, it was believed that fecal vomiting could occur only under one condition, namely, when the ileocecal valve became insufficient; this was considered an indispensable condition, for it was believed that fecal vomiting could not occur unless feces were driven backward from the colon into the small intestine. This view has been refuted long ago, and the fact has been definitely established that feculent vomiting can occur just as well when the obstruction of the intestine is in some other part of the bowel than the colon. Cases are on record in which feculent vomiting occurred in occlusion of the lower ileum and even in the lower and the very highest portions of the jejunum. The clinical proof of these facts is so abundant, and so much fresh clinical evidence is adduced nearly every day, that it would be superfluous to quote references bearing on this question. Morgagni in his day interpreted this phenomenon correctly and recognized the necessary underlying factors which must exist in fecal vomiting.

The so-called feculent character of the intestinal contents is due to the presence of certain decomposition-products of the proteid material contained in the bowel. This class of substances undergoes putrefactive decomposition, owing to the action of definite species of bacteria (see pp. 35–60). In order that this putrefactive decomposition of the proteid material by the action of bacteria may occur, the action of the latter must be prolonged. In the large intestine these conditions are all normally fulfiled—that is, the bacteria are present and the material remains in the bowel for a certain length of time; in the small intestine, however, the conditions are not favorable to the development of feculent decomposition of proteids, chiefly because the contents of the small intestine are propelled onward so rapidly that there is not sufficient time for the disassimilative action of bacteria. If, however, abnormal conditions arise which favor a prolonged stagnation of the bowel-contents in the small intestine or which prevent the onward passage of the bowel-contents and necessitate its stagnation in the small intestine, the contents of this portion of the bowel may readily assume a feculent character. Conditions of this kind are particularly likely to occur in obstruction of the small intestine.

The conditions just outlined also explain another phenomenon which will be discussed in detail later on, namely, that in certain forms of occlusion of the bowel fecal vomiting is relatively rare, even though the passage of the bowel-contents through the intestine is completely stopped. Fecal vomiting is usually absent in very acute cases of internal strangulation of the bowel and in volvulus of the intestine. In these forms of occlusion of the bowel two factors prevent the conversion of the bowel-contents into feculent material, or at least render this conversion difficult, namely, in the first place, the occurrence of violent vomiting that prevents prolonged stagnation of bowel-contents, and, in the second place, the early fatal issue that usually supervenes in these cases before the formation of feculent bodies can occur.

It would be of great clinical import to know what time must elapse between the establishment of occlusion and the earliest appearance of

stercoraceous vomit. For obvious reasons, this is more readily ascertained from occlusions of sudden onset. The fact is that in acute internal incarceration stercoraceous vomit may appear as early as the second day, though this is not the rule. It has been maintained that it can occur within a few hours, but this must be most exceptional.

A very animated controversy has for some time taken place with regard to the following question : What are the factors which drive the contents of the bowel in an opposite direction to that in health and ultimately lead to their evacuation by the mouth? Two attempts to explain this phenomenon will be made here : a dynamic and a mechanical. Galen originally gave the apparently simple explanation of antiperistaltic action of the bowels (motus antiperistalticus). Subsequently the causation of fecal vomiting was explained by mechano-hydraulic laws. Leichtenstern credits Haguenot, of Montpellier (1713), with having been the first to formulate this explanation of this phenomenon, which until quite recently has been credited to van Swieten, and has been coupled in the literature by nearly all writers with the name of the latter investigator. The genesis of fecal vomiting as interpreted by Haguenot has been fitly called by Henle " running over " of the stagnating contents of the uppermost loops of the small intestine.

As soon as occlusion of the bowel occurs, those loops of intestine that are situated above the stricture begin to fill slowly or rapidly with gas, and chiefly with liquid material. The latter consists in the first place of the stomach-contents, which are being propelled downward, and, in the second place, of the copious secretions of the glands of the intestinal wall above the affected area. The amount of material that can accumulate immediately above the occlusion is limited, for the wall of the intestine can be stretched only to a certain degree, and as soon as the maximum degree of tension is reached, no more material can be forced into a dilated loop. Gradually, therefore, the stagnating mass fills longer portions of the intestine above the stricture, and consequently moves upward toward the stomach. While such large portions of the intestine are filled and distended with accumulated material, vomiting may occur for some reason or the other. As a result, the diaphragm and the abdominal muscles contract, the volume of the intra-abdominal space is reduced, and a certain amount of pressure is exercised on the intestine and transmitted to the contents of the bowel. The volume of the liquid material contained in the loops of intestine situated above the obstacle cannot be reduced by these forces, nor can the pressure be relieved in a downward direction toward the anus, owing to the presence of an insurmountable obstacle in the bowels ; consequently the accumulated material necessarily passes upward toward the stomach ; in this way fecal vomiting is produced. Even if vomiting does not occur in the first instance and is not the chief cause of the increased abdominal pressure and of the consequent pressure upon the material accumulated in the bowels, any contraction of the wall of the intestine itself, even if the peristaltic movements proceed in a normal direction,

must, in an analogous manner, exercise compression on the bowel contents and mechanically force it backward toward the stomach. Brinton in particular called attention to the latter possibility.

The question to be answered is, How can it occur that the lowest portions of the accumulated bowel contents—that is, those immediately above the obstruction, in which feculent metamorphosis is probably most advanced—become mixed with the other portions of the accumulated material situated further up and nearer to the stomach? how can this mixture take place unless antiperistaltic movements of the affected loop of intestine occur? Experiments on animals teach us that they are not unessential in the mixing of the contents. Irrespective of diffusion (for the gaseous and free substances) and the mechanical effect of the strangulating and retching preceding the stercoraceous vomiting, the mixture of the materials can occur by the kind of action described by me as "recoil contraction" (Rückstosscontraction), which occurs as follows:

As soon as the part of bowel situated immediately above the occluded area becomes filled and distended to a certain degree by the mass of material that enters this cul-de-sac from above, it contracts. As a result of this contraction, the material contained in the loop near the occlusion is forced backward for a certain distance so that, in cases of acute occlusion, for instance, the bowel may become completely empty immediately above the occluded spot. This process is repeated a number of times, and on each occasion the intestine immediately above the occlusion will be seen to be empty. Gradually, however, as these contractions are repeated, the latter portion of the bowel slowly becomes filled and distended with bowel contents and is not emptied completely during each contraction. Then the same play of violent peristalsis and recurrent (Rückstoss) contractions is repeated immediately above this filled and distended portion, and gradually these waves move upward and backward while, at the same time, the intestine is slowly filled and distended from below upward. In a sense this peculiar sequence of movements resembles antiperistaltic movements; in reality, however, it is not regular antiperistalsis, and I have never seen true antiperistaltic movements in this condition.

Another argument in favor of the view that the act of vomiting and not antiperistaltic movements of the bowel determines the propulsion of the bowel contents backward and produces fecal vomiting is the fact that in some instances large portions of the bowel may be completely filled with thin feculent matter and still no feculent vomiting occur. I observed a case of enterostenosis, for instance, in which the occlusion was situated in the sigmoid flexure and in which the whole large intestine and the small intestine as far as the jejunum were filled with thin feculent material, but in which no fecal vomiting occurred. As a matter of fact, this patient never vomited during the whole course of the disease—did not even retch.

Even though the mechanohydraulic theory suffices to explain the stercoraceous vomit, it is still a question whether there is not an actual

antiperistalsis, a reversal of catastalsis into anastalsis, involved with the physical factors.

I think I have proved, by conclusive experiments on animals, that true antiperistalsis may be excited even in a healthy intestine. I have also demonstrated on man that a concentrated (colored) normal salt solution of 250 c.c. injected into the rectum will reach a point 40 cm. above the ileocecal valve. Kirstein, Mühsam, Enderlen, and Hess have reversed large stretches, or almost the entire length, of the intestine in dogs (430 out of 485 cm.). They sewed the upper jejunum to the cecal end and the lower ileum to the duodenal end of the intestine and found that the reversed intestine performs normal peristalsis, transporting the digested food into the colon. The animals live for weeks, even to three months. There is, therefore, no doubt that the intestine can act antiperistaltically, and not only as in Grützner's experiments, with upward flowing marginal streams (Randströme). I have proved that a special form of irritation is requisite to cause anastaltic movements—*e. g.*, strongly irritant chemicals—and that these, furthermore, must act upon " unphysiologic " points.

Now the question arises, are these last requirements present in intestinal obstruction. Leichtenstern deems this not improbable, for it seems to him not unlikely that among the many putrefaction products arising there may be some that act similarly to the concentrated normal salt solution and other chemic irritants used in my experiments. (Bokai observed energetic contractions in the intestine of a rabbit after injection of only 0.002 skatol.) Kirstein considers from his experiments that occlusion of the lumen due to damage of the intestinal wall disturbs the catastaltic movement of the intestine, or at least of its upper part. It is also possible that antiperistalsis is active in the genesis of stercoraceous vomit, but it is not essential, and, above all, its actual coöperation has not been proved.

The stercoraceous vomit, in which actual masses of formed feces are rejected *per os*, must be completely differentiated from the feculent vomit in mechano-anatomic obstruction. The former type assumes a very important place in the pathogenesis, and the discussions concerning feculent vomit in obstruction would, in my opinion, be greatly simplified were the foregoing distinction always remembered.

Schloffer and also Langmann have collected the cases of vomiting of formed feces that are to be found scattered throughout our literature. In one there is manifest fraud; other cases are doubtful. Still there are few cases in which even the most skeptical must believe in the actual vomiting of formed feces, sometimes before the physician's eye, as in the cases of Rosenheim, Desnos, and Schloffer. All these occurred in individuals decidedly hysteric or nervous, whose general syndromes were very similar to that in hysteria. Laparotomy performed on several revealed no anatomic impediment, but Schloffer and Slajmer, found portions of the intestine in tetanic contraction. Leube, in a similar case, found by palpation the entire large intestine contracted into a mass of stony hardness. Particulars of these cases (for which see

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originals) force us, in my opinion, to the conclusion that the vomiting of fecal masses and of actual scybala can be caused only by a decided, at times even exceedingly active, antiperistaltic movement of the intestine. The cause of this total reversal of the normal movement of the intestine is entirely unknown; but the same uncertainty hovers over many other phenomena in the wonderful and enigmatic clinical picture of hysteria, which, in like manner, have eluded all our attempts at a physiologic explanation, yet nevertheless indubitably exist.

The consideration and description of other symptoms of intestinal obstruction will now be resumed. It might be expected that in complete obliteration of the intestinal lumen a considerable degree of meteorism would necessarily appear. While it is true that a certain degree of meteorism is often recognized in the form of occlusion that we are discussing,—namely, occlusion following chronic stenosis of the bowel,—the condition is never so severe nor so violent as in some other forms of acute occlusion and in diffuse acute peritonitis. The reason for this is carefully considered in the section on Meteorism. The greater part of our knowledge of this subject is based on the experiments of Zuntz-Tacke, which are described in the above-mentioned section. Whenever the intestinal lumen becomes occluded by some simple means or some uncomplicated process that does not cause circulatory disturbances in the walls of the intestine and mesentery, the passage of gas downward is, of course, stopped and no flatus is developed; at the same time the other process that removes accumulated gas from the intestine is not interfered with—namely, the absorption of gas from the lumen of the bowel by the blood circulating through the walls of the intestine. The latter process is more important in the removal of superfluous gas from the intestine than the former, and as long as it remains intact, the accumulation of excessive quantities of gas in the intestine is to a great degree prevented. As soon as the blood circulating through the intestinal wall becomes relatively or absolutely insufficient to absorb the gas that is formed in the intestine, the latter must accumulate; in other words, as soon as more gas is developed than can be absorbed, distention of the bowel by gases must occur. In occlusion of the bowel in which the circulation of the intestinal wall remains intact, gas may, therefore, still accumulate and lead to meteorism, if abnormally large quantities of fermentable material are present in the bowel contents. If no such material is present in excess and the development of gas in the intestine is not abnormal, the degree of meteorism that develops in simple occlusion of the bowel (without strangulation) remains very slight. Those portions of the intestine that are in the immediate neighborhood of the occluded spot are usually most severely distended. The distention of these portions of the intestine is so peculiar and so characteristic that important conclusions in regard to the localization of the occlusion may frequently be drawn from the position of dilated loops of intestine.

The last direct symptom of occlusion of the bowel to be mentioned is the increase in the excretion of indican and of other conjugate sul-

phates in the urine. (For all the details of this important phenomenon the reader is referred to the discussion in the ninth section, and also to the paragraphs on the localization of occlusions of the intestine.)

All the symptoms enumerated remain the same even if occlusion occurs in an intestine that was perfectly normal and patent up to the time of the closure. They also develop, therefore, in the rare cases of obstruction of the intestinal lumen by foreign bodies or by gall-stones, or in acute compression of the bowel by some displaced abdominal organ. This statement must be slightly modified, however, to exclude all those cases of acute obstruction of a previously normal intestine in which, in addition to an interruption of the intestinal passage, interruptions and perversions of the circulation in the intestinal wall and the mesentery occur, or those cases of acute occlusion that are accompanied by very painful irritation of the sensory nerves of the intestine leading to reflex shock (compare below).

The general health remains remarkably good in this form of occlusion of the intestine, and the victims for a comparatively long time feel fairly well considering the seriousness of the event. This, of course, does not apply to those cases in which occlusion of the bowel constitutes the termination of some severe disease, for example, when carcinoma is the primary cause of the condition. The facial expression of the patients, it is true, may indicate suffering, chiefly on account of the extremely painful character of the attacks of colic; the patient may also be very much depressed, but there is rarely pronounced general weakness, nor is the action of the heart seriously impaired; in fact, there may be stercoraceous vomiting with a perfectly strong and good pulse.

The signs and symptoms presented by cases of occlusion combined with strangulation of the bowel are very much more severe and painful. The course of the disease in these cases is very acute, violent, and at the same time exceedingly rapid. Such conditions are seen, for instance, in acute hernia, volvulus of the intestine, and in part also in invagination of the bowel.

In the account of each one of these different organic forms of acute occlusion of ths bowel a clinical description will, of course, be given of the manifestations presented by the disease, and stress will also be laid on the peculiarities of the syndrome produced by each one of these different forms of occlusion. A brief sketch will only be given here of the general aspect of the disease, with a description of the characteristic general traits common to them all.

The patients develop violent pain in the abdomen that usually appears quite suddenly; in some instances this pain is strictly localized to some one portion of the abdomen; in other cases it is more diffusely distributed throughout the whole belly. It may be continuous and persistently severe, it may be remittent in character, or it may appear in paroxysms of colic. Soon after the occurrence of the pain, vomiting, nausea, and hiccup develop. The vomited material at first consists of the contents of the stomach; later it becomes bile-tinged; still later it

may acquire a most disagreeable odor and finally become distinctly feculent in character. In the early stage, that is, soon after the onset of the pain, an evacuation of the bowel usually occurs; the passage of stools through the lower portion of the rectum is frequently accompanied by severe tenesmus. All the fecal matter passed is derived from the lower portion of the intestine. After this first motion has been passed, the evacuation of intestinal contents as a rule stops completely, and no more feces or flatus are passed. The abdomen now becomes meteoristic and distended, the character of the meteorism depending largely on the anatomic character of the obstruction and its situation in the intestine. In some of these cases meteorism of the abdomen reaches the greatest possible degree of development. In addition to these symptoms of occlusion, which resemble essentially those symptoms of the lesion described in preceding paragraphs, although, of course, they are pathogenetically in part different, symptoms of serious collapse develop, the general health becomes rapidly impaired, and the patients feel utterly miserable. The face acquires a frightened expression and is distorted with pain. The patients are collapsed, the extremities are cold and bluish, the pulse is rapid, small, and easily compressible. Gradually the gastro-intestinal symptoms increase in severity, hiccup becomes more violent, nausea becomes very distressing, and feculent vomiting continues. In addition, the patients suffer from unquenchable thirst, the abdomen becomes more and more distended, consequently respiration becomes rapid and shallow, and a most distressing feeling of oppression tortures the sufferer. The secretion of urine is arrested, so that occasionally complete anuria develops. The patients gradually get weaker and weaker, and finally present a horrible picture, resembling the appearance of a patient in a severe attack of cholera. The condition is made more distressing by the fact that the sensorium is perfectly unaffected throughout. In the last stages the skin is sallow and flaccid, without turgescence, quite cool, livid, and frequently covered with cold sweat. The eyes of the patient are sunken and surrounded by dark rings. The face looks peaked, the skin of the face appearing like the skin of the body. The nose usually looks sharp and pointed; the resonance of the voice is lost; the pulse is weak and thread-like, and the patients suffer from the most severe degrees of general prostration.

In this disease-picture a distinction can at once be readily made between local gastro-intestinal symptoms on the one side and remote symptoms due to disturbances of the nervous system and the circulatory apparatus on the other. Let us briefly analyze these two groups of symptoms.

It might be imagined that in these instances, as in the other form of occlusion due to chronic progressive stenosis of the intestine, the gastro-intestinal symptoms might be the simple and direct mechanical results of the occlusion of the intestinal lumen; two of the gastro-intestinal symptoms described are, in fact, entirely due to this factor, namely, the complete stoppage of the passage of stools and flatus *per anum* on the one hand, and the vomiting of fecal material on the other. I will

not, therefore, enter into a discussion of these two symptoms again, but merely refer the reader to what has been said about them in previous paragraphs. All the other symptoms, however, as simple vomiting, pain, and meteorism, are only in part direct results of the mechanical obstruction of the bowel. To some extent the latter symptoms are due to another cause which will be described presently.

In the account of the various organic forms of intestinal obstruction, such as volvulus, incarceration, and invagination of the bowel, it will be shown in detail that the pain produced by all these lesions may be due to two causes. As the pain is one of the most constant and prominent symptoms of acute occlusion of the bowel, it may be worth while to enter into a more detailed account of it here. In part the pain is directly due to local changes in the intestine ; in part it must be considered to be due to radiation and transmission of nervous stimuli, originating locally in the intestine, to other and remote areas. The strictly localized pain is local in origin, and is produced by the local change in the intestine. The colicky pain, on the other hand, which appears in paroxysms and increases and decreases in severity, must be considered to be due to the energetic peristaltic movements, tetanic stiffening, and rigidity of the bowel that occur in acute occlusion. Finally, the pain that radiates into remote portions of the body is, I consider, due to irritation of the large ganglionic plexus.

The degree of pain that the patients suffer in the early stages of the disease depends largely on the extent of the intestine that is involved in the process ; the larger the portion involved, the greater presumably the pain. Very little, relatively speaking, depends on the individual irritability of the patient. This follows from our knowledge of the fact that the severity of the pain is so extreme, especially in the acute forms of internal hernia, that it necessarily produces the greatest suffering even in subjects who are naturally insensitive or who may possess the greatest will-power.

We know from experience that the initial pain of acute occlusion of the small intestine is usually more violent than in acute occlusion of the large intestine ; in hernia, which almost without exception occurs in the ileum, the initial pain is more severe than in volvulus, which is found most frequently in the sigmoid flexure. Bellon is inclined to attribute these peculiar differences in the severity of the pain in different portions of the intestine to the nervous connections existing between the different plexuses and the nerve-supply of these two portions of the bowel. He calls attention to the fact that the nervous apparatus of the small intestine is in connection with the superior mesenteric plexus and the solar plexus, whereas the nerve-supply of the large intestine is in connection with the inferior mesenteric plexus and the plexus lumbo-aorticus. In the former instance, therefore, the nerves of the intestine are connected with nervous apparatus that contain very many fibers and have more intimate and numerous centripetal connections, whereas in the latter the two plexuses do not consist of so many nerves and have few or no centripetal fibers ; consequently the symptoms

transmitted through the nervous system are much more severe in obstruction of the small intestine than in obstruction of the large intestine. This important matter, however, will be referred to later when a number of other allied symptoms will be explained.

Treves has called particular attention to the fact that in complete occlusion of the intestine the pain is constant. It is undoubtedly true, however, that the pain in acute occlusion occasionally remits and occasionally becomes exacerbated, but the remissions and exacerbations are due to the more or less energetic contractions of the bowel above the obstruction. The pain never remits completely, as it does in incomplete occlusion or stricture of the bowel, and it is this latter point that Treves emphasizes. Even in cases of acute occlusion of the bowel the patients may be free from pain at intervals, but this can occur only when the occlusion is not absolutely complete. This phenomenon, therefore, occasionally occurs early in the course of invagination of the bowel. Pain may also remit in those cases of occlusion of the bowel in which, from the very nature of the lesion, the intestinal passage may temporarily become pervious—for instance, those rare cases of occlusion of the bowel due to rotation of the sigmoid flexure half way around its mesentery.

The initial pain is, as a rule, increased by external pressure. Sensitiveness to pain on pressure usually indicates the existence of some peritonitis. In all forms of intestinal obstruction the pain, after having persisted throughout the whole course of the disease, may occasionally stop toward the end. In rare instances the pain may cease a few days before the fatal issue and sometimes quite suddenly. When this occurs, it may be concluded either that paralysis of the intestine has occurred, so that all peristaltic movements of the bowel-wall are abolished, or that perforation of the intestine has taken place, for in the latter accident, as is well known, the pain passes off. When paralysis of the intestine has occurred, meteorism becomes more pronounced and the patients become more seriously collapsed. If perforation of the bowel occurs, the well-known sequelæ of this accident will be noted.

The pathogenetic significance of vomiting in acute occlusion of the bowel may also be different. Vomiting in the course of the disease is explained on the same mechanical grounds as pronounced fecal vomiting. This applies to all forms of vomiting that occur at this stage, even when the vomited material is not stercoraceous. The only theory, then, which explains vomiting in this condition is that advanced by Haguenot—namely, the mechanical one. The other one is that vomiting is a secondary symptom and occurs consecutively to peritonitis. The vomiting, on the other hand, which occurs early in the course of the disease—the initial vomiting—is directly due to the shock of the acute obstruction and the irritation of the nervous system which results from the accident itself. The same applies to the attacks of hiccup that frequently supervene, both initial vomiting and hiccuping being reflex symptoms. It is well known that the nerves of the peritoneum and the intestine are violently irritated when invagination, hernia, or

occlusion in general of the bowel takes place, and irritation of these nerves, as we know, can readily produce the reflex phenomena indicated. As a matter of fact, the vomiting that occurs later in the disease is, to a great extent, reflex in character.

If this view of the reflex character of the initial vomiting in intestinal obstruction be accepted, it can readily be understood why in acute forms of occlusion of the small intestine this symptom is always present. The nervous apparatus of the small intestine is connected in so many ways with other nerve centers that any severe irritation of its nerves must necessarily lead to vomiting. As a matter of fact, this symptom is always very pronounced in this condition and usually appears at once; in fact, it constitutes one of the most characteristic features of the onset of acute occlusion of the small intestine. Another condition in which it is invariably present is acute internal strangulation, which occurs only in the small intestine; this fact again explains why vomiting is an absolutely essential symptom of this condition.

Meteorism develops chiefly in the strangulated mass of intestinal loops. In previous paragraphs all the essential features of the pathogenesis of this symptom have been considered, both in the description of the pathologic anatomy of occlusion of the bowel (p. 350) and, in addition, in the paragraphs immediately preceding this one (p. 367). The reader should refer to what has been said in these two places for all the details. Occasionally meteorism develops with startling rapidity, so that the distention of the abdomen becomes enormous. This rapid form of meteorism is particularly frequent in volvulus and in strangulation of the intestine by a band. Meteorism may originate in many different ways, and the rapidity with which the symptom develops may also vary in different conditions. In the paragraphs on the different organic forms of occlusion of the bowel the specific differences in the character of meteorism and the rapidity with which it develops in these different varieties of the lesion have been dealt with; for the details the reader should refer to these paragraphs. Here, however, special attention should be directed to a few general points of view that are also significant and interesting in the treatment of this disease.

One of the symptoms which is almost universally present is a great increase in the intra-abdominal pressure. This increase in pressure is produced, in the first place, by the great degree of distention of the intestine; in the second place, by the reflex rigidity and tension of the diaphragm and the abdominal muscles. The increase in intra-abdominal pressure produces a certain number of local symptoms by exercising a definite effect on the contents of the abdominal cavity, and, in addition, has a marked general effect. The distended loops of intestine are pressed against other loops of intestine that are collapsed. In addition—and this is more important and significant—they are pressed tightly against the obstructed portion of the intestine. This constitutes a serious factor, inasmuch as it interferes with the possibility of a cure. It becomes impossible, for instance, for twisted loops of intestine to return to their normal position, and intestinal coils which are kinked

must remain so. Some coils which are pressed into fissures and holes or are constricted by pseudoligaments or Meckel's diverticulum cannot be released from their position, and in consequence the stricture of the bowel produced by the abnormal position of the loop and by the constricting effect of the surrounding tissues remains unrelieved.

Another common symptom in occlusion of the bowel, complicated by rapidly developing and severe degrees of meteorism, is cardiac weakness, which is also more or less dependent on the increase of intra-abdominal pressure. Jürgensen was able to demonstrate the following in regard to this matter by experiments : He raised the intra-abdominal pressure artificially by forcing air into the abdominal cavity, and found that the arterial pressure fell greatly. The fall in the arterial pressure, as a matter of fact, was so great that the blood did not squirt out of the femoral artery when this vessel was opened, but merely oozed out in drops. The arterial blood at the same time was very dark—almost black. This experiment seems to show that meteorism itself may directly bring about a fatal issue and cause the death of the patient by paralyzing the heart.

Another very important point which should be borne in mind in intestinal obstruction is that active and distinctly visible peristaltic movements and tetanic stiffening of the bowel are only exceptionally seen in cases of acute occlusion where the intestine was previously perfectly healthy and unobstructed. It is important to remember, moreover, that these peristaltic movements and the attacks of tetanic rigidity of the intestine, if they do become visible, are never so marked or severe as in cases of complete intestinal obstruction supervening on a chronic stricture of the bowel. There is, therefore, a fundamental difference in this respect between cases of occlusion occurring acutely in a normal intestine and cases of occlusion in the course of chronic stenosis of the bowel. In the latter case we see most active tossing and pitching and most energetic rolling of hard and resistant coils of intestine, whereas in the former we see only that the abdomen is distended, very tense and resistant, and rarely shows visible contractions of the intestine. The degree of abdominal distention, it is true, in uncomplicated intestinal obstruction occurring in a patient whose intestines were previously normal is frequently very considerable. These differences are due to the following causes : In cases of combined occlusion and strangulation of intestinal loops that are complicated with local meteorism complete paralysis of the intestine usually develops ; as soon as this occurs, all movements of the bowels naturally cease. But this may occur also in simple meteorism resulting from the stagnation and accumulation of bowel contents above the uppermost point of occlusion. Here no interference with the circulation in the intestine or the mesentery in this area is brought about. Kocher and I, however, have demonstrated by experimental investigations that the paralysis of the bowel musculature in this instance is due to overdistention of the bowel-wall. We find that the muscular coat of the intestinal wall under these conditions no longer contracts when stimulated, even with the strongest faradic cur-

rent. It can readily be understood, therefore, why under these conditions all peristaltic movements of the bowel are quite impossible, and *a fortiori*, peristaltic movements sufficiently vigorous to be visible through the abdominal wall. Another factor which may be regarded as responsible for the slight degree of peristalsis in acute occlusion in a previously normal intestine as against the energetic peristalsis seen when occlusion supervenes in the course of chronic enterostenosis is the complete absence of muscular hypertrophy above the obstruction in the former condition. This very hypertrophy of the muscular coat, which always develops, as has been shown above, in cases of chronic stricture of the intestine, determines the active character of the peristalsis in this disease, and is the chief cause of this phenomenon.

These differences, let me repeat again, must be remembered. The following rules, therefore, may be formulated for the differential diagnosis between acute and chronic narrowing of the bowel. In chronic stenosis, or when the obstruction is merely a narrowing or an occlusion of the bowel lumen without strangulation, violent peristaltic movements always occur and occasionally tetanic stiffening of intestinal coils. In the acute form with strangulation there is distention of some fixed loop of intestine, without, however, any visible movements.

Slight movements are undoubtedly occasionally performed by a strangulated coil of intestine. The contractions of a strangulated loop, however, are never so active or energetic as in chronic forms of stenosis with or without occlusion; this refers more especially to the wave-like twisting movements that are so characteristic of the latter form. It is also undoubtedly true that occasionally the afferent intestinal coils—viz., those situated above the uppermost point of occlusion in bileptic closure of the bowel—show increased peristaltic movements, but, in the first place, these movements are very slight (owing to the absence of hypertrophy of the muscular coat), and, in the second place, they can hardly ever be directly seen, because the strangulated loop is always so distended that it covers these afferent loops and conceals them from view.

I need hardly describe in detail why meteorism, when it forces the diaphragm upward, complicates the disease, and why this symptom greatly impairs the general well-being of the patient and renders the whole aspect of the disease more distressing and more dangerous. In previous paragraphs (p. 367) I have already said all that is necessary with regard to the results of the physical examination in cases of meteorism in general and of meteorism occurring in intestinal obstruction in particular.

In this connection, however, it is necessary to consider briefly the attacks of diarrhea which are met with now and then in acute intestinal obstruction. Occasionally diarrhea and vomiting occur together, and the clinical aspect thus produced has been called " *choléra herniaire* " by Malgaigne. This peculiar syndrone, it is true, is not often seen. In a few instances it has led to very serious errors in diagnosis, especially early in the disease, and a few cases are on record in which the diagnosis of true Asiatic cholera was made. Attacks of profuse diar-

rhea are occasionally seen in all the various organic forms of internal occlusion of the bowel (invagination, incarceration). The exact pathogenesis of this symptom has not so far been completely and clearly made out. According to some authors, there is an extreme profuse secretion from the intestinal mucous membrane due to paralysis of the mesenteric nerves. , This view has been called the transudation hypothesis. The whole subject is still indefinite and requires careful study. More information is particularly wanted as to the special factors and conditions producing diarrhea in any given case.

In addition to these local gastro-intestinal symptoms, which may either be the direct or the indirect result of strangulation-occlusion, another series of phenomena is frequently met with in cases of acute intestinal obstruction ; while the gastro-intestinal symptoms are usually predominant and constitute the characteristic clinical features of the disease, these additional symptoms, which will be dealt with, though more general in character, are also of great importance. These phenomena, taken as a whole, consist in the serious depression of the circulatory and the nervous systems, known in ordinary clinical language as "collapse" or "shock." As a matter of fact, the clinical aspect of some of these cases exactly resembles that of most serious collapse from a variety of primary causes. Combined with symptoms of collapse there are, of course, always a number of phenomena which are the direct mechanical result of the occlusion of the bowel. In cases of moderate degree, and particularly in cases that do not run so fulminating a course, the general health of the patient may not be seriously impaired—in fact, the subjective feelings of the patient and his general appearance may in no way correspond to the severity of the local affection. In the great majority of cases, however, the medical attendant is confronted with a horrible picture of serious collapse in a more or less developed form.

A review of the literature on this subject shows that the older publications on intestinal obstruction, namely, those prior to the seventh decade of the last century, do not discuss the causes determining the symptoms of collapse in these cases. Reference to the writings of Graves, Bamberger, and Trousseau shows that even the most renowned authors were absolutely silent on this important question. One of the first investigators who definitely formulated this question, and at the same time was the first to furnish a clear and positive answer to it, appears to have been Le Fort. This author, in describing a case of strangulated hernia, expresses himself as follows :

It seems to me that the bad results of strangulated hernia are more physiologic than mechanical, and are due not so much to incarceration and constriction of the intestine as to the nervous effects thus produced, and that they are to some extent independent of fecal accumulation and retention.

At the present time two distinct views are advanced with regard to the causes determining the general symptoms developed in acute intestinal obstruction. One may be called the "nervous reflex theory," the

other the "intoxication" theory. Personally I am inclined to the belief that the former is the one by which by far the most of the manifestations are most freely indicated, this includes at least the symptoms clinically most frequent and important accompanying the onset, and with which practically all the facts in the syndrome of acute occlusion most nearly coincide. The latter, on the contrary, comes into consideration only in certain cases, and then not until a late period in the course of the affection.

The reflex theory is naturally of comparatively recent origin, for it could not be formulated and applied to the clinical conditions observed in occlusion of the bowel until modern physiology had taught us what now we know in regard to the innervation of the heart and the blood-vessels. Our knowledge of this complicated subject is chiefly due to the classic investigations of Goltz, von Bezold, C. Ludwig, and his pupils. Assuming that the reader is familiar with all these original investigations and the researches that followed them, a very brief account of the subject will be given here. The serious character of the clinical picture in occlusion of the bowel is due to the violent stimulation of the sensory nerves of the intestine and the peritoneum in all cases of strangulation, of volvulus, of invagination, and of obstruction of the intestine by gall-stones. The more violent and the more sudden this irritation, as shown by the severity of the initial pain and the violence of the paroxysms of vomiting, the more severe and the more wide-spread the effect on those nerve-tracts reflexly stimulated from the primary seat of the trouble.

In intestinal strangulation the intestinal and peritoneal branches of the pneumogastric and the sympathetic nerves are first stimulated. Irritation of these fibers leads to alterations of the heart's action, and, by causing reflex paralysis of the cardiac branches of the pneumogastric and of the splanchnic nerves, further produces alterations in the general distribution of the blood throughout the general circulation. If these changes are carefully and systematically studied, it will be found that in the area of the pneumogastric the first sign of occlusion of the bowel is a primary but transient stimulation which usually leads to a considerable slowing of the pulse immediately after the onset of occlusion. It is quite probable that this initial bradycardia would be observed more frequently if physicians had an opportunity of studying cases of occlusion of the bowel more frequently at the very beginning—that is, when the first attack of pain occurs. Whether or not this primary vagus stimulation occurs, there is subsequently, in almost all cases, reflex paralysis of the cardiac branches of the pneumogastric, producing increased frequency of the pulse. At the same time reflex paralysis of the splanchnic nerves occurs, and as they are the vasomotor nerves of the intestine, this paralysis leads to the overdistention of the abdominal organs with blood. The direct result of this overdistention is a disturbance in the general distribution of the blood throughout the different parts of the body, for the abdominal organs are capable of retaining such enormous quantities of blood that much of the blood that is

destined to supply other portions of the body is retained here. As a result, the peripheral portions of the arterial tree become relatively empty, the skin becomes pale and cold, the pulse small, of low tension, empty, and may even become extremely thready. In addition, there is usually anemia of the brain from a depression of the tone of the vagus; this cerebral anemia also leads to increased frequency of the pulse-beat. The temperature of the skin is lowered; in the latest stage of the disease, however, the surface temperature may rise, particularly if peritonitis supervenes, or possibly from intestinal autointoxication. The temperature in the rectum is also reduced in these cases, and Leichtenstern utilizes this symptom in combination with the general cooling of the surface of the body to advance the view that in cases of occlusion of the bowel the general heat-production is considerably reduced. He argues that this phenomenon is the direct result of shock following occlusion, and is caused by reflex disturbances of the nervous apparatus governing heat-production.

The anemic condition of the skin is also in part responsible for the extreme pallor and facial aspect of grave disease. This hippocratic face is, however, due to another factor present in this disease—namely, the great loss of water from the tissues, which is directly due to the profuse vomiting and the attacks of profuse perspiration that occasionally supervene in occlusion of the bowel. The loss of water from the tissues in addition causes wasting of the skin, dryness of the tongue, torturing thirst, and aphonia, conditions which are all occasionally seen in these cases. Leichtenstern has calculated the amount of water lost from the tissues, and his investigation shows that, together with the concentration of the blood, there is a relative increase in the percentage of hemoglobin. The loss of water may be very considerable and may amount to 24 per cent. of the volume of the blood.

Another symptom that I have mentioned, which has an entirely different cause, is the occurrence of very profuse perspiration. This sweating occurs chiefly in the very severe cases of intestinal obstruction, and has the same significance as the attacks of perspiration in the death agony and in syncope. Immermann has carefully investigated the pathogenesis of these paroxysms, and has shown, by a series of convincing tests, that the sweating in these instances is undoubtedly due to the paralysis of the central nervous apparatus which inhibits the secretion of the sweat-glands.

While sweating, therefore, is an indirect symptom, the abnormalities of the urinary secretion that are occasionally very pronounced in acute and severe cases of strangulatory occlusion of the bowel must, on the other hand, be considered to be a direct symptom of collapse. The amount of urine excreted may become very small—in fact, complete anuria has been observed. This diminution in the urine is undoubtedly, to a certain extent, due to the loss of water by vomiting and hyperhidrosis, but these two factors alone are not the essential cause, and certainly not the only factors that produce oliguria and anuria in occlusion of the bowel. That this is the case can readily be shown by

examining the blood in these cases, for it will be found that oliguria and anuria may appear before concentration of the blood by loss of water has occurred. The chief cause for the decrease in the urinary secretion is the fall of arterial blood pressure, and this, in its turn, is due to the shock produced by the occlusion of the bowel. Albuminuria, cylindruria, and other urinary phenomena sometimes met with in intestinal strangulation have been discussed separately elsewhere (p. 165).

The patients, as we have already said, remain conscious until the end in the great majority of cases. This is a very distressing feature of the disease, for the suffering of the patient is, of course, greatly increased thereby. In very rare instances we occasionally see delirium or coma, and in other rare instances local or general convulsions. It is difficult to determine the exact cause of these nervous symptoms : they may be due to anemia of the brain following collapse ; they may be due to the concentration of the blood, owing to the loss of water that occurs ; or, as Leichtenstern remarks, they may be " uremic " in character, particularly if the nervous symptoms appear after anuria has persisted for some time. Finally, they may possibly be an expression of a form of autointoxication which will be discussed presently. As I have said, it is impossible at present to put one's finger on the exact cause of the nervous syndrome occasionally presented ; it is quite possible that in different cases different factors are operative, or that in any given case several factors are active at the same time.

In one of the preceding paragraphs I said that two theories have been advanced to explain the significance of the symptoms presented in occlusion of the bowel. One of these was the " nervous reflex theory " ; the other one, the " intoxication theory." Humbert, who is an adherent of the latter theory, has discussed all its possible consequences, and comes to the conclusion that the symptoms produced by occlusion of the bowel are an expression of " koprhémie." Bouchard, Buchanan, Albu, Kukula, and a number of other authors also seem inclined to support this theory. The quintessence of this theory is that as soon as the obstruction occurs and the propulsion of intestinal contents is interfered with, excessively large quantities of bacterial decomposition-products of proteid material are formed. These products, it is believed, are absorbed into the blood, and there exert their toxic effect ; the result is the serious clinical picture observed. Which of these substances exerts the poisonous action is still in doubt. Indol, phenol, and skatol have been incriminated and also the aromatic oxyacids ; perhaps, too, hydrogen sulphid, acetone, and aceto-acetic acid. Kukula obtained from the alcoholic extract a substance giving the reactions characteristic of alkaloids, and considers this as the toxic agent.

Quite recently a number of authors have attempted to explain the syndrome presented in the early stages of intestinal obstruction by intoxication, and have called the condition " septic collapse." According to Buchanan and others, this intoxication is brought about by the bacillus of malignant edema, and that general scapegoat, the Bacterium coli commune. This view is supported to an extent by the work of

Reichel and others, who found that in strangulation the wall of that portion of the bowel that is included in the strictures becomes readily permeable for intestinal microbes. These organisms, after penetrating the wall of the intestine, enter the peritoneal cavity and may then spread and give rise to general infection throughout the organism, but without necessarily producing purulent inflammation of the peritoneum.

It is quite impossible to enter into all the details of the intoxication theory here, and the same course must be adopted as in the account of the nervous reflex theory—namely, a very brief description only.

In my opinion it is possible, and even probable, that some of the very rare cerebral symptoms of this disease—the delirium, the coma, the rare syndrome called " strangulation typhoid," the rise of temperature without peritonitis—are all due to an autointoxication or infection such as we have described. Even the nephritis may possibly be due to the absorption of toxic products. The latter view is doubtful, however, and such a careful observer as Senator expresses serious doubts in regard to the validity of this assumption ; nevertheless it must be conceded that the autointoxication theory, in the light of our present knowledge on the subject of intestinal poisons and their absorption, has much in its favor, but let me emphasize expressly that in my opinion autointoxication can be made responsible only for a very small proportion of the symptoms presented in acute occlusion of the bowel, such as some rare symptoms which occasionally complicate the ordinary clinical picture. Those symptoms, on the other hand, which are perhaps the most characteristic feature of occlusion of the bowel,—for instance, collapse,—must be explained on the reflex theory. It seems much more probable that the latter symptoms are produced by reflex irritation in the manner we have outlined. It seems almost impossible to explain a large series of facts on the basis of the intoxication theory. How, for instance, can it account for the occurrence of the most serious symptom of occlusion of the bowel—namely, the profound collapse within six, twelve, or twenty-four hours after the onset of occlusion ? How account for the facts that, as a rule, the severity of the collapse corresponds to the suddenness of the strangulation, the violence of the internal pain, and the individual irritability of the nervous system, and finally that these symptoms may be entirely absent when occlusion of the bowel occurs gradually ? I need only mention this one clinical fact in order to show that its pathogenesis cannot be explained on the basis of the autointoxication theory. A detailed consideration of the whole question is impossible here, but I wish to place on record my emphatic opinion that unprejudiced clinical observation necessarily forces us to the views I have just enunciated.

PROGNOSIS AND COURSE.

Complete occlusion of the bowel, whatever its primary cause, is a horrible disease. There are certain differences in the course of the malady, according to the underlying organic causes. Some forms are

more malignant and run a more rapid course than others, but all are disastrous in the extreme. If any arrangement into groups and categories be made, the most unfavorable forms of acute occlusion of the bowel, and the ones with the worst prognosis, are the acute forms of knotting of the bowel and of internal hernia. It is true that occlusion of the bowel supervening on chronic stenosis of the intestine offers an equally unfavorable prognosis as soon as occlusion is complete, as the two forms mentioned above. If occlusion of the bowel supervenes in the course of chronic stenosis due to carcinoma, the situation becomes still more acute and distressing.

The spontaneous cure of any form of occlusion due to a chronic progressive process is quite out of the question. The life of a patient suffering from occlusion of the bowel following a chronic process, provided the occlusion is not due to blocking of the bowel by fecal matter, or, as in certain rare instances, to compression of the bowel from without, can be saved only by surgical interference. In some of those forms of occlusion of the bowel that occur acutely the prognosis may be considered somewhat more favorable, in particular if the occlusion is due to obstruction of the bowel lumen by gall-stones or foreign bodies, and when due to intussusception. Cases of this kind occasionally recover spontaneously or under purely medical treatment. In the sections on Volvulus of the Intestine and on Internal Hernia of the Bowel it will be shown how and to what extent a spontaneous cure of these conditions is possible. In the latter conditions a cure is possible, but unfortunately it rarely occurs in actual practice, since when intestinal obstruction has fully developed, death usually occurs unless prevented by operative interference.

The prognosis of fully developed occlusion of the bowel is dependent on a variety of factors: in the first place, the anatomic nature of the process determines the course of the disease in the acute forms; the violence of the shock that follows the occlusion, the rapidity and the severity of the symptoms of collapse that follow this accident, all have an important bearing on the prognosis. The more intense all these symptoms, the more serious the case. Up to a certain degree individual and constitutional conditions must be considered, for they too exert some influence on the course of the disease. Very young children, for instance, and decrepit and senile subjects, and weak and cachectic persons are, of course, in greater danger than vigorous and healthy subjects. The life of these patients is not threatened so much by the interference with the onward passage of feces and by the occlusion of the intestine, nor by the different phenomena which are the direct mechanical result of these conditions, as by the symptoms of collapse that develop. Even fecal vomiting does not constitute so immediate a danger to life as collapse. In acute occlusion of the bowel, moreover, the seat of the occlusion is of some importance in the prognosis, in so far as occlusion of the small intestine is more dangerous than occlusion of the large intestine. This generalization, however, is hardly justifiable; in fact, I consider it quite valueless in prognosis, for the extremely severe

symptoms produced by volvulus of the sigmoid flexure show that occlusion of the large intestine may be just as dangerous, and more so, than occlusion of the small intestine in general. Another very important factor that determines the prognosis is the occurrence of peritonitis.

It is impossible to formulate any general rules with regard to the duration of life after occlusion of the bowel has occurred. Individual cases vary greatly in this respect; as has been said, the anatomic character of the obstruction and a number of individual factors influence the course and termination of the disease. In intestinal obstruction with sudden onset the duration of the disease may only be eight hours, or the patient may linger for twenty days. If the disease terminates in recovery,—for instance, when obstruction due to intussusception is relieved by sloughing of the intussuscepted portion of the intestine,— the process may, of course, be indefinitely prolonged. The reader is referred to the paragraphs dealing with the different anatomic forms of occlusion of the bowel for other statements with regard to the course of these different forms.

Death in acute occlusion of the bowel occurs from collapse, from general exhaustion, or from peritonitis. The latter condition may be due to perforation of the bowel, but in the majority of cases occurs without such perforation. (For the symptoms of this condition the reader should refer to the section on Peritonitis.) In those forms of occlusion of the bowel that run a slow course death may be caused by a number of other possible factors—for instance, phlegmonous processes and suppuration in the abdomen, septicemia in all its different forms, and, finally, pneumonia from aspiration of particles of feculent material during stercoraceous vomiting.

MALIGNANT NEOPLASMS OF THE INTESTINAL CANAL
(Neoplasmata Maligna Intestini).

MALIGNANT tumors of the intestine are comparatively rare, so that, from their incidence, they occupy a very subordinate position in the consideration of diseases of the intestine.

[Payne's[1] statistics tend to show that cancer is becoming increasingly frequent as a cause of death, and that in this increase there is a marked predominance of cancer of the digestive organs.—ED.]

However, the clinical picture presented by them is exceedingly grave and severe. These lesions of the intestine, therefore, in this respect occupy one of the most important positions in the pathology of the intestinal canal. Among the malignant tumors met with in the intestine carcinomata are by far the commonest. Sarcomata and lymphosarcomata are much less frequent than carcinomata, but present a clinical picture which is so typical, and in the majority of cases so uniform, that a separate section will be devoted to them.

[1] Payne, "Increase of Cancer," *Lancet*, 1899, vol. ii.

CARCINOMATA.

Etiology.—Our knowledge of the etiology of carcinoma of the intestine is in the same state as that of carcinoma in general. The various theories that have been advanced with regard to the origin and the nature of carcinomata in general apply also, of course, to cancer of the intestine in particular. I need only mention some of the theories that have been advanced—for instance, Thiersch's hypothesis with regard to the disturbance of histogenetic equilibrium in advanced years; Cohnheim's hypothesis of the embryonal origin of carcinomata; then the parasitic infectious theory advocated by a number of authors, recently also by Leyden; and the theory of irritation, according to which traumata, chronic irritation, and chronic inflammatory processes are credited with an important and significant rôle in the genesis of carcinomata. We need scarcely go further into this discussion. The established clinical and anatomic relations in carcinoma lend a certain amount of support to the hypothesis that in its development chronic irritants are instrumental, at least in a secondary way. This has been frequently advanced, recently again by Brosche and Gockel-Boas. Of course, these irritants can be regarded only as a species of *causa occasionalis.*

A summary of the facts that I have indicated reads approximately as follows: Carcinoma of the intestine, in the overwhelming majority of cases, occurs in certain points of election in the intestinal canal (for the details see the next paragraph). Cancer seems to form particularly in those portions of the intestinal tract in which it may be assumed that rapid and constant irritation of the mucosa occurs, probably as the result of relatively prolonged stagnation of the intestinal contents in these portions of the bowel. These points of election are the same as those in which so-called decubital or stercoral ulcers most frequently develop. In the case of the stomach it may be considered as proved that carcinoma develops comparatively often from the cicatrix of a simple ulcer of the stomach, and from analogy the assumption seems at least not improbable that occasionally the well-known annular form of carcinoma of the intestine in the sigmoid flexure and the other flexures of the colon develops on the basis of cicatricial tissue resulting from annular decubital ulcers of these areas. As a matter of fact, a number of observers have noticed that carcinoma of the intestine may develop from dysenteric cicatrices of the bowel. I have had occasion to verify this fact; in a case operated on by Billroth, in which I had diagnosed a dysenteric cicatricial stricture of the bowel in the region of the splenic flexure, stricture was found, but, in addition, there was a carcinoma in the scar. In the account of sarcomata of the intestine in a subsequent section a very interesting case in which multiple lymphosarcomata developed in tuberculous cicatrices of the intestine will be referred to.

[Letulle [1] found an extensive ulcer in the first part of the duodenum, with colloid carcinoma developing near its center. The patient, a man

[1] Letulle, *Bull. Soc. Anat.*, Paris, 1897, p. 721.

aged fifty-five, had been treated two years previously for melena. Perry and Shaw [1] refer to five, and Nattan-Larrier [2] to five more, including Letulle's, making ten in all.—ED.]

Sex is of no significance in regard to the relative frequency of carcinoma of the intestine. It seems, however, to judge from the majority of statistics on the subject, that carcinoma of the intestine is somewhat commoner in males than in females. Age has a remarkable influence as regards the incidence of carcinoma of the intestine. In general, carcinoma is more frequent in the second half of life than in the first, and this general rule also obtains in carcinoma of the intestine. This lesion is commonest between the fortieth and the sixty-fifth year. At the same time it is a very remarkable fact that carcinoma of the intestine is encountered quite frequently before the fortieth year. I suppose that every medical man who has had much clinical experience has observed this. Carcinoma of the intestine has been found even in children. I have seen a case of carcinoma of the cecum in a boy of twelve years; Zuppinger, carcinoma of the sigmoid in a twelve-year-old girl; Czerny, carcinoma of the rectum in a thirteen-year-old boy; Stern, the same in an eleven-year-old girl. Schöning has reported two cases of rectal carcinoma in girls of seventeen; Clar has described a carcinoma medullare of the colon in a boy of three years; Duncan, carcinoma of the small intestine in a three-and-a-half-year-old boy. Lonart has found in the literature 61 cases of carcinoma of the large intestine between the ages of twenty and thirty.

[Kanthack and P. Furnival [3] described a colloid carcinoma of the ascending colon in a boy aged seventeen years. W. A. Garrard [4] reported a colloid carcinoma of the sigmoid flexure in a boy aged twelve years.—ED.]

Maydl, from his own clinical material and that of the General Hospital in Vienna, has calculated that of all cases of carcinoma of the intestine, one-sixth occur between the thirtieth and the fortieth year, and one-seventh before the thirtieth year. He compared these statistics with those of 69 cases of carcinoma of the esophagus, in which there was no case under thirty years of age. Among 331 carcinomata of the lip, only 5 cases occurred before the thirtieth year, and among 27 of carcinomata of the breast, only 5 occurred in women under thirty. It is well to remember this early occurrence of carcinoma of the intestine, in order to avoid serious errors in diagnosis.

Pathologic Anatomy.—*The Seat of the Carcinoma.*—In studying the site of carcinoma of the intestine it is a remarkable fact that cancer of the bowel is very rare in the small intestine, occurs frequently in the colon, and very commonly in the rectum. Combined tables of statistics, as well as the statistics of numerous individual observers on cancer of the bowel, all agree as regards these peculiar differences in

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[1] Perry and Shaw, *Guy's Hosp. Repts.*, 1893, p. 274.
[2] Nattan-Larrier, *Gaz. des Hôp.*, December 9, 1897.
[3] Kanthack and Furnival, *Trans. Path. Soc.*, vol. xlviii., p. 99.
[4] Garrard, *Quarterly Medical Jour.*, April, 1897.

the situation of cancer. Here and there the details of the statistics show slight discrepancies, but in general there is a consensus of opinion to this effect. From a rather wide experience in this branch of medicine I can corroborate this.

In order to illustrate the differences in the distribution of carcinoma throughout the intestine let me quote a few figures : Maydl reports the following : During the twelve years from 1870 to 1881, 20,480 autopsies were performed in the Pathologic Institute of the Vienna General Hospital. Among these, 1460 cases of carcinoma were discovered, and of these carcinomata, 100 were found in the intestine. Of the 100 intestinal carcinomata, 2 were found in the duodenum, 4 in the ileum, none in the jejunum, 46 in the large intestine (1 in the vermiform appendix, 9 in the cecum, 6 in the ascending colon, 17 in the colon in general, 13 in the sigmoid flexure) ; finally, 48 were found in the rectum.

I have studied the statistics of the Pathologic Institute in Vienna during the following twelve years, namely, from 1882 to 1893, and can supplement Maydl's figures as follows : During these years there were 21,358 autopsies, among which there were 2125 cases of carcinoma, and of these carcinomata, 243 were found in the intestine. Of the intestinal carcinomata, 5 were in the duodenum, 6 in the ileum, none in the jejunum, 118 in the large intestine (1 in the vermiform appendix ; 14 in the cecum ; 63 in the colon in general ; 40 in the sigmoid flexure), and 114 in the rectum. Altogether, therefore, 343 cases of carcinoma of the intestine were discovered within a period of twenty-four years among the autopsy material of the Pathologic Institute of the General Hospital in Vienna. Of these 343 carcinomata, 7 were found in the duodenum, 10 in the ileum, none in the jejunum, 164 in the colon, and 162 in the rectum.[1]

Bryant collected statistics of 110 autopsies ; among these, carcinoma was found 6 times in the small intestine, 7 times in the cecum and the ileocecal region, 19 times in the transverse colon, including the hepatic and splenic flexure, 78 times in the sigmoid flexure and the rectum. Leichtenstern has also published some statistics based on his notes of living subjects and of pathologic examinations. He found 616 carcinomata in the large intestine, 42 in the sigmoid flexure, 11 in the descending colon, 30 in the transverse colon, including the hepatic and splenic

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[1] It is astonishing to find that there is so marked a difference between the first twelve years (1870 to 1881) and the second twelve years (1882 to 1893) in the number of carcinomata of the intestine ; in the first period there were only 100 ; in the second, 243. This is particularly striking, because so far as I know the clinical material admitted to the General Hospital in Vienna did not differ much in these two periods of twelve years. I cannot refrain from repeating a remark that I made in 1893 when discussing this peculiar fact with Billroth, namely, that in my private practice I had also met with a much larger proportion of cases of carcinoma of late years than formerly. Is this due purely to external circumstances, namely, that cases of carcinoma of the intestine come to Vienna because we have succeeded in perfecting the surgery of the intestine to such a degree in this city ? I do not think that this can be settled for the present, and I prefer to withhold my judgment on the matter ; at the same time it is certainly remarkable that the increase of carcinoma of the intestine is observed both in the hospitals and in private practice. [Payne's conclusions, based on English statistics and showing that the incidence of carcinoma and especially of the intestinal tract is increasing, bear out these remarks.—ED.]

flexures, 6 in the ascending colon, 20 in the cecum, 3 in the vermiform appendix, 9 in the ileocecal valve, 13 in the lower ileum, 3 in the middle portions of the ileum, 17 in the jejunum and duodenum. Müller studied 5621 autopsy-protocols of the Pathologic Institute in Bern from the year 1886 to 1891, and found 521 cases of carcinoma of all organs in general. Of these, 19 were in the rectum, 13 in the large intestine, and 9 in the small intestine; of the latter, 6 were in the duodenum and 3 in other portions of the small intestine.

Statistics based on observations on the living demonstrate that carcinoma of the rectum is by far the most frequent form of intestinal cancer. This is also shown by Leichtenstern's data, according to which 80 per cent. of all intestinal carcinomata are found in the rectum. According to Maydl's communications, 246,827 patients were treated in the General Hospital of Vienna during the period of twelve years mentioned. Of these cases, 6287 were afflicted with carcinoma; of these cancer cases, 254 were suffering from cancer of the intestine, and of the latter, 224 cases were cases of cancer of the rectum. This extraordinary prevalence of carcinoma of the rectum in statistics gathered from case reports in the living, as compared to the comparatively small number of rectal carcinomata that are found on autopsy, is, of course, due to a variety of factors. In the first place, carcinoma of the rectum is absolutely more frequent than any other form of carcinoma of the intestine; in the second place, this form of carcinoma, owing to its location near one of the orifices of the body, is readily accessible and can be diagnosed without difficulty; in the third place, cancer of the rectum can readily be treated by surgical methods, so that many cases of carcinoma of the rectum are operated upon and are not examined postmortem in the general hospital.

A very characteristic point in these statistics is the great rarity of carcinoma of the small intestine proper as compared to the frequency with which carcinoma of the large intestine is discovered. Du Castel and Journet claim that the small intestine is quite frequently affected with carcinoma; in this statement they place themselves in opposition to the generally accepted view, but their peculiar position can readily be explained by the fact that they consider the ileocecal valve as part of the small intestine and consequently include carcinomata of this valve among carcinomata of the small intestine. Lubarsch has correctly criticized this manner of subdivision, and has called attention to the fact that the ileocecal valve cannot properly be considered a portion of the small intestine. Another remarkable fact is the peculiar immunity of the jejunum and ileum from carcinoma as compared to the duodenum, for the duodenum is almost as frequently involved by primary carcinomatous lesions as the whole jejunum and ileum together.

It is unnecessary to quote further statistics. While a number of other statistics differ in certain details, they all point to the conclusion emphasized above, and show that I am correct in stating that the rectum is the part of the bowel most frequently affected by carcinoma. Next in order of frequency comes the sigmoid flexure, then the cecum, and

then the other portions of the colon. The small intestine is much less frequently involved in this process. Finally, the duodenum is the part of the small intestine relatively most often affected.

Carcinoma of the intestine is almost always primary in character; secondary carcinomatous growths only exceptionally occur in the intestines. If we deduct 41 cases of primary carcinoma of the intestine from the 521 cases of carcinoma of Müller that we have quoted above, we will find that among the 480 cases that remain only 2 gave rise to metastases in the intestine—namely, one case of carcinoma of the stomach in which there was a metastatic growth in the mucous membrane of the intestine, and one primary carcinoma of the testicle with a secondary growth in the rectum. Weigert has reported an interesting case of a carcinomatous ulceration of the leg that was followed by widespread secondary carcinomatosis. In this instance a number of the carcinomatous nodules were also found in the upper portion of the small intestine.

[In melanotic growths metastases may occur in the intestine. In a case of a primary melanotic tumor of the right big toe described by H. C. Thomson[1] (as a sarcoma), there were 20 melanotic growths, varying in size from a marble to a pigeon's egg, projecting into the lumen of the small intestine. In a case of widely generalized melanotic sarcoma secondary to an intra-ocular growth I[2] found numerous small melanotic growths on the mucous membrane of the small intestine.—ED.]

Carcinoma may involve the intestine in another way—namely, by direct continuity of tissue; thus carcinoma of some neighboring organ may extend directly into the intestine. This class of secondary carcinomata of the intestine is, of course, radically different in character from metastatic carcinomata, and cannot be included in statistics of cancer metastases. As a matter of fact, even this method of development of carcinoma of the intestine is comparatively rare. Occasionally carcinoma of the stomach, the gall-bladder, the mesenteric glands, the female genital apparatus, etc., extends directly to the intestine and leads to a cancerous infiltration of the bowel. In these cases it must be distinctly remembered that the intestine is directly involved by contiguity and not with metastatic transportation of cancer particles through the blood or lymph-channels.

[Carcinoma of the pylorus rarely spreads into the duodenum, but it may do so. In 10 specimens, in which excision for carcinoma of the pylorus had been performed, Cuneo and Lucène[3] found that in only 1 was the extension of carcinoma into the duodenum visible to the naked eye, but microscopic examination showed that in 3 other cases there were discrete foci of carcinoma in the lymphatic vessels of the duodenum, which, however, did not extend further than 2 cm. from the pylorus. This point is of practical importance, for even if the

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[1] H. C. Thomson, *Trans. Path. Soc.*, vol. l., p. 237.
[2] Rolleston, *Lancet*, 1901, vol. i., p. 1121.
[3] Cuneo and Lucène, *Bull. Soc. Anat.*, Paris, 1900, p. 732.

duodenum appears healthy in a case of pyloric carcinoma, the surgeon would still do well to remove the first 3 cm. of the duodenum together with the pylorus.—ED.]

[Pye Smith [1] has recorded a case in which there were two carcinomatous growths in the duodenum—one just beyond the pylorus, the other, probably more recent, three inches lower down. The latter was regarded as having been formed by *direct transference or grafting* from the other growth, and not as a metastasis.—ED.]

While primary carcinoma elsewhere rarely gives rise to secondary growths in the intestine, primary carcinomata of the intestine conversely have a great tendency to cause metastases in other organs. There is no constant rule with regard to the distribution of these metastases; each individual case differs in this respect, and almost any organ of the body may be infected with carcinoma in this way. To judge from the statistics published by Müller, carcinoma of the small intestine seems to form metastases with greater frequency than carcinoma of the colon or the rectum.

In carcinoma of the intestine secondary metastases are most often found in the lymph-glands. This applies generally to all carcinomatous metastases, the lymph apparatus being the most prone to metastatic involvement. As in all other forms of lymphatic involvement, the regional lymph-glands are first involved in carcinoma of the intestine— namely, those situated nearest to the growth. In performing operations for the removal of carcinoma of the intestine, the lymph-glands in the immediate vicinity of the affected area should always be carefully palpated, for the general technic of the operation will require modification accordingly. The general operative procedure is different when the lymph-glands are healthy than when they are diseased or are thought to be infected with secondary growth. There is considerable diversity of opinion among different authors as regards the absolute frequency of secondary metastatic involvement of lymph-glands in carcinoma of the intestine. According to Müller's statistics, the lymph-glands are relatively free from secondary metastatic involvement, especially in carcinoma of the rectum. Confirmation of this fact would be of great interest and significance as regards the indications for operative treatment of this extremely common form of carcinoma. Müller's views have been confirmed by a number of writers. Maydl, for instance, goes so far as to say that the fact that secondary metastatic infection of the lymph-glands is comparatively so rare in carcinomata of the large intestine greatly favors the radical cure of cancer of the colon by operative interference. Hauser is not in accord with this view, for he claims that in many cases of carcinoma of the rectum, even when the growth is excised at an early stage, the lymph-glands in the periproctal cellular tissue are frequently infiltrated with carcinoma.

Secondary metastases in the liver are found almost as commonly as metastases in the lymph-glands. The liver may be involved in all the forms of primary carcinoma of the intestine, wherever situated; next in

[1] Pye-Smith, *Trans. Path. Soc.*, vol. xlv., p. 63.

order of frequency come the peritoneum, then the omentum, the mesentery, and the lungs. The other organs of the body are less commonly the sites of secondary growths. The kidneys appear to be involved with relative frequency as compared with the other organs of the latter class.

Hauser calls attention to the remarkable fact that certain peculiar differences in the character of the metastases and the manner of the development of secondary metastatic nodules can be noticed in the different forms of carcinoma of the large intestine. Colloid carcinoma, for instance, of the large intestine very rarely produces metastases in the internal organs, but chiefly involves the serosa. Large medullary tumors, on the other hand, usually produce carcinomatous infiltration of neighboring lymph-glands only, and not in any other organs. Finally, simple and scirrhous carcinomata of the intestine, even when small, frequently produce enormous tumors of the liver.

Histology.—As I have mentioned above, the overwhelming majority of malignant neoplasms found in the intestine are carcinomata. Sarcomata or lymphosarcomata are only rarely met with in this location. Hauser has recently investigated the histologic features of carcinoma of the intestine with great thoroughness, and in this section his excellent description will be closely followed. Within the narrow limits of this hand-book, however, a brief summary of his results is all that can be admitted, while the available space will prevent a review of the extremely copious literature on carcinoma of the intestine.

Hauser's exhaustive investigations thoroughly confirm the views of Thiersch and Waldeyer as regards the nature of carcinoma. Their view that carcinoma can develop only from epithelial structures is in opposition to a number of other theories that were enunciated many years ago by Virchow, and are adhered to by him even to this day. In the intestine in particular carcinoma always starts from the cylindric epithelium of the intestinal glands (*carcinoma cylindro-epitheliale adenomatosum*). All histologic modifications of carcinoma met with in the intestine can readily be shown to be of this origin. By far the most common form of carcinoma is *carcinoma adenomatosum simplex*. This form of cancer is most frequently found in the large intestine, chiefly the lower portion of the rectum immediately above the sphincter ani. *Carcinoma adenomatosum simplex* is, however, also occasionally found in the small intestine. The medullary carcinoma (*carcinoma adenomatosum medullare—" Markschwamm "*) and true colloid carcinomata (*carcinoma adenomatosum gelatinosum—"* Gallertkrebs ") are also characteristic forms of intestinal carcinoma. There has been some controversy as regards the histogenesis of the latter form of carcinoma; Hauser's view, which differs from that of other investigators, is that colloid carcinoma also starts in the glands of the mucous membrane of the intestine. A rare form of carcinoma of the intestine is that called *carcinoma adenomatosum muciparum microcysticum* by Zenker. Lastly, the form of carcinoma which is the commonest type found in the stomach—the so-called scirrhus (*carcinoma adenomatosum scirrhosum*)—is very rare in the

intestine; at the same time it must be remembered that simple adenomatous carcinomata of the rectum frequently show changes in certain parts of their periphery or at the base of ulcerations, which are frequently of a scirrhous character.

This is not the place to discuss the merits or demerits of the different views that have been advanced with regard to the genesis and the histologic details of the different forms of carcinoma. A rather animated polemic on this subject has been waged for some time, but it is not necessary to refer to the literature of the subject even in abstract. I shall merely mention in the briefest terms that, according to the investigations of Waldeyer, Hauser, and others, the primary changes in the development of carcinoma of the intestine are limited to the mucous membrane of the intestine. The fact that the proliferation of epithelium starts without exception from preëxisting glandular epithelium can easily be verified (there is one exception to this rule— *i. e.*, carcinoma of the duodenum; here the primary proliferation may start from the glands of Brunner, which are situated deep down in the wall of the intestine; in cases of carcinoma, moreover, that develop from cicatrices, the epithelial proliferation may start from the lining of glandular tubules which have grown deep down into the tissues of the intestine). The degeneration of glandular epithelium characteristic of carcinoma always begins at the fundus of the glands. In the subsequent course of the process the epithelium of the degenerated mucous glands of the intestine always proliferates through the membrana propria of the gland, then perforates the muscularis mucosæ, and finally continues its proliferation in the submucosa and the deeper tissues of the intestinal wall. A detailed description of the various histologic processes involved in the stages of development of intestinal carcinoma cannot be undertaken, but the sequence of events may be summarized as follows: In the first place, changes of the glandular epithelium; then proliferation of this epithelium; changes in the form of the glands; development of several layers of the epithelial cells of the tubules; obliteration of the glandular lumen and metamorphosis of the glands into solid cylinders of epithelium; changes in the interglandular tissues; progressive changes in the character of the epithelium; proliferation and changes which are due to retrogressive metamorphosis. The latter changes, according to the character they assume, may produce different varieties of simple adenomatous carcinomata, consisting of cylindric epithelium. Later the inflammatory reaction of the connective tissues in the vicinity of the neoplasm in its turn exerts an influence on the histologic and anatomic character of the cylindric epithelioma. (For a clear description of all the details of these changes the reader should refer to Hauser's excellent monograph, which contains some admirable illustrations.)

Macroscopic Appearance.—The size, the shape, and the surface of carcinoma of the intestine vary so greatly in individual cases that, from the point of view of morbid anatomy, several types of intestinal carcinoma may be described. Marked differences are also observed in

the distribution of the neoplasm and in the sequelæ which result from its presence in different parts of the intestine. It can readily be understood that there is a great difference between an annular, cartilaginous, hard induration, resembling scar tissue, in the flexures of the colon, and a circumscribed nodule with a smooth surface at the ileo-cecal valve, or an ulcerating, gangrenous, enormous, cauliflower growth in the lower portion of the rectum.

Occasionally an opportunity occurs of examining a carcinomatous growth in the first stages of its development. As a rule, the neoplasm under these conditions appears as a very small nodule and occupies a circumscribed portion of the intestinal wall; generally it can be felt distinctly through the uninjured mucous lining of the intestine. The subsequent growth and development of a nodule of this character depend in part on the histologic structure of the neoplasm, in part on other unknown factors. Great variations in the form are seen. The nodule may develop into a single massive tumor as large as two adult fists or larger, or it may develop into several, usually smaller, masses of proliferating tissue. The latter development is much less frequently met with than the former. As a rule, soft tumors and tumors containing very many cells grow to much greater dimensions than other neoplastic growths. In fact, the size of the tumor is usually directly proportionate to the number of cells it contains and the softness of its structure. In some instances the neoplasm protrudes into the lumen of the intestine like a polypus; in other cases the growth extends over large surfaces of the bowel so that the whole bowel-wall may be converted into an almost rigid tube by this development of new growth throughout long portions of the intestine. Finally, an annular form of growth is common. Annular carcinomata of the intestine may develop either from one of the small nodules described above or in the base of an annular cicatrix due to ulceration of the bowel; the latter method of development is the most frequent one in annular carcinomata. These small carcinomata involve the circumference of the intestine, and are occasionally only 1 or 2 cm. in diameter. They are hard and, as already stated, annular. They differ very little from simple cicatricial tissue, and it is often impossible to distinguish between cicatricial tissue and this form of carcinoma, except by microscopic examination.

When death occurs relatively early, owing to some complication or accident, the surface of carcinomata of the intestine is occasionally perfectly smooth. In the great majority of cases, however, the surface of the neoplasm is disintegrating and ulcerating. This ulceration is frequently gangrenous in character, and may be regarded as directly responsible for a number of clinical symptoms which occasionally develop in the course of intestinal cancers, such as the passage of blood or of pus by the bowel, etc. It can readily be understood why carcinomata of the intestine undergo ulceration earlier than carcinomata in many other organs, for the intestinal contents and secretions naturally exercise an effect on the neoplasm that produces disassimilation of those parts of the growth which protrude into the lumen of the intestine.

Some authors call attention to what they consider an anatomic peculiarity of carcinoma of the intestine—namely, the tendency of this form of neoplasm to develop in an annular manner; that is, to involve the whole circumference of the bowel. As a matter of fact, this anatomic arrangement of cancer of the intestine is seen in the great majority of cases. The annular character of the growth is quite typical, and occasionally neoplastic tissue encircles the intestine like a cicatricial ring or like a thread of scar tissue. It is important to remember this when considering the advisability of surgical interference and in deciding upon the operative technic for removing this form of carcinoma. It can readily be understood that a neoplasm completely encircling the intestinal lumen must necessarily produce stenosis of the intestine. In fact, strictures of the bowel due to growth are often so extreme that nothing but a thin pencil can be passed through them, while occasionally it is difficult to pass even a fine sound through the stenosis. The other forms of carcinoma of the intestine may also narrow the lumen of the bowel; they may effect this by infiltrating wide areas of the intestinal wall so that the area of the bowel involved in this process is converted into a rigid cylinder or into a funnel-shaped piece of intestine. Another way in which stenosis of the intestine can be produced by carcinomata of the intestinal wall is by the proliferation of cancer tissue into the lumen of the bowel. There is nothing typical about the production of stenosis of the intestine by carcinoma, for the same process is seen in carcinoma of other tubular organs—as, for instance, of the esophagus, the pylorus, etc.

In by far the largest number of cases we have but one stenotic point, but exceptionally a case is encountered in which stenosis is multiple. Küttner had a case with 22 points of narrowing—18 in the small and 4 in the large intestine. The primary carcinoma was in the sigmoid flexure (*cf.* p. 470).

As soon as stenosis of the bowel occurs, a series of secondary consequences develop which are typical of this condition: the contents of the bowel stagnate above the stenosis; the intestine becomes dilated immediately above the stenosis; its walls undergo a variety of changes that are characterized by catarrh, hypertrophy of the muscular walls, ulceration, and finally perforation. Only a brief summary of the sequelæ of carcinomatous stricture of the intestine is given here. These conditions have all been carefully dealt with in the general description of chronic enterostenosis, and the reader should refer to these sections for all the details.

One of the most important sequelæ of carcinomatous stenosis of the bowel, at least so far as the clinical picture of this disease is concerned, is the stagnation of the bowel contents. Whenever this occurs, palpation of neoplastic tumors of the intestine becomes difficult, inasmuch as the scybala which accumulate above the stenosis form hard nodular masses and are frequently indistinguishable from the carcinoma proper. It is evident that under these conditions the most serious mistakes can be made. Any attempt to estimate the size of the tumor is

specially fallacious unless the presence of scybala in the immediate vicinity of the growth can be excluded. It is often surprising to find a very small neoplasm at the autopsy of a case in which, during life, an enormous growth was diagnosed. In such cases the autopsy often shows that the main part of the tumor felt during life is composed of hardened masses of fecal matter accumulated above the stricture.

Carcinoma of the intestine, however, does not always lead to narrowing of the intestinal lumen, so that the symptoms of stricture of the bowel are not necessarily present in every case of intestinal cancer. Occasionally, from the symptoms of enterostenosis, the diagnosis of cancerous stricture is made, and yet, after a time, all the signs of this condition disappear again. This is always due to the fact that from ulceration and degeneration of the neoplasm, the lumen of the bowel, previously narrowed by this neoplasm, again becomes patent. In other instances the intestinal channel may become patent in very peculiar ways; for instance, when a direct connection is established between two loops of intestine which happen to lie in close proximity to each other. This process of anastomosis, brought about by a carcinomatous growth, will be described in one of the following paragraphs. In sarcomatous infiltration of the intestine there is often a very peculiar form of direct dilatation of the intestinal lumen. This secondary effect of a neoplastic growth will be considered in the section on Sarcoma of the Intestine.

Peritoneal adhesions very frequently develop around the part of the intestine affected with carcinoma, and may unite it with other portions of the intestine or with any one of the organs in its neighborhood, according to the situation of the growth. When different loops of intestine become adherent in this way, operative interference is rendered more difficult; in fact, it may be said that the formation of these adhesions between different portions of the intestine and between the intestine and other abdominal organs constitutes the most serious obstacle to the surgical treatment of intestinal carcinoma. This difficulty is enhanced by the fact that the presence of these adhesions can only rarely be diagnosed in advance; in fact, there are hardly ever any grounds for even suspecting their presence. The condition of affairs becomes still more complicated when the adhesions, as they frequently do, bring about grotesque and extremely bizarre deformities and displacements of the abdominal viscera. We may, for instance, on opening the abdomen, find that the carcinomatous cecum is adherent to the sigmoid flexure, and that the former is pulled over to the left, or, conversely, that the latter has been drawn over to the right by this adhesion; or, again, a tumor of the hepatic flexure of the colon may be adherent to the peritoneum, the omentum, the small intestine, and the cecum, so that all these organs form a large conglomerate mass in the peritoneal cavity.

When the neoplasm extends to other adjacent organs of the abdominal cavity by direct contiguity and undergoes ulceration, perforation of the bowel naturally results and communications are established between

the lumen of the loop of intestine originally affected and other coils of intestine or other organs, as the stomach, the bladder, or the vagina. In this way a great variety of fistulous passages may be developed between the hollow viscera of the abdomen. In very rare instances a carcinoma of the bowel may become adherent to the abdominal parietes and ultimately perforate outward through the skin, producing a cutaneous intestinal fistula.

[Pollosson[1] has recorded two cases of carcinoma of the colon in which the growth produced a spontaneous anastomosis between the large and the small intestine, and so presented the symptoms of obstruction. I have recently had a case under my care in which, after death, there was a similar fistulous passage between the large and the small intestine. —ED.]

The occurrence of peritoneal adhesions in carcinoma of the intestine has just been mentioned. Peritonitis following carcinoma may present a great variety of anatomic pictures. There may either be a simple circumscribed form of peritonitis, without, or more commonly with, adhesions, or there may be chronic diffuse or finally suppurative peritonitis (fecal abscess). These different forms of peritonitis will be considered in detail in the section on Diseases of the Peritoneum. Finally, rupture of the intestine into the general peritoneal cavity may occur, so that the contents of the bowel are poured into this sac. This occurs when the cancer of the intestine undergoes very rapid destruction, so that there is no time for the formation of peritoneal adhesions around the affected portion of the bowel. When infiltrated with carcinoma, the omentum and the mesenteric tissues may be converted into rigid, plate-like masses. Under these circumstances the free margin of the omentum becomes anchored and fixed, so that it forms a band across the abdomen. This fibrous band is a source of considerable danger, for the bowel may easily become knuckled or twisted when pressed against it. The mesentery also occasionally becomes kinked, and the intestine is, therefore, twisted.

Symptomatology.—Carcinoma of the intestine is relatively so frequent and possesses such marked clinical significance that it will, I think, be well to give a somewhat detailed account of the symptomatology of this disease. I do this even though I realize that the clinical picture is in many respects the same as that of other forms of stricture of the intestine.

It is quite impossible to paint a typical uniform clinical picture of carcinoma of the intestine. It is true that in this disease some phenomena and certain definite symptoms appear more frequently than others, but even the important symptoms, and those regarded as characteristic of carcinoma of the intestine, may be entirely absent in individual cases of the disease. These variations in the clinical features depend, in the first place, on the seat of the neoplasm in the intestine. In addition, a great deal naturally depends on the rapidity of growth and on the histologic structure of the cancer. The group of symptoms in a rapidly breaking-down medullary carcinoma will be very different

[1] Pollosson, *Lyon Médical*, April 16, 1899, p. 557.

from that in a case of a cicatrizing hard scirrhus. The sequelæ of a carcinoma of the intestine will also depend on infection of other organs, and will be modified according to the organ involved. A study of a number of the case-records of carcinoma of the bowel might at first sight lead to the impression that each given case is a different disease, for the picture presented may resemble simple habitual constipation in one instance, some form of piles in another, enterostenosis here, peritonitis there. Some cases may present the syndrome of severe icterus or advanced cachexia of unknown or undetermined origin. Careful analysis and examination of these cases, however, soon make it clear that carcinoma has to be reckoned with.

Owing to the special features of carcinoma of the intestine and the complicated and variable character of its clinical picture, it may be useful to give separate descriptions of the symptoms of cancer of the intestine corresponding to the different situations of the bowel in which the growth occurs. I believe that the description of the general clinical aspect of the disease will be clearer and more comprehensive if this plan is adopted, and shall, therefore, describe the symptoms—(*a*) Of carcinoma of the large intestine (from the cecum to the sigmoid flexure); (*b*) of carcinoma of the rectum; (*c*) of carcinoma of the duodenum; (*d*) of carcinoma of the small intestine.

Before beginning the separate description of cancer of the different portions of the bowel, a brief account will be given of a few general symptoms common to all forms of carcinoma, not only in the intestine, whatever part of the bowel be involved, but also of all other organs of the body. The most important of these general symptoms are the cancerous anemia and cachexia. As a rule, they are both seen together; occasionally one develops before the other. In many patients general weakness, pallor, and emaciation are the first symptoms which arouse a suspicion of the existence of some serious disease. This is particularly the case when there are only slight local symptoms, such as slight constipation, which is easily controlled, and possibly, in addition, an insignificant sense of weight or discomfort in the abdomen, which hardly attracts any notice. This sequence of symptoms is, however, comparatively rare. As a rule, the general symptoms appear at the same time and with the same severity as the local symptoms. Sometimes exactly the reverse holds good—namely, the local symptoms are very pronounced and severe, while the appearance of the patient belies the existence of any dangerous malady. It is quite impossible at present to state what factors determine these differences. The clinical and experimental data capable of throwing light on this subject are as yet too scanty to justify any far-reaching conclusions. In some instances there is early loss of appetite. Fenwick has shown that in a few cases of carcinoma of the intestine the glands of the intestine undergo serious changes early in the disease. Whether this is a universal occurrence in intestinal carcinoma remains to be shown by systematic examination of a large number of cases.

All forms of carcinoma, particularly those which run a rapid course,

frequently produce a rise of temperature and general fever. I believe this fact is now generally recognized and conceded by all clinicians. The fever is particularly marked in carcinoma of the stomach. This has been emphasized chiefly by Hampeln and a number of other observers. Uncomplicated carcinoma of the intestine may also produce a febrile rise of temperature. The number of positive observations on this sequel of carcinoma are scanty, but still I believe that the fact may be accepted. Kraussold, for instance, reports a case of carcinoma of the intestine in which there was fever. Two of his cases were particularly interesting and quite remarkable. Two individuals who had been perfectly healthy up to the time when they came under observation developed all the symptoms of what appeared to be an acute attack of perityphlitis. There was a slight degree of fever, which in one of the cases persisted from the beginning of the disease to the death of the patient, with evening exacerbations up to 103.8° F. (39.8° C.). In the other case the pyrexia persisted with slight remissions for five months, also up to the death of the patient. Both of these cases had carcinoma of the cecum—indeed, a few days after the onset of fever a tumor could be felt in this region.

CARCINOMA OF THE LARGE INTESTINE.

Pain is a very frequent symptom, but, of course, possesses no independent, specific diagnostic significance in this condition; besides, it may vary greatly in intensity and in the way in which it appears and starts, according to a variety of circumstances.

In many cases of carcinoma of the large intestine spontaneous local pain may be absent for a long time, even after a tumor is definitely palpable. Later in the course of the disease, however, localized pain usually appears. In isolated cases pain is completely absent throughout the course of the disease. This occurs particularly in cases of carcinoma of the large intestine running a very rapid course, and in cases in which acute intestinal obstruction occurs as a result of carcinoma of the intestine, and in this way causes death in a patient who possibly suffered no pain whatever up to the onset of acute obstruction. Cases of this kind, of course, are the exception, but still it is well to remember that they may occur, particularly in judging of the significance of the symptoms present in any given case of suspected carcinoma of the large intestine.

Spontaneous pain is frequently very slight, and may be manifested merely by a dull feeling of abdominal fulness or a general vague sense of discomfort. In other cases again the pain is very severe, but it rarely becomes unbearable. It is important to notice that the pain is always referred to the same region of the abdomen. This fact alone, of course, has no bearing whatever on the underlying cause of the pain, or, in other words, on the presence of carcinoma, for we know that localized intestinal pain may result from a great variety of causes. There is one other point on which special stress must be laid here, for it is frequently overlooked and may be a prolific source of error. In

several cases of carcinoma of the large intestine I have observed that the patients in the early stages of the disease complain for a time of circumscribed pain in some portion of the abdomen other than that in which the neoplasm is found later on. They may, for instance, complain of pain in the region of the sigmoid flexure when the carcinoma is situated in the cecum, or vice versâ. Here we are dealing with radiating pain; it may be said in general, however, that in carcinoma of the large intestine radiation of pain is relatively rare. Occasionally the picture of neuralgia of the sciatic nerve or of the anterior crural nerve is presented. In this case it will usually be found that the neoplasm exerts pressure on these nerves within the abdomen. Cases of this kind are, however, very rare. The local pain in carcinoma of the large intestine is usually increased by pressure over the painful area, whether or not a tumor can be palpated. This pain is frequently due to circumscribed peritonitis in the neighborhood of the tumor.

Another form of pain frequently complained of in carcinoma of the large intestine is an attack of colic. These paroxysms are very much more significant than spontaneous local or radiating pain, for they originate from entirely different causes and are different in character. Colicky pain in carcinoma of the large intestine is usually very severe. It may either be limited to a certain definite portion of the abdomen, or—and this is more frequently the case—may be diffuse throughout the abdomen. The histories of many cases of carcinoma of the large intestine show that the patients complain only of these paroxysmal attacks of colic. They state that before the onset of these attacks they were perfectly well, with a daily and regular evacuation of the bowels. Suddenly, either after eating some article of food that did not agree with them or after exposure to some other injury that they remember, a violent attack of pronounced colicky pain immediately developed. In other cases again the following history is given: The patients state that they gradually became more and more constipated, but that they suffered no pain whatever until suddenly the first attack of colic appeared. These paroxysms of colicky pain in carcinoma of the large intestine are the expression of gradually progressive stenosis of the caliber of the bowel and are produced by stagnation of the bowel contents. Attacks of colic of this character are without exception complicated with constipation, a condition which will presently be considered. Sometimes vomiting also occurs during these attacks. The paroxysms of pain recur at frequent intervals; in very exceptional cases only a single attack of colic appears; this may occur if the disease progresses very slowly and is far advanced before the first attack of colic appears. Here, a single attack ushers in the final catastrophe, and is the first sign of rapidly or suddenly developing complete intestinal obstruction. In the intervals between the paroxysms the patients may feel perfectly well and complain of no subjective symptoms whatever. The length of these intervals varies, for in many cases the appearance of an attack of colic is determined by a number of accidental factors, as, for instance, the ingestion of some article of diet, which, after entering the intestine,

mechanically obstructs the passage of the intestinal contents through the stenosed bowel. The following sequence of events is quite common : First, a number of short paroxysms appear for one or several days ; then there is an interval of quiescence lasting for several weeks or months, then again a number of attacks occur in the course of a short time, and finally occlusion of the bowel or other symptoms supervene.

A description of these attacks of colic naturally leads to the consideration of a second very important symptom of carcinoma of the large intestine—namely, constipation. The great majority of patients suffering from this disease are constipated. In many cases—I may say in the majority of instances—constipation is the first sign of this mysterious, insidious disease. In the early stages the constipated condition of the bowels is either overlooked or neglected ; finally, however, constipation becomes so obstinate that the patients cannot overlook it. Sometimes ten, twenty, forty, or even, as in one observation, eighty-eight days may pass before a copious evacuation of the bowel occurs, either spontaneously or with the aid of artificial means. In other cases again constipation persists for very long periods of time, and no evacuation whatever occurs up to the death of the patient. During these prolonged periods the most minute quantities of masses of fecal matter are passed. This condition usually produces the well-known signs of chronic constipation, namely, a feeling of tension and of fulness in the abdomen, failure of the appetite, and occasionally circumscribed spontaneous pain in certain portions of the bowel. Finally the attacks of colic that I have described appear unless a copious and sufficient evacuation of the bowel is brought about by artificial means or unless spontaneous diarrhea occurs and causes the evacuation. This spontaneous diarrhea is produced by the irritation exerted on the intestinal wall by the stagnating bowel contents. As soon as an attack of colic appears, the condition should be recognized and the evacuation of the bowel promoted by all the means at our disposal. If this is not done, or if, in unrecognized cases, spontaneous diarrhea does not occur, the condition of constipation becomes more and more pronounced, until finally occlusion of the bowel supervenes. While the occlusion is in process of formation, a clinical picture is presented which has been described as the slow development of occlusion of the intestine. In individuals who pay no attention to the care of their body, and who consequently might overlook the obstinate constipation that they are suffering from, these symptoms of occlusion may be the first signs that the patients themselves notice, and may for the first time call attention to the possible existence of carcinoma of the intestine. Careful inquiry into the history of such cases will, in the great majority of instances, show that the subjects had been suffering from constipation for a long time before. These points have all been dealt with at great length in the section on Enterostenosis, and in order to avoid repetition, the reader should refer to this section for all the details and for a careful analysis of the various factors which play a part in the production of this lesion.

In some instances the condition of constipation is relieved after a time and the permeability of the intestine is restored. This is a very misleading symptom, for it creates the impression that the patient is improving. As a matter of fact, the lumen of the bowel can become patent only when the carcinoma becomes smaller as the result of ulceration. If the stools are carefully examined every day, evidence of a process of this kind can usually be discovered, for whenever a carcinoma of the intestine ulcerates, blood and pus appear in the dejecta.

In isolated cases the patients may pass diarrheic stools for long periods of time—sometimes even for many weeks. This diarrhea is due to a catarrhal condition of the bowel. Pronounced degrees of inflammation of the mucous lining of the intestine are not rare in carcinoma of the bowel. The appearance of diarrhea may be regarded merely as an episode in the course of carcinoma of the intestine. It has been stated that in some instances of carcinoma of the large intestine there is diarrhea from the beginning to the end of the disease; personally I have never seen a case of this disease in which constipation was completely absent and diarrhea was present instead. At the same time I do not feel justified in stating that such a condition is impossible (compare the section on Carcinoma of the Rectum).

The constitution, form, and color of the stools vary greatly, and are dependent on a great variety of different factors; the same applies to the abnormal material that is occasionally present in the stools. In some cases of carcinoma of the large intestine the stools may be perfectly normal. In other cases, again, the stools show all the features characteristic of stenosis of the intestine—that is, the dejecta resemble sheep's dung, or are flattened, ribbon-shaped, or pencil-shaped. (For the details the reader should refer to the section on Enterostenosis.) When dejecta of these abnormal forms are passed, it may, with certain reservations, be assumed that narrowing of the bowel lumen has occurred, but these data do not, of course, prove that the stenosis is due to a carcinomatous growth of the intestine.

The presence of mucus in the feces is without any significance, being merely the expression of some catarrhal lesion of the intestinal mucous membrane. The presence of pus in the stools is much more important. In carcinoma of the intestine the amount of pus passed is, as a rule, not very considerable. In some cases of carcinoma of the intestine, of course, pus may be derived from other sources—for instance, from an abscess cavity opening into the lumen of the intestine. Stress need hardly be laid on the fact that pus appears in the stools only when the neoplasm is ulcerated, and that it never appears when the neoplasm remains intact and firm. The absence of pus *per se* does not militate against carcinoma in any sense. Conversely, the presence of pus never positively proves the existence of a carcinoma, for the only deduction to be drawn from the appearance of pus in the feces is that an ulcerative process is going on somewhere in the intestine. When ulceration of a carcinomatous growth of the intestine is suspected in any given

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case, the discovery of pus in the feces may be considered valuable corroborative evidence.

All that has been said about pus in the feces applies with equal force to the appearance of blood in the dejecta, for both blood and pus are derived from essentially the same source. There is this difference, however, that pus is secreted from the surface of a neoplasm only when it is ulcerated, whereas blood may be derived from the surface of a neoplasm without there being any ulceration. Blood may ooze from the surface of small carcinomata, but only small quantities appear in the feces, and, moreover, depend on some mechanical irritant. The most frequent cause of bleeding of this kind is the passage of very hard fecal masses through a portion of the intestine that is stenosed from the projection of a carcinoma into its lumen. When this occurs, small fissures and tears (rhagades) are produced on the surface of the tumor, and in this way some blood becomes mixed with the column of fecal material that is passing through the stenotic area. It is well to remember, however, that blood may be absent from the stools throughout the whole course of a carcinoma of the intestine. [In 271 cases it was present in 58, or 1 in 4 or 5 (R. de Bovis[1]).—Ed.] In some cases blood appears in the stools for a long period of time, or at least for a number of days in succession. I remember a patient who was suffering from a carcinoma of the sigmoid flexure and who for two years passed small quantities of blood in the stools almost daily. The quantity of blood lost in this way varies greatly. In general the amount is very small, while violent hemorrhages that are repeated must be considered very exceptional. In the latter instance pure blood unmixed with feces may be expelled from the bowel. In the former case the blood is always mixed with fecal matter and pus. In a patient with some of the symptoms of stenosis of the bowel the appearance of blood, in the same way as the appearance of pus, is in favor of the suspicion that the stenosis is of carcinomatous origin.

The appearance of blood and of mucopurulent or purulent masses in the stools is particularly important in the diagnosis of carcinoma of the large intestine, since we know from extended observation that blood, pus, and mucus appear in the stools only in two conditions—namely, carcinoma of the large intestine and dysenteric ulceration of the large intestine. This statement is true, at least when pus and blood are passed in the manner described in the preceding paragraph. I do not remember ever having seen exactly the same conditions in regard to the passage of blood and pus in other ulcerative processes of the bowel. As dysentery can usually be diagnosed without great difficulty, the great importance of the study of the dejecta in carcinoma of the bowel becomes apparent, for the appearance of blood and pus must be considered one of the most important features of the symptomatology of intestinal carcinomata. Exceptionally some confusion may arise in the presence of one other condition besides carcinoma or dysentery—namely, when an abscess in the neighborhood opens into the bowel, for in this form

[1] R. de Bovis, *Rev. de Chirurg.*, 1900, vol. xxi.

of abscess very similar dejecta, consisting of a mixture of blood and pus, are occasionally passed. In this affection, however, the amount of pus passed is, as a rule, so much greater than the amount of blood that suspicion is immediately directed to the existence of an abscess opening into the intestinal lumen.

In cases in which sanious or gangrenous disintegration of the intestinal carcinoma occurs, the odor of the dejecta becomes quite characteristic of gangrenous material. In very rare instances small particles of neoplastic tissue are found in the stools; such an occurrence, of course, is of fundamental importance and immediately clinches the diagnosis. The appearance of shreds of new growth in the stools is relatively very rare; in some instances, however, shreds as large as a nut have been discovered.

The most important direct symptom of carcinoma of the intestine, and the one that places the diagnosis of carcinoma on a solid basis, is the presence of a tumor. If a tumor can be palpated in a case in which other symptoms raise the suspicion of carcinoma of the intestine, the diagnosis is very much strengthened.

Let me emphasize that the presence of a palpable tumor is not necessarily an obligatory symptom of carcinoma of the intestine nor a symptom that is always present. I have already explained in the paragraphs on the pathologic anatomy of carcinoma of the intestine that in many forms of intestinal cancer no true tumor formation occurs. This is seen, for instance, in the small circular form of carcinoma of the intestine which slowly produces gradually progressive stenosis of the bowel lumen without ever leading to the formation of a tumor that could be palpated. On the other hand, a tumor may actually be present, but be situated in a portion of the bowel where it is not accessible to palpation—for instance, because it is covered by some other organ, as the spleen, the liver, or other portions of the intestine.

In the great majority of cases, however, careful palpation of the abdomen will reveal the presence of a tumor in carcinoma of the intestine. The size of the swelling may vary greatly: it may be no larger than a nut, and it may be as large as a child's head. If the abdominal walls of the patient are very thin, the tumor may produce prominence of certain portions of the abdomen and in this way become visible externally. As a rule, carcinomata of the intestine are solid and very hard, like cartilage. The form and outline of these tumors also vary greatly: they may be either extremely irregular in outline, or they may be roundish or elongated. When carcinoma infiltrates large areas of the intestinal wall, it may convert the intestine into rigid cylinders of varying length. In these cases the intestine on palpation gives the impression of a solid, thick cord. The surface of carcinomata of the intestine may be smooth, nodular, or rough and covered with ridges. In general these tumors are moderately tender to pressure—in some instances, in fact, they may be extremely painful. Conversely, however, it must be remembered that occasionally these carcinomata are quite insensitive even on marked pressure. The complete absence of

pain in these tumors, even on pressure, is very remarkable and must be remembered. If the growth is quite small, it is naturally incapable of modifying the character of the percussion-note in any way, so that percussion over the tumor merely elicits a loud intestinal note. If the tumor is very large and massive, on the other hand, it produces dulness. No great importance, however, can be attached to the percutory changes produced by carcinoma of the intestine.

The tumors produced by a carcinoma of the intestine are distinguished from all other forms of abdominal tumors by their great motility. This peculiarity is by some considered to be a characteristic feature of this form of neoplasm, both in carcinoma of the small intestine and also in carcinoma of the large intestine. All the various possible factors that can play a part in the production of abnormal motility of tumors of the abdomen are present in the case of carcinoma of the intestine. This form of tumor can be moved—(a) By pressure exercised from without—as, for instance, the hand of the medical man ; (b) by pressure exerted from within (weight) ; (c) by the peristaltic action of the intestine ; (d) by the respiratory movements of the diaphragm.

The most important of these is the fact that carcinomata of the intestine can be moved by the palpating hand of the physician. The rule is generally accepted that carcinomata of the sigmoid flexure and of the transverse colon possess the greatest passive motility of all carcinomata of the large intestine. This is explained by the great length of the mesentery belonging to these parts of the bowel ; but carcinomata of the cecum and of the ascending and the descending colon can also, to a certain degree at least, be moved to and fro with the hand pressed into the abdomen. I have repeatedly examined patients with carcinomata of the cecum and of the ileocecal valve in whom the diagnosis of carcinoma in these situations was corroborated by operation or by postmortem examination, and in whom the abdominal tumor was distinctly movable and could be pushed to and fro without difficulty.

From a clinical point of view the changes of position of carcinomata of the intestine which can be produced by pressure from without are exceedingly important. As indicated above, this pressure from within is practically the same as the tendency to produce displacement manifested by the weight of the growth itself. Displacement of the tumor inside the abdomen, however, is rarely due to the weight of the tumor itself. As a rule, large quantities of fecal matter accumulate above the tumor, distend the intestine, make it very much heavier than normal, and in this way produce displacement of the affected portion of the bowel. If a carcinoma of the intestine, after becoming displaced in this way, forms adhesions with neighboring organs, the physical signs may be exceedingly confusing, and many errors in diagnosis, especially as to the anatomic starting-point of the abdominal tumor, may arise. It may happen, for instance, that a carcinomatous sigmoid flexure becomes fixed and adherent in the right side of the abdomen in the region of the cecum, or that a tumor of the transverse or the ascending

colon is found tightly fixed and adherent to the upper inlet of the pelvis.

As already mentioned, another factor may produce changes in the position of carcinomata of the intestine—namely, the peristaltic movements of the bowel itself. This factor, however, is, comparatively speaking, of subordinate importance, for peristaltic contractions do not lead to any real displacement of a carcinomatous intestine, though this may appear to be the case from the fact that the affected coil of intestine becomes covered by others. This condition is usually recognized by the following physical sign—viz., the tumor disappears for a time from a certain situation, and then, after a longer or a shorter interval, reappears in its original position. This peculiar sign, however, is not by any means typical or pathognomonic of tumor of the intestine, since all other abdominal tumors may produce the same symptom. The last factor which is capable of altering the position of a carcinoma is the excursions of the diaphragm. Respiratory motility is seen only in cases of carcinoma of the transverse colon or in those parts of the ascending and descending colon which are in the neighborhood of the diaphragm. In cases in which a carcinoma of the intestine is adherent to the liver, the spleen, or the stomach, the tumor may, of course, share in the respiratory movements transmitted to these organs by the diaphragm.

It need hardly be specially mentioned that motility of any kind is immediately stopped and prevented when the neoplasm becomes adherent to the abdominal wall or to other organs of the abdominal cavity which are fixed by peritoneal adhesions. In other words, immobility of an abdominal tumor of unknown character does not necessarily prove that the tumor does not belong to the intestine.

It is very important to remember that as soon as the lumen of the intestine becomes stenosed by a carcinoma, large amounts of fecal matter accumulate above the neoplasm; in this way so-called fecal tumors are produced which may lead to most striking errors in diagnosis, particularly in the diagnosis of the shape and the size of the tumor itself. It is often surprising to find at the autopsy how thoroughly mistaken the opinions formed during life on these points really were. It is very important to emphasize the importance of carefully excluding fecal tumors, for in many instances the indications for operative interference depend on the estimate formed of the shape and the size of the neoplasm to be removed. It must be confessed that it is very difficult to avoid errors, particularly in estimating the size of a carcinoma of the intestine. This is due to the fact that the dried fecal accumulations in the portion of the intestine above the tumor may assume the same degree of hardness and impart the same sensation of resistance as the tumor itself, so that on palpation the fecal tumor and the neoplasm together feel like a uniform, inseparable mass. In still other cases the carcinoma itself does not form any pronounced swelling or tumor, but merely consists of a small circular growth. In cases of this kind fecal tumors may be formed above the constricted portion of the bowel, so that what

is regarded as the carcinoma is in reality nothing but fecal material. Finally, no neoplasm at all may be present, and we may commit the serious diagnostic error of diagnosing a simple accumulation of fecal matter as a carcinoma of the intestine.

This point is of such clinical importance that it is necessary to consider in detail the differential diagnosis. In many cases—and I may say in my experience in the majority of cases—there is no doubt about the diagnosis of the carcinoma, so that the only question is to decide whether or not a fecal tumor is present in addition. If all the data of each individual case are carefully analyzed, a careful history taken, and the symptoms reviewed as a whole, this fundamental question can usually be answered without difficulty. The only point, then, that remains is to decide how much of the tumor is formed by the carcinoma and how much by fecal material. In rare instances it is possible to discover certain differences between the resistance of the carcinomatous and of the fecal parts of the tumor, for, generally speaking, the fecal portion of the tumor is less firm and more doughy, and can, in fact, be kneaded a little. Our most valuable guide to diagnosis, however, is the administration of internal laxatives (preferably castor oil or salts), and at the same time copious water or oil enemata. It is frequently, though not always, possible to clear up the situation by these measures.

In cases in which it must be decided whether there is a neoplasm at all or only a fecal tumor the question is still more complicated. I am convinced that cases of this kind occasionally occur, although, as a general rule, they may be considered rare. Attention need only be called to the fact that blood and pus may be completely absent in the stools in carcinoma ; that a fecal tumor, owing to the development of secondary peritonitis, may occasionally become painful, both spontaneously and on pressure ; that a large number of accidental complications may produce a certain degree of cachexia in the course of the development of a fecal tumor ; and that the same factors which are concerned in producing motility of a carcinoma of the intestine play an active part in altering the position of a fecal tumor of the intestine ; that, finally, a fecal tumor may lead to narrowing and occlusion of the intestinal lumen in the same way as a carcinoma of the bowel. In cases of this kind recourse will naturally be had to the different adjuvants to the diagnosis mentioned in the preceding paragraph—namely, large irrigations and laxatives. Leube has called attention to the fact, and I agree with his view, that we must not allow ourselves to be misled by the fact that some of the fecal material may remain behind even after the evacuation of the main mass. It often occurs that some portions of the fecal tumor are so tightly adherent to the intestine and so tenaciously held in the haustra of the colon that only the central portions of the mass are washed away by irrigation or removed by the administration of laxatives. In this way a central passage is created, while, at the same time, the peripheral portions of the tumor, so to say, remain behind. Another important point in the differential diagnosis between fecal tumors and carcinomata of

the intestine is that fecal tumors are occasionally multiple, whereas it is very rare to find more than one carcinomatous tumor ; in other words, multiple fecal tumors are not rare, whereas multiple carcinomata of the intestine may be considered exceedingly rare. Sometimes the history of the case will aid us in arriving at a decision. If the patient has always had perfectly regular evacuations of the bowel to within a few months, there are hardly ever sufficient grounds for suspecting the presence of simple and genuine fecal tumors, for they develop only in the subjects of long-standing, persistent, and habitual constipation (compare also what has been said on this subject in Section III.).

Occasionally the diagnosis is practically impossible, and it may be quite out of the question to form an accurate idea as to the nature of the abdominal condition. As an instance in point I may refer to a case in which a single fecal tumor develops above an innocent tumor or a stricture of the intestine. A single tumor is expressly specified, because, under the same circumstances, a number of fecal tumors may develop. In the latter case, as pointed out above, the diagnosis is comparatively simple. Here presumptive evidence, at all events, is in favor of fecal tumor, particularly if the patient has for some time had habitual constipation. But when there is a single fecal tumor above a benign stricture in which the tumor is hard and nodular and is, in addition, exquisitely tender from local peritonitis, the diagnosis becomes extremely doubtful and difficult ; for, owing to the tenderness of the tumor, careful palpation is quite impossible, and no help can be obtained from this important means of differential diagnosis. In addition, careful study of the history may show that the patient has no recollection of anything bearing on the existence of some former intestinal disorder, and that there is no reason to suspect the presence of a stricture of the intestine. Under these conditions no conclusions can be drawn as regards the possible presence of sequelæ of such a stricture. The matter may become still more complicated if the patient is very much reduced and pulled down, owing to the presence of some other disease elsewhere, such as suppuration. It will readily be seen that under these conditions, as stated above, a diagnosis is quite impossible. I assume that every medical man who has had much experience in this line of practice has met with cases of this kind and has committed serious diagnostic errors.

I cannot refrain from reporting the following case in detail, as it illustrates in an excellent manner the difficulties which may occasionally arise in making a differential diagnosis between neoplasm of the intestine and fecal tumor.

Marie Sch., aged forty-one, a washerwoman by occupation. No history of any hereditary taint could be elicited. The patient never suffered from any intestinal affection, and never developed any cough. She was in excellent health until May, 1895. About this time the present illness began.

The disease was ushered in by constipation. The patient had, up to this time, had normal evacuations of the bowels almost daily, but after May of 1895 she was obliged to have recourse to the injection of infusions of senna to get her bowels open. In June she began to suffer from paroxysms of pain, which were colicky in character and situated in the right lower abdominal region. During an attack of colic there was much distention, and loud gurgling sounds were heard

in the intestine.   According to the patient's own description, visible peristaltic movements of the bowel could be observed on the abdominal wall at the same time.   During these paroxysms she frequently suffered from attacks of vomiting. The vomited material consisted either of food eaten a short time before or of mucous masses with an acid taste.   As soon as vomiting occurred, the pain was relieved.   When the bowels were evacuated, the pain disappeared, and at the same time the rolling and the gurgling noises in the abdomen became more distinctly audible and louder.   The stools passed after taking a laxative were always thin and liquid.

At the beginning of July the patient came to the out-patient clinic, and at that time her attention was called to a swelling in the right iliac fossa.   She states that this swelling has persisted ever since and has gradually increased in size. She also volunteers the information that the swelling is smaller immediately after defecation and is larger as soon as she allows herself to remain constipated for some time.   She states that since the month of August there has been a daily rise of temperature in the evening, and that the attacks of colic are more frequent and last longer.   The condition of constipation has been obstinate ever since this time. If she takes larger doses of infusion of senna, the stools passed (very soon afterward) contain remnants of food which she believes were eaten weeks or even months before.   Since the middle of October there has been persistent pain in the right iliac fossa while walking, or even when lying on her back.   Since August, moreover, the appetite has greatly diminished and she has been very weak and has become exceedingly emaciated.

Status præsens (at the end of October).—The patient was placed on the right side in the semidorsal position, and instructed to flex the right thigh on the abdomen and the leg on the thigh.   The woman is of medium height, with poor muscular development, and is exceedingly emaciated.   The facial expression indicates great suffering.   The color of the skin and of the visible mucous membrane is very pale.   In the morning the temperature is subfebrile—98.6° F. (37° C.) ; the maximum temperature is reached in the evening—101.2° F. (38.5° C.).

On inspection it is found that the upper portion of the abdomen forms an inclined plane extending from the lower thoracic aperture toward the umbilicus, and that this portion of the abdomen is quite flat.   The lower portion of the abdomen from the umbilicus downward rises to form a low ridge.   The pole of this ridge is situated about two fingers below the umbilicus.   Below the umbilicus, moreover, the surface of the abdomen appears to be uneven.   On careful inspection fairly active peristaltic movements of the bowel can be seen through the thin abdominal walls.   Cylindric portions of the intestine can be seen to protrude, while between these cylindric portions distinct valleys can be observed.   The right lower quadrant of the abdomen appears filled out and more prominent than the left lower quadrant.   On the left side the anterior superior spine of the ilium can be distinctly seen to protrude, whereas on the right side this bony point is obliterated.   The left flank forms a straight line; the right flank, a convex line, the apex of which is situated at a point lying half-way between the level of the umbilicus and the anterior superior spine of the ilium ; in other words, there is on the right side a bulging that extends backward as far as the scapular line.

In front the skin can everywhere be picked up in folds.   In the right flank, from the lower margin of the ninth rib to a point 3 cm. beyond the crest of the ilium in one direction, and from the scapular line to the anterior axillary line in the other, the skin is edematous, but not reddened.   The temperature in this area is higher than in the corresponding area on the left side.

On the left side the abdomen is moderately tense.   On the right side, two fingers above Poupart's ligament in the nipple-line, a hard tumor can be felt. At this point the dull rounded pole of the swelling can be felt.   On deep inspiration the tumor seems to move a little backward.   The mesial margin of this tumor was found to form a convex line extending from the pole toward the median line ; immediately below the umbilicus it approaches the median line of the body very closely—that is, within two fingers-breadths.   From this point the margin extends backward and then horizontally outward toward the anterior end of the twelfth rib.   In the nipple-line, finally, almost at the height of the umbilicus, the outline of its edge is lost in a diffuse tumor mass.   The lower margin can be followed along Poupart's ligament as far as the anterior superior spine of the ilium.   Posteriorly the outline of the tumor cannot be felt, as the growth is diffuse and can

be felt only indistinctly. The margin of the tumor is rounded throughout, so that the growth seems to be spheric in form. The surface of the swelling is covered with small nodules.

It was found that the tumor could be moved to and fro in the abdomen, and was certainly not anchored or fixed. The swelling was tender on pressure; palpation in the right flank, in the region of the main area of edema, and pressure on the crest of the ilium, were particularly painful.

Examination of the thorax showed the following facts: The percussion-note over the apex of the right lung, both anteriorly and posteriorly, was duller and of higher pitch than over the left lung. The sound, however, was not in the least tympanitic; otherwise the percussion-note was normal throughout the chest. On auscultation the inspiratory sounds at the apex of the right lung were indistinct. Expiration was prolonged and blowing. At the height of inspiration very slight, fine, resonant râles could be heard. Throughout the rest of the lung there was normal vesicular breathing and no râles. There were no cough and no expectoration. The heart-sounds were clear and distinct, and the second pulmonary sound slightly accentuated.

Nothing abnormal was found in any of the other organs. On analysis, the urine was somewhat darker in color than normal, perfectly clear, of an acid reaction, and contained neither albumin nor sugar, but abundant quantities of acetone and diacetic acid and a large amount of indican.

The analysis of the stools showed the following: The dejecta, when presented for examination, were greatly diluted with urine, so that they constituted a thin, light-yellow liquid with a feculent odor. In it were suspended a few light-colored particles that looked very much like coagulated milk. There was a feeble bile-pigment reaction. Weber's blood test was negative. No mucus was seen by the naked eye. Microscopic examination revealed the presence of isolated droplets of fat and of a small number of epithelial cells that were partially degenerated, some in the "flaky" form of degeneration that I have described, some in a state of fatty degeneration. Enormous numbers of bacteria and a great quantity of detritus were also seen. No tumor particles were discovered; tubercle bacilli were also absent, or at least could not be demonstrated.

Course of the Disease.—While in the hospital, the woman continued to lose strength rapidly. She suffered much pain in the right abdominal region that was intensely exacerbated on the slightest movement and on the slightest touch. The patient became emaciated to an extreme degree. Every day she passed from three to six catarrhal stools which came from the small intestine, the dejecta presenting the appearance described above. The quantity of acetone and indican in the urine remained large; the temperature was subfebrile—somewhere between 98° and 101° F. On three occasions there were paroxysms of colicky pain with meteorism and energetic peristalsis. These attacks stopped as soon as flatus and stools were passed. The tumor remained unchanged; no changes, moreover, were seen in the edematous condition of the skin over the tumor. Bronchial breathing and scanty resonant râles persisted in the apex of the right lung, but did not increase or decrease in intensity. There was no expectoration at any time.

On November 9th the edematous skin above the right crest of the ilium began to turn slightly red, and on November 12th indistinct fluctuation could be made out at a point 7 cm. above the crest of the ilium and 12 cm. to the right of the median line. In the course of the next few days this fluctuation became more and more distinct, so that finally, on November 15th, the patient was transferred to the surgical clinic.

On November 17th the patient was put under ether and the abscess opened. A considerable quantity of brownish-green pus was evacuated that contained a number of shreds of tissue and emitted a gangrenous odor.

The patient continued to lose strength after the operation, and died on November 18th.

On November 19th an autopsy was performed and the following conditions were found: "A stricture was discovered in the first portion of the ascending colon which was so extreme that nothing could pass through the lumen of the stenotic part of the bowel excepting a sound. The stenosis was produced by a constricting cicatrix that had developed on the base of a tuberculous ulceration of the colon. Above the stenosis the ileum was found dilated and the walls of this portion of the bowel hypertrophied. In the lumen of the lowest portion of

the ileum a large quantity of different kinds of fruit-seeds and pips were found (cherry-stones, plum-, and a very large number of grape-seeds). This material formed a conglomerated mass which had simulated a hard tumor. The mucous membrane in this portion of the bowel was also ulcerated (decubital ulcers). At the base of one of these decubital ulcers a perforation was found leading backward into a large abscess cavity. Chronic tuberculosis of the apex of the right lung."

Remarks.—In my opinion all the statements elicited in the history of this case, and all the observations on examination of this patient, necessarily forced one to make the diagnosis of carcinoma of the region of the cecum. I was also justified, from the history, in believing that this person, who was forty-one years of age, had always been healthy before, and that the stools had always been regular. It was exceedingly misleading to find that in May, without any recognizable external cause, constipation followed by attacks of colic and visible peristalsis of the small intestine should appear. When it was found, moreover, that the patient's distress was relieved and that the abnormal symptoms stopped completely after the administration of laxatives and the consequent evacuation of the bowel contents, the diagnosis "stenosis of the intestine" was the most probable one. In July, then, as we saw, a tumor developed in the region of the cecum which was very hard and uneven, painful on pressure, could be moved slightly to and fro, became smaller after the administration of laxatives, but never disappeared altogether. In addition there was great emaciation. All these factors indicated the presence of carcinoma in the region of the cecum—in other words, the diagnosis "carcinomatous stricture in the region of the cecum complicated by fecal tumor" seemed justifiable. The symptoms that developed later, namely, the subfebrile temperature, the appearance of circumscribed local edema of the skin, great tenderness on pressure, and pain in the region of this edema toward the flank, all pointed to the development of an abscess. This latter lesion, we were justified in assuming, developed from the lesion in the cecum and may be considered a common sequel of carcinoma of this region.

Owing to the phenomena discovered in the apex of the right lung, the possibility of a tuberculous change in the cecum was naturally considered, even though the patient had never developed a cough and had never expectorated anything; but in view of the fact that intestinal symptoms had not been present before the onset of her disease; in view of the fact, further, that no tubercle bacilli were found in the dejecta; in view, finally, of the general configuration and the whole development of the tumor, the diagnosis of a tuberculous disease of the cecum was given up, chiefly on the grounds that the symptoms could be explained more satisfactorily and naturally by the diagnosis of carcinoma.

Nevertheless, in spite of all these factors and considerations, the autopsy showed a simple stricture of the intestine which had developed on the base of a tuberculous ulcer. All the remainder of the intestine was free from tuberculous lesions of any kind. No vestige of carcinoma was discovered. The hard tumor felt was chiefly composed of a large number of hard stones and seeds of different kinds of fruits that had accumulated above the stricture. This tumor, moreover, was slightly enlarged by the addition of ordinary bowel contents.

A number of other factors may make the diagnosis of carcinoma of the intestine difficult. This applies particularly to tuberculosis of the cecum, further described on page 468, and to circumscribed peritonitic exudates in the ileocecal region. Such masses of exudate may form hard and solid swellings, and may seriously interfere with the onward passage of the bowel contents. They do this either by compressing the bowel from without, by causing kinking of the intestine, or by producing stricture of the bowel lumen by adhesions. Occasionally they may simulate carcinoma, especially by perforating into the intestine, and so leading to the appearance of blood and pus in the stools. In cases of this kind, as a matter of fact, a decision in regard to the true character of the disease may occasionally be utterly impos-

sible unless certain concomitant symptoms—as fever, inflammatory edematous infiltration of the skin—or a careful study of the course and progress of the swelling throw additional light on the subject.

Leube has called attention to the fact that chronic thickening of the sigmoid flexure by certain inflammatory changes may occasionally occur. He calls this condition sigmoiditis chronica, and states that the bowel in this affection forms an elongated mass with the outline of a tumor, so that occasionally the sigmoid flexure, when it is in this condition, may be mistaken for a carcinoma. The only way in which to avoid this mistake in diagnosis is to study each individual case persistently until the question is positively cleared up. It is true that Leube claimed that an experienced clinician would be able to differentiate this form of sigmoiditis chronica from carcinoma of the sigmoid flexure, and I agree with him, for on palpation an inflammatory infiltration of this nature conveys a different feel to the hand than does a carcinoma.

The differential diagnosis in any given case between a foreign body and a neoplasm may present difficulties. At the same time the fact that doubt may arise as to this differential diagnosis points out the proper method of procedure to arrive at a correct decision. Foreign bodies in the intestine which remain latent and do not raise the question of their presence or of the differential diagnosis between them and carcinoma, can, of course, be discovered only by chance. This point is considered in the sections on Foreign Bodies and on Intussusception of the Bowel, to which the reader should refer. An account is given there of the most important diagnostic features in the recognition of this condition. I do not think that this difficulty will often arise, or that a foreign body in the intestine will often suggest the diagnosis of "carcinoma of the bowel."

Another possible source of error is the following: Tumors not arising in the intestine or in its immediate neighborhood may occasionally be confounded with a carcinoma of the intestine. This applies particularly to carcinomata of the pylorus, especially when the stomach is displaced. In a case of this kind I believe that the attention of the medical man will immediately be attracted to the predominance of gastric symptoms, and that in this way a correct diagnosis will be made. If, moreover, from the presence of gastric symptoms, the medical attendant takes the trouble to examine carefully the stomach and its contents with regard to motility, absorptive powers, reaction, etc., there will be less chance of forming an erroneous diagnosis, such as might possibly be arrived at from the discovery of a tumor of the stomach in an abnormal position. A detailed account of the differential diagnosis between carcinoma of the intestine and other abdominal tumors, whether genuine tumors or only conditions simulating tumors, such as floating kidney, wandering spleen, corset liver, etc., is beyond the scope of this article, and would merely tend to confuse the general description that I am giving here. A very short review would be quite inadequate and of little practical value to the medical man, and

an exhaustive account, to be of any value, must include a detailed and full description of the pathology of the various conditions. The latter description, however, will be found in those portions of this work in which the diseases of the different organs are described—viz., the volumes on diseases of the female genitals, the pancreas, the kidneys, the gall-bladder, aneurysms, mesenteric and retroperitoneal glands, and the peritoneum. Any one who has had much experience in this field of medicine will have to confess reluctantly that there are cases in which the most careful examination of the patient, the most conscientious consideration of all the features of the case, and the greatest possible clinical experience may still be insufficient to prevent a wrong diagnosis.

The fundamental features of the clinical picture presented by a carcinoma of the large intestine are the pain, the changes in the character of the stools and in the method of defecation, the tumor, and the cachexia. In individual cases, however, this fundamental syndrome may be modified in many different ways, sometimes to such an extent that the primary disease becomes quite unrecognizable. These alterations of the natural features of a typical case of carcinoma of the intestine depend chiefly on three anatomic sequelæ which may develop in the course of carcinoma.

One of these factors is the formation of metastases in other organs. Whenever metastases are formed, certain series of symptoms appear which are produced by the development of the secondary carcinoma in organs such as the liver, the lungs, etc. The symptoms of secondary carcinoma of these different organs will be described at length in the volumes on these organs. The subject of secondary carcinomata in the peritoneum will be considered at length in the section on Peritonitis Carcinomatosa.

The second factor is the direct extension of the growth into neighboring organs. Abnormal adhesions are formed which unite the different organs so firmly that they cannot be separated. When a hollow organ becomes adherent to an ulcerating carcinoma of the intestine, perforation may occur, and a direct communication between the two may thus be established. A study of the records of cases shows that a great variety of communications of this kind can be formed. It would be impossible to describe all the forms that have been observed, and the account must, therefore, be restricted to a few examples which are of special importance in general practice, first, from their comparative frequency, and in the second place, because they produce more or less typical and special clinical pictures.

Perforation may occur from the transverse colon into the stomach (it is well known that the reverse also occurs). In this way an abnormal communication is formed between the transverse colon and the stomach, or a gastrocolic fistula. This condition can occasionally be diagnosed with great certainty, especially in the presence of two symptoms at the same time—viz., lientery and feculent vomiting. One of these symptoms alone may be very misleading, even when the diagnosis

of carcinoma of the stomach or carcinoma of the colon is beyond question. I have repeatedly observed vomiting of material that emitted a feculent odor in simple carcinoma of the stomach. In these cases the peculiar odor was due to putrefactive decomposition of the stomach-contents. On the other hand, lientery may only be apparent—that is, it may happen that undigested food passes very rapidly through the intestine and appears in the stools in this condition. If both symptoms, however, appear together, I think, as already stated, that this coincidence is pathognomonic of the existence of a communication between the stomach and the colon, for I do not know of any other conditions under which it can occur. Another useful point in the diagnosis of this abnormal communication is the following : when colored fluids taken by the mouth are rapidly expelled from the bowel without being decolorized or discolored. Another valuable means of diagnosis which is of use in very rare cases is to force air into the rectum. If the stomach becomes inflated sooner than the ascending colon, it may be assumed that an abnormal communication exists between the colon and the stomach.

The stomach is not the only organ with which the colon may form abnormal communications in cases of carcinoma. A cancer of the colon may perforate, for instance, into other loops of intestine and in this way lead to fistulous communications. It is a remarkable fact that such communications are more frequently formed between the carcinomatous portion of the colon and some other part of the large intestine than between the carcinomatous portion of the colon and some loop of the small intestine. Abnormal communications, for instance, have been found between the cecum and the ascending colon, between the cecum and the transverse colon, between the cecum and the sigmoid flexure, between the sigmoid flexure and the transverse colon, or between the sigmoid flexure and the descending colon. It can readily be understood how these abnormal communications between parts of the intestine that are normally far apart in the abdominal cavity can be brought about in carcinoma of the bowel. It has been shown that the weight of the tumor can cause displacement and locomotion of the portion of the bowel in which it is developing, or that the masses of feces which stagnate and accumulate above a carcinomatous stenosis of the bowel may make the affected loops so heavy that they become dislocated and in this way are brought in close proximity to distant portions of the bowel. It is, of course, very important to recognize conditions of this character, particularly when operative interference is under consideration. In fact, the whole indication for an operation may depend on the recognition of the true anatomic conditions within the abdomen. Unfortunately, however, it is only in exceptional cases, and then only under the most favorable circumstances, that the physician is able to make a correct diagnosis of these intercommunications for the benefit of the surgeon. The symptoms of the different forms of adhesions and communications mentioned above are not really typical and pathognomonic, either functionally or physically. A number of artificial means of

arriving at this diagnosis have been advised, but personally I regard most of them as of doubtful value. I do not believe, for instance, that it will ever be possible to diagnose a direct communication between the sigmoid flexure and the cecum by inflating the lower bowel with air.

Sometimes an abnormal communication may form between a loop of intestine and the urinary bladder. This condition can, as a rule, be recognized. In carcinoma of the sigmoid flexure communication of the bowel with the bladder is most liable to occur, and I presume that every practitioner who has had much experience in this field of medicine has had to deal with cases of this kind. Other portions of the large intestine, for instance, the transverse colon and the cecum, may, however, also perforate into the bladder in carcinoma. The most important symptom of this condition is the passage of a peculiar urine. The urine in these cases emits a feculent odor and is frequently mixed with fecal material. In extreme cases a certain amount of flatus may even be expelled through the urethra. I have personally had occasion to observe a case of this kind. Whenever feculent matter appears in the urine, or whenever flatus is expelled through the urethra, it is certain that a perforation into the bladder has occurred, and that an abnormal communication exists between the colon and the bladder. Very soon after the perforation the character of the urine becomes catarrhal. Another way in which to diagnose this condition would be to look for urine in the feces. As a matter of fact, however, it is a very difficult matter to recognize the admixture of urine with the stools. In cases of abnormal communication between the colon and the bladder both these possibilities must be considered—viz., that fecal material may appear in the urine or urine may appear in the feces. The determining factor is the conformation of the perforative opening. A valuable means of establishing the diagnosis on a solid basis is to inject carmin or some indifferent stain into the rectum or into the bladder, and then to see whether this colored material appears in the urine or in the feces.

Another class of cases of carcinoma of the colon is very important clinically—namely, those in which the carcinoma leads to the development of local peritonitis. Cases of this kind are by no means rare. The direct consequences of local peritonitis following carcinoma of the intestine are the formation of adhesions with the abdominal parietes, the development of abscesses in the abdominal walls, occasionally gangrene of the abdominal walls, and, finally, perforation through the skin. When this sequence of events occurs, a so-called colic fistula develops. The favorite situations for perforation of a colic fistula through the skin are the iliac fossæ and the inguinal regions; perforation may, however, occur through any other portion of the anterior abdominal wall.

Adhesions with the uterus, the tubes, and the ovaries produce no direct clinical symptoms. Occasionally such conditions are recognized in the course of a gynecologic examination. In a few rare instances adhesions have been known to form with the gall-bladder, but it is very doubtful whether a condition of this kind can ever be diagnosed.

The third factors that may complicate carcinoma of the colon and obscure the clinical picture presented by the primary disease are certain anatomic processes that develop in the peritoneum. Carcinomatous peritonitis has been referred to in a preceding paragraph. Whenever carcinomatosis of the peritoneum develops, a large exudate is poured into the abdominal cavity. This naturally renders the physical examination of the abdomen difficult, and makes it almost impossible to discover the presence of a tumor and to determine its exact location, motility, feel, etc., so that the diagnosis both of primary carcinoma of the colon and of the exact causes responsible for the peritoneal exudate are rendered difficult or impossible.

An entirely different clinical picture is presented when the carcinoma, after having formed adhesions with other organs, perforates into the sacculated space formed by these adhesions, or when it perforates into the subserous cellular tissues in the neighborhood. Whenever this occurs, intraperitoneal and subperitoneal abscesses are formed. These lesions and the clinical symptoms they produce will be described at length in the section on Diseases of the Peritoneum.

Perforation may, of course, also occur into the general peritoneal cavity. This accident always leads to absolutely fatal peritonitis, a condition which will be described in another portion of this volume. (See the section on Perforation of the Bowel and Peritonitis Perforativa.)

[From the symptoms, Boas [1] describes four clinical types of carcinoma of the colon : (1) In which there are no local symptoms, the diagnosis being made from the cachexia ; (2) where there are obscure local symptoms ; (3) where there are marked local symptoms, as colic and diarrhea ; (4) where sudden intestinal obstruction occurs.—ED.]

CARCINOMA OF THE RECTUM.

The symptoms of carcinoma of the rectum are essentially the same as those of carcinoma of the colon. This is quite natural, for the only differences that can exist between these two forms of carcinoma of the bowel are directly due to the anatomic peculiarities of the portion of the intestine involved by the carcinoma. Certain peculiarities in the course of the rectum and its construction lead to a few deviations from the clinical picture presented by carcinoma of the large intestine in general. One reason why certain typical signs are found in carcinoma of the rectum alone is that this portion of the bowel is readily accessible to palpation and inspection, so that certain conditions can easily be recognized which are difficult to detect in carcinoma of other portions of the bowel. The diagnosis of rectal carcinoma, moreover, is very positive and very easy. In the great majority of cases a carcinoma of the rectum can be palpated without difficulty with the fingers, and thus enables us, in the great majority of cases, to make an absolutely certain diagnosis. Unfortunately, many medical practitioners do not examine the rectum even in cases in which a number of symptoms point to disease of this portion of the bowel. This applies particularly to early

[1] Boas, *Deutsch. med. Wochenschr.*, 1900.

stages of this disease. If practitioners were more careful in this respect, the diagnosis of incipient rectal carcinoma would be more frequently made.

[Although ballooning of the rectum is not diagnostic of stricture of the lower part of the colon, it is often found in this connection (Treves).[1] The rectum is greatly dilated as the result of paralysis and not from distention with flatus ; the surface is smooth and gives the impression of having rigid walls.—ED.]

In rectal carcinoma pain is usually much more pronounced than in carcinoma of the colon. At the beginning of the disease—that is, for instance, when isolated carcinomatous nodules develop slowly in the region of the ampulla—the condition may not give rise to much pain for some time, or may even not be painful at all. In the majority of cases, however, there are both local pain and a certain amount of radiating pain, so that the patients complain of much suffering in the region of the sacrum, the lower portions of the back, in the bladder, the genitals, and the sciatic nerve. Frequently, too, there is a desire to urinate. When the growth is situated in the anal portion of the rectum, the pain is always worse during defecation. In these cases violent tenesmus is almost constant. A large carcinomatous mass in the rectum produces stenosis and finally ulcerates and may become a source of greatest torture to the patient. In these cases the passage of fecal matter through the anus is so excruciatingly painful that the patients try to retain the bowel contents as long as possible. Finally, however, under most horrible torture, a mass of fecal matter, mucus, blood, pus, or sanious material has to be evacuated.

In carcinoma of the rectum there is usually constipation. This may be due to two causes : in the first place, it may be more or less voluntary, owing to the great pain that is experienced on defecation ; on the other hand, it may be involuntary, and the result of some degree of stenosis which is nearly always present. Everything that has been said in regard to stenosis of the bowel in the description of carcinoma of the colon applies with equal force to stenosis of the bowel in carcinoma of the rectum. The higher up the site of the carcinoma in the rectum, the more does the clinical picture presented resemble that of carcinoma of the sigmoid flexure. (For some of the special points the reader should refer to the section on Strictures of the Rectum.) Occasionally the carcinoma sloughs ; when the growth is close to the anus, insufficiency of the sphincter ani follows, either as a result of direct destruction of the sphincter muscle or from paralysis of the circular fibers of this muscle. Whenever this occurs, the intestinal contents, mixed with blood and pus, are passed involuntarily, as the patients lose control over the act of defecation. The sanious material which is frequently present in the dejecta in these cases may, in its turn, produce inflammatory irritation of the skin around the orifice of the anus. Special stress must be laid on the fact that diarrhea may occasionally supervene in carcinoma of the rectum in the same way—*i. e.*, as an

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[1] Treves, *Intestinal Obstruction*, ed. 1899.

intercurrent phenomenon—as in carcinoma of the colon. I remember a number of patients who stated expressly that the disease began with attacks of violent diarrhea. I once had a case under observation in which diarrhea existed throughout the whole course of the disease. This diarrhea began in August, 1892, after the patient, who had been apparently perfectly healthy up to this time, had eaten a turnip and taken some beer at the same time. The onset of this diarrhea was perfectly sudden ; the patient was obliged to defecate five or six times a day ; after two weeks blood appeared in the stools, and gradually the number of motions containing blood increased until, finally, about 20 very liquid stools were passed daily. At the same time there was the most excruciating tenesmus. This diarrhea gradually subsided and became intermittent, so that every two or three weeks he would pass a semi-solid mass of material with the most violent pain. I examined this patient in June, 1893, and found an enormous fissured carcinomatous nodule in the rectum, situated 4 cm. above the anus. Leube, so far as I know, was the first to call particular attention to another phenomenon occasionally observed in carcinoma of the rectum, which is of sufficient clinical importance to be specially mentioned here. He pointed out that piles very frequently develop in carcinoma of the rectum, and insisted on the importance of remembering that they may appear very early in the course of this disease. This fact is specially important, because the appearance of hemorrhoids necessarily calls the attention of the medical man to the presence of something being wrong with the rectum, and leads him to examine the parts, and in this way tends to protect him from very gross errors in diagnosis. An examination made early in the disease may prevent most serious consequences for the patient. Unfortunately, the great majority of medical men when they are confronted with a patient who complains of constipation, and at the same time states that a certain amount of blood is occasionally passed by the rectum, content themselves with a brief inspection of the anal region, and the diagnosis, " chronic piles." They omit careful examinations, particularly when a few dilated veins or piles are found near the external sphincter. Leube, I think, is fully justified in insisting that in all these cases a careful digital examination of the rectum should be performed. As I have said, he states that in many of these cases the swelling of the hemorrhoidal veins will be found to be due to the presence of a carcinoma of the rectum. From his personal experience he considers the appearance of piles to be one of the earliest symptoms of rectal cancer. I might add to Leube's description of this condition and to his interpretation of hemorrhoids that the suspicion of carcinoma of the rectum is very much more probable when constipation and hemorrhoids come on more or less suddenly in an individual who previously had his bowels open perfectly regularly, whereas the harmless form of hemorrhoids usually develops as the result of prolonged and persistent constipation. The latter, moreover, rarely appears suddenly, but usually develops slowly and exists for

many years before the patient consults his medical attendant for constipation and the passage of blood in the dejecta.

The only way in which to clinch the diagnosis is to perform a digital examination of the rectum. If this is done, the presence of a tumor can always be recognized with absolute certainty, provided, of course, it is within reach of the fingers. In the great majority of cases it is quite unnecessary to use a speculum. As a rule, the introduction of a speculum into the rectum is very painful, and nothing can really be learned from this form of examination which contributes anything more to the diagnosis than could have been discovered by palpation. The educated finger will be able to appreciate certain differences in the feel of the tumor and to interpret the sensations imparted to the sense of touch. In this way the size, the outline, the stage of development, and the exact situation of the neoplasm can usually be determined quite positively. In some cases the finger comes on the neoplasm as soon as it is inserted through the anus; in other cases again the finger will have to be pushed in very far, and may barely reach a growth in the upper portions of the ampulla; while again, in other cases, the cancer may be situated so high up in the rectum that it is not accessible to palpation at all. R. Volkmann gives an emphatic warning against any attempt to introduce the whole hand into the rectum in cases of carcinoma. Some clinicians, however, advise doing this, and claim that by this manipulation those portions of the rectum that are situated above the reflexion of the peritoneum and the third sphincter can be palpated; in other words, that the upper end of the ampulla of the rectum can thus be examined by the fingers. Some clinicians even advise inserting the whole hand in order to perform very careful palpation of portions of the rectum that are situated within comparatively easy reach of the finger. This procedure is to my mind very dangerous, for in carcinoma the intestine is rigid, and to some extent fragile, so that there is always danger of rupturing the bowel. The feel of the neoplasm varies, of course, according to its form : in some cases isolated circumscribed nodules covered with soft and freely movable mucosa are felt; in other cases a hard, plate-like mass that involves large portions of the bowel circumference; in still other cases again there is a prominence resembling a mushroom growth, or again a massive, hard, or solid cauliflower mass. The surface of the latter may be more or less uniform, or it may be extremely uneven and nodular. The last-named form of carcinoma may fill out the whole rectum. Finally, we occasionally encounter hard, annular strictures of the bowel that feel like cicatricial tissue. If such a carcinomatous stricture is situated in the higher portions of the rectum, a feel is imparted to the finger that is similar to the sensation experienced on palpating the cervix of the uterus. In cases of carcinoma of the rectum the finger usually becomes covered with blood, pus, or sanious material, particularly when the carcinoma is in the stage of breaking down. In some instances the neoplasm is felt to be firmly adherent to the surrounding parts.

As a rule, it is comparatively easy to determine whether a tumor that is felt in the rectum really belongs to this portion of the bowel. Occasionally, however, it may be very difficult to decide in which part of the pelvis the growth is developing. Tumors of the female genital organs are particularly misleading and are responsible for many wrong diagnoses. To be sure on this point, a careful gynecologic examination is necessary. Certain diseases of the bladder and the prostate may also be confusing, and in this connection the whole urinary apparatus must be examined in order to exclude tumors or swellings of these parts. In very rare cases morbid conditions starting from the bones of the pelvis or from the periproctal connective tissue must be considered in making a differential diagnosis. Finally, it is occasionally necessary to differentiate some innocent tumors of the rectum from the malignant form. It is frequently impossible in the latter instance to arrive at a positive conclusion unless small particles of the tumor are excised and submitted to microscopic examination. Sometimes a stenotic ring is felt in the rectum, and the question arises whether this annular stricture is carcinomatous in character or of a cicatricial nature (most frequently syphilitic). This decision can usually be made by simple palpation, for a simple cicatricial stricture of the rectum feels smooth and uniform and is not ulcerated, whereas a carcinomatous stricture is usually nodular, uneven, and frequently ulcerated. In many cases, however, these differences are not sufficiently marked to justify a positive differential diagnosis, for the lower margin of a large carcinoma of the rectum may form a narrow ring around the circumference of the bowel and still feel smooth and uniform, whereas the larger mass of the carcinoma situated above this ring may be in a state of advanced ulceration, although not accessible to the palpating finger. In a case of this kind, again, a piece must be excised and submitted to histologic examination, otherwise the diagnosis of carcinoma cannot be made.

The same factors which are responsible in carcinoma of the colon for changing the clinical picture presented by the simple local symptoms of the neoplasm may also be at work in carcinoma of the rectum.

In the first place, metastases frequently occur in other organs. It is a remarkable fact that metastatic growths in other organs are more frequent in small carcinomata of the rectum than when there are large growths of this portion of the bowel. It is an old and excellent rule always to examine the rectum carefully in carcinoma of the liver when the primary seat of the carcinoma cannot be found. In this disease, for instance, the stomach would naturally be examined first; if no definite evidence for the diagnosis "primary carcinoma of the stomach" is found, the suspicion is always directed toward the rectum. In many of these cases a small primary carcinoma of the rectum is found, even in patients who have never shown any symptoms pointing to the existence of such a lesion.

In the second place, the tumor may directly involve neighboring organs in the carcinomatous process, and in this way lead to the production of definite symptoms. Owing to the anatomic position of the rec-

tum, the possibilities in this direction are less varied than in the case of the colon. Rectal carcinomata, therefore, can become adherent only to the bladder and the vagina, or occasionally perforate into these organs and set up abnormal communications and fistulæ. Fistulous openings between the bladder and the rectum and between the vagina and the rectum are quite common. On the other hand, as emphasized by Kraske, rectal carcinoma very rarely gives rise to periproctitic abscesses and fistulæ, in contradistinction to syphilitic ulcerations and stenoses, in which this occurrence is relatively frequent.

The third factor which may complicate and confuse the simple clinical picture of the local effects of carcinoma of the rectum is involvement of the peritoneum. It has been seen that in carcinoma of the colon numerous complications may arise from this source, and that involvement of the peritoneum is one of the most dangerous sequelæ of this disease. In carcinoma of the rectum the peritoneum is very rarely involved. This is chiefly due to the fact that the rectum is situated in a position where it is not in such intimate contact with the peritoneum. The anatomy of the rectum shows that direct involvement of the peritoneum by contiguity of tissue can occur only in carcinoma situated high up in the rectum—that is, in carcinomata that are essentially in the lower end of the sigmoid flexure. When the peritoneum, therefore, is involved in these cases, the general symptom-complex developed is essentially the same as that seen in carcinoma of the sigmoid flexure.

[PRIMARY CARCINOMA OF THE VERMIFORM APPENDIX.

This is a rare condition, but Kelly,[1] in 1900, collected 20 cases, and since then others have been recorded by Hurdon,[2] Rolleston,[3] Whipham,[4] Harte and Willson,[5] Jessup.[6] It must not be confused with carcinoma of the cecum involving the appendix, a much commoner condition, of which Kelly has collected 79 examples. The condition is probably much less rare than it appears to be, as a number of the cases have been detected only in the course of systematic microscopic examination of appendices removed during life for appendicitis. In most of the cases the growth is a spheroid-celled carcinoma, and thus differs from carcinoma of the colon, which is columnar celled. In some cases the patients were very young for carcinoma; in Jessup's 13 cases 2 were under twenty years of age and 4 others under thirty. In about half the cases the symptoms have been those of appendicitis, and in some, of the relapsing form; it is quite possible that the development of carcinoma is the outcome of inflammatory hyperplasia. All the cases of carcinoma of the vermiform appendix which have been published do not stand impartial criticism. Thus Elting[7] has collected 43 recorded

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[1] A. O. J. Kelly, *Proc. Path. Soc.*, Philadelphia, 1900, p. 109.
[2] Elizabeth Hurdon, *Johns Hopkins Hosp. Bull.*, July–August, 1900, p. 175.
[3] Rolleston, *Lancet*, 1900, vol. ii., p. 11.
[4] T. R. Whipham, *ibid.*, 1901, vol. i., p. 369.
[5] Harte and Willson, *Medical News*, August 2, 1902.
[6] D. S. D. Jessup, *Medical Record*, August 23, 1902.
[7] Elting, *Annals of Surgery*, April, 1903, pt. cxxiv., p. 549.

cases, but only admits 23 as undoubted examples. In 17 of the 23 the patients' ages were available, and of these, no less than 53 per cent. were under thirty years of age, while 24 per cent. were under twenty years of age. His youngest case was in a child aged twelve years.—Ed.]

CARCINOMA OF THE DUODENUM.

Although carcinoma of the duodenum is a very rare condition and consequently commands comparatively small practical interest, the disease, nevertheless, produces so typical and peculiar a group of clinical manifestations that it deserves to be described here. Among carcinomata of the small intestine they occupy the first place, exceeding in frequency their incidence in the ileum and even more in the jejunum. Carcinoma of the duodenum has a number of symptoms in common with all carcinomata of the intestine: in the first place, there are always anemia and general cachexia; then there is pain, which, as a rule, is felt chiefly in the right hypochondriac region—sometimes, however, in the middle epigastric region or in the whole upper part of the abdomen. Finally, there is the tumor. It is true that frequently the tumor cannot be felt; when, however, palpable, the growth is usually found in the right hypochondriac region near the middle line. It has been shown that carcinomata of the large intestine are freely movable, and it is also known that carcinomata of the jejunum and the ileum are particularly motile—in fact, carcinomata of these portions of the small intestine are frequently movable to a most astonishing degree. Carcinomata of the duodenum, on the other hand, in the descending or in the lower horizontal parts, are, as a rule, quite fixed, or at most slightly movable. This applies not only to those cases of carcinoma of the duodenum which have become adherent to adjacent parts by the extension of the growth into neighboring organs or by the formation of peritoneal adhesions, but also to all carcinomata of this part of the bowel, even when they are perfectly free and unattached. The reason for this peculiar immotility of carcinomata of the duodenum is that the middle and lower portions of the duodenum are attached and firmly fixed to the posterior abdominal wall by tight bands. The upper horizontal portion of the duodenum is not so tightly anchored, consequently carcinomata of this division may possess a slight degree of motility.

In the preceding paragraphs it was shown that the symptoms of carcinomata of the rectum and the colon are chiefly due to interference with the normal functions of the intestine. The same applies to carcinomata of the ileum, for here all the disturbances seen are really perversions of intestinal function. In the case of carcinoma of the duodenum, however, nearly all the symptoms that appear are stomach symptoms. Even when gastric symptoms are not the only ones present, and some intestinal symptoms appear in addition to the gastric symptoms, the latter predominate so greatly that disease of the stomach is frequently simulated. As a matter of fact, cancer of the duodenum resembles cancer of the stomach in the majority of its features so much that it is often quite impossible to make a direct differential diagnosis

between the two diseases. In the course of cancer of the duodenum there are loss of appetite, belching, and vomiting. The vomited material varies greatly in character and in appearance; occasionally so much blood is mixed with the vomit that it may be described as pronounced hematemesis. In some instances, it is true, the disease runs its course without vomiting, while in others vomiting occurs only at long intervals. Although in carcinoma of the colon and the rectum the character of defecation and the nature of the stools are of paramount importance, these features are of subordinate importance in carcinoma of the duodenum. As a rule, it is true, patients suffering from carcinoma of the duodenum are constipated; in other cases, however, exactly the opposite condition—namely, diarrhea—is present, while in other cases defecation is perfectly normal. The appearance of the abdomen is peculiar in this disease, for it is always flat or may be hollow, even in cases of stricture in the lower portion of the duodenum.

All these symptoms occur in all cases of carcinoma of the duodenum. In addition, there are certain symptoms that develop in the course of this disease and are more or less characteristic of involvement of certain divisions of the duodenum. This seems surprising when we consider how short the duodenum is, but it is true, nevertheless, that the symptom-complex is fundamentally different when the upper horizontal portion of the duodenum is involved from that when the lower horizontal portion is involved, and, again, is different when the middle portion of the descending part with the diverticulum of Vater becomes carcinomatous. Boas, Rosenheim, and Whittier have called particular attention to these fundamental differences, and A. Pic, in particular, has given a detailed description of these peculiarities. The latter author distinguishes between parapyloric, prejejunal, and periampullary carcinoma of the duodenum. In the first case the upper portion of the duodenum is involved; in the second case, the lower portion; in the third case, the middle portion. These designations are very useful and very brief, and will, therefore, be employed in the following description. Boas makes use of the terms supra-, infra-, and circumpapillary carcinomata, according to their position relative to the papilla Vateri.

[As the symptoms and signs are in great measure determined by the position of the tumors as regards the biliary papilla, the following terminology has advantages—viz., suprapapillary or supra-ampullary, for growths above the papilla which imitate growths of the pylorus; periampullary or perivaterian, for growths involving the mucous membrane of the duodenum in the region of the biliary papilla; and infrapapillary or infra-ampullary, for growths involving the remainder of the duodenum.—Ed.]

If the carcinoma of the duodenum is parapyloric (suprapapillary), the upper horizontal part of the duodenum becomes stenosed. Whenever this occurs, the same sequelæ are produced as in stenosis of the pylorus—namely, pronounced dilatation of the stomach with all the attendant symptoms. It can readily be understood that the diagnosis between parapyloric carcinoma of the duodenum and carcinoma of the pylorus is

not only very difficult, but in actual practice is, as a rule, quite impossible.

Prejejunal (infrapapillary) cancer of the duodenum produces a clinical picture in which the gastric symptoms again predominate; at the same time a congeries of symptoms appears, due to the occlusion of the duodenum below the entrance of the common duct into the bowel; in other words, there is stasis of bile and of pancreatic juice, and regurgitation of these secretions into the stomach. As a natural result of this stagnation and regurgitation, bilious vomiting and a number of other changes in the stomach-contents result. In order to avoid repetition, the reader is referred for the details of the analysis of the stomach-contents in these cases to the section on Stenosis of the Duodenum, where this subject is dealt with at length.

Periampullary (circumpapillary) carcinoma of the duodenum—that is, a cancer of the descending division of this portion of the bowel—presents yet another disease-picture. Pic insists that a careful study and differentiation of the few reported cases of this disease are necessary. In some of the recorded cases the neoplasm involves considerable portions of the bowel, so that the carcinoma is not periampullary alone, but also involves, to a certain extent, the upper or the lower portion of the duodenum. Here, then, the middle portions are not involved alone, and the affection of the upper and lower portions will naturally cause symptoms of parapyloric and prejejunal cancer to appear. It may happen, therefore, that even in cases in which the middle portion of the duodenum is distinctly involved in the carcinomatous process, symptoms of peripyloric or prejejunal cancer will, nevertheless, be conspicuous. A few cases, however, are actually on record in which carcinomata of different size were strictly limited to the region of the ampulla of Vater. These are the cases that usually, although not always, are complicated with icterus. These are the cases, moreover, in which anemia, cachexia, and jaundice develop together and slowly increase in severity, without, at the same time, presenting any severe local gastro-intestinal symptoms. Pain is usually absent. On careful examination the tumor can frequently be made out deep down in the right hypochondriac region, usually near the middle line. The feel of these tumors is hard, and, as a rule, they are immotile. Pic discusses the question whether these carcinomata of the ampulla of Vater are not, properly speaking, carcinomata of the pancreas or of the duodenum. I think Pic is correct in formulating this question and in discussing it. He calls attention to the identity of the clinical picture presented by periampullary carcinoma of the duodenum and the picture presented by carcinoma of the head of the pancreas. He also calls attention to the fact that glandular acini are present in the ampulla of Vater, from the cells of which the glandular epithelioma could originate. There is another argument in favor of this view which I wish briefly to mention—namely, the macroscopic appearance of carcinomata of the duodenum found in this region. These neoplasms, as a rule, constitute circumscribed tumors that occupy only a portion of the intestinal wall and consequently resemble in this respect

carcinomata that are situated in the head of the pancreas itself, whereas true duodenal carcinomata show the same tendency that is so common in other forms of intestinal carcinoma—namely, to develop in an annular or cylindric manner and so involve the whole circumference of the intestine.

[Carcinoma of the duodenum is a disease of late middle or advanced life—in 41 cases the average age was fifty-two years, while in 10 out of 33 cases collected by Nattan-Larrier [1] the patients were over seventy years of age. It occurs more often in the male sex—in 43 cases 10 only were females (Rolleston).[2]

Incidence of Carcinoma in the Different Parts of the Duodenum.—In 41 collected cases the first part was affected alone in 8, and in common with the second part in 5 more. The second part is most often affected—in 41 cases it was involved in 24, and in 5 more was involved in common with the first part. The third part is the least often affected—in 41 cases it was affected only in 4 (Rolleston).

Carcinoma has a special tendency to arise in the mucous membrane of the duodenum covering the biliary papilla. This condition must be distinguished from carcinoma arising from the mucous membrane inside the biliary papilla, or, in other words, the ampulla or diverticulum of Vater. The term carcinoma of the ampulla of Vater is sometimes erroneously applied to a duodenal carcinoma involving the intestinal surface of the biliary papilla.

When duodenal carcinoma involves the biliary papilla, jaundice is usually present—it was so in 23 out of Mathieu's [3] 25 cases, but it may be intermittent, and thus differs from carcinoma of the head of the pancreas or of the common bile-duct, where jaundice, when it once appears, is permanent and progressive. In rare instances (Lannois and Courmont,[4] Mauclaire and Durrieux,[5] Descos and Bériel [6]) the growth, although involving the papilla, does not produce jaundice. Carcinoma of the duodenal surface of the biliary papilla is very prone to be followed by infection of the common bile-duct and suppurative cholangitis.

In carcinoma of the third part of the duodenum the symptoms are those of obstruction, with intermittent vomiting and bile in the vomit. The constant occurrence of bile in the vomit should suggest infra-ampullary duodenal carcinoma or a gastrobiliary fistula, and in such a case the vomit should be tested for trypsin by seeing whether fibrin is digested in an alkaline solution, so as to make a diagnosis between these two conditions.—Ed.]

CARCINOMA OF THE SMALL INTESTINE.

It has repeatedly been pointed out in the previous pages that carcinoma of the ileum is rare, and that carcinoma of the jejunum is still

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[1] Nattan-Larrier, *Gaz. des Hôp.*, Paris, Dec. 9, 1897.
[2] Rolleston, *Lancet*, 1901, vol. ii.
[3] Mathieu, *Traité des Maladies de l'estomac et l'intestine*, p. 921.
[4] Lannois and Courmont, *Rev. de Méd.*, 1894, p. 291.
[5] Mauclaire and Durrieux, *Bull. Soc. Anat.*, Paris, 1897, p. 721.
[6] Descos and Bériel, *Rev. de Méd.*, 1899, p. 633.

rarer. The symptomatology of cancer of these portions of the bowel is practically identical with the symptomatology of cancer of the upper portion of the colon. In both conditions there are anemia and cachexia, pain, and attacks of colic, a tendency to constipation which occasionally alternates with a tendency to diarrhea; there may even be repeated violent hemorrhages from the anus, and occasionally the clinical picture of enterostenosis is presented. The only way in which to decide whether the neoplasm is in the small or in the large intestine is to examine the tumor very carefully, provided, of course, that it can be felt. Unfortunately, it is by no means always possible to feel carcinoma of the small intestine. Treves goes so far as to state that of 10 cases of carcinomatous stricture of the small intestine that he collected and analyzed, only 3 presented a distinctly palpable tumor. If a tumor can be felt at all, the first thing to be done is to determine its exact position; that is, principally, to compare its seat with the natural topography of the colon. In general it is very difficult to draw any definite conclusions as regards the existence of carcinoma of the small intestine unless the tumor of the intestine is anchored and fixed somewhere in the abdomen, either by extension of the carcinoma into neighboring organs or by peritoneal adhesions. Carcinomata of the small intestine, it is well known, are the most motile of all forms of intestinal cancer. Carcinoma of the transverse colon is the only form which is in any way comparable to them in this respect. This motility raises almost insurmountable difficulties to the diagnosis, for carcinomata of the small intestine, as well as carcinomata of the colon, may become displaced downward. When this occurs, it is quite impossible to draw any conclusions from the position of the tumor or from its motility. The only way in which a diagnosis can be made in these cases is to study the general development of the disease and to consider all those factors in the differential diagnosis which have been discussed in the paragraphs on the localization of enterostenosis.

Course.—The termination of carcinoma of the intestine when allowed to run its natural course is unavoidably death. The exact manner in which death is caused can readily be understood from the description of the anatomic and clinical features of the disease already given. Death may be produced either by occlusion of the intestine or by general exhaustion resulting from bloody, sanious, or purulent diarrhea. Death may be due to general cachexia or to the very varied group of symptoms produced when perforation into some other organ occurs and abnormal communications with some viscus are formed. Finally, one of the different forms of peritonitis that are known to develop in the course of cancer of the intestine or secondary growths in some vital organs, or, finally, septic fever, may all terminate the scene. No case of spontaneous cure of a carcinoma of the intestine is on record. It can readily be understood that no exact data exist in regard to the average duration of carcinoma of the intestine, for it is almost impossible to determine the exact time that elapses between the earliest stage of an intestinal carcinoma and the lethal issue; for even in cases of

rectal carcinoma the disease is rarely recognized before it has progressed to a certain extent. This is due to the fact that the very first stages of a rectal carcinoma produce no symptoms, and the neoplasm must have reached certain dimensions before the appearance of symptoms which lead the medical man to make a careful local examination. Eichhorst observed a patient with carcinoma of the rectum in the beginning of the fourth year after the onset of the disease, the patient having obstinately refused operation up to this time. Other authors have made statements in regard to other cases of carcinoma of the colon that lasted as long as, or even longer than, this case of Eichhorst's, one author speaking of "several years," another one of "at least five to six years." I think these statements must be regarded with a great deal of distrust, because, as a matter of fact, no one can prove positively that the intestinal symptoms that were produced as long ago as "several" or "five or six" years were really due to the presence of a carcinoma; it seems much more plausible to argue that these early symptoms were due to the existence of some other lesion, such as cicatricial stenosis of the bowel, and that a carcinoma secondarily developed on the basis of such primary lesions. Maydl has made the statement—which, by the way, is entirely hypothetic—that scirrhous forms of carcinoma in other regions of the body are known to have persisted for from five to seven years, and quotes several examples of this disease. As a matter of fact, even if all this were positively demonstrated, this statement would prove nothing in regard to the duration of scirrhous carcinomata in the intestine, for whenever scirrhous cancers are found in the intestine, they are annular, and consequently must rapidly produce severe symptoms of stenosis of the bowel.

In general, therefore, the statements and observations of the majority of investigators are to the effect that the total duration of a carcinoma of the colon which runs its natural course without any surgical interference averages approximately from six months to two years. Possibly carcinoma of the rectum runs a somewhat longer course. On the other hand, there are cases in which death may occur very much earlier, owing either to the spontaneous development of the process or to the appearance of some intercurrent complication. As prototypes of these two forms of termination, two cases of Bamberger's which are frequently mentioned in the literature will be quoted:

The first patient was a woman of fifty-seven years who looked very much younger than she was and was well nourished. She was perfectly well until one month before her death, when the following symptoms appeared acutely: Great loss of appetite, vomiting, pain in the epigastrium, and, two weeks later, general weakness. On physical examination only one abnormality could be discovered—viz., a tumor situated deep down in the right side of the abdomen. The growth was hard, barely as large as a pigeon's egg, and not in the least painful. In the course of the next four days, however, this tumor developed very rapidly, so that it became as large as an adult's fist. The liver and the spleen at this time were normal in size. At the expiration of another

four days the liver was found to be enormously swollen, so that it constituted an uneven, hard swelling that was not painful spontaneously or tender on pressure. The general weakness increased, the patient developed meteorism, and two days later death occurred. On autopsy, a medullary carcinoma of the cecum was discovered, also carcinomatous adhesions between numerous loops of intestine, multiple carcinomata of the peritoneum and mesentery, and numerous carcinomatous nodules disseminated throughout the liver, which was probably due to embolic infection through the portal vein.

The second patient was a man of forty years who had been perfectly healthy and robust up to the time of his final illness. The disease in his case, too, was of sudden onset. He ate a large quantity of lentils and suddenly began to suffer from violent stomachache, severe meteorism, constipation, and vomiting. In three days he was dead. On autopsy an annular carcinomatous narrowing of the sigmoid flexure was found. The lumen of the intestine was not very much narrowed, but masses of undigested lentils were found that had become lodged in the stricture and had caused complete occlusion of the bowel.

SARCOMA AND LYMPHOSARCOMA.

These two forms of malignant tumor will be considered together, inasmuch as the clinical symptoms produced by sarcomata and lymphosarcomata cannot be separated, even though the two forms of neoplasm are anatomically very different. Perhaps when the future has supplied more histologically examined material, it will become possible to differentiate clinically lymphosarcoma from other forms of sarcoma of the intestine. I believe many indications point to such a result. It need hardly be pointed out that the clinical pictures presented by carcinomata of the intestine, on the one hand, and sarcomata and lymphosarcomata of the intestine, on the other, resemble each other in many essential points. There are, however, certain differences between the two forms of tumor that are sufficiently pronounced to make a separate description possible, and, in fact, necessary. It will not, however, be necessary to enter into all the details of the symptoms produced by sarcoma and lymphosarcoma of the intestine, and these remarks will practically be confined to bringing out the differences that exist between the clinical pictures presented by these tumors and by carcinoma of the intestine.

Sarcoma of the intestine is far less frequent than carcinoma of the intestine. On page 403 I mentioned that in the course of twelve years—from 1882 to 1893—2125 cases of carcinoma (in 21,358 sections in general) were examined postmortem in the General Hospital in Vienna, and that of these 2125 cases, 243 were carcinomata of the intestine. In the same period of time 274 autopsies were made on cases of sarcoma; of these, only 3 were sarcomata of the intestine. Finally, of 61 cases of lymphosarcoma that were examined postmortem, only 9 lymphosarcomata belonged to the intestine. These figures

I consider absolutely reliable, especially as Kundrat, who was then the director of the Pathological Institute, paid particular attention to this very subject. Müller examined the postmortem material in Bern and found that, of 521 cases of carcinoma examined postmortem, 41 were carcinomata of the intestine, and that of 102 sarcomata, only 1 was a sarcoma of the intestine (sarcoma of the ileum). Reports from other pathologic institutes are entirely coincident. In Prague, in 13,036 sections in fifteen years, there were only 13 intestinal sarcomata (7 of ileum; 3 of jejunum and ileum; 3 of cecum)—all lymphosarcomata.

Seat of the Sarcomata.—In opposition to carcinoma, which occurs in the large intestine with disproportionate frequency, sarcoma occurs at least as frequently in the small intestine; in fact, one form, the lymphosarcoma, preponderates in the small intestine.

Of the 9 cases of lymphosarcoma before mentioned, 1 was in the duodenum, 3 were in the jejunum, 3 in the ileum, and 2 in the cecum. Of the 3 sarcomata, 1 in each of the ileum, cecum, and rectum. Numerous statistics show that the various parts of the small intestine are involved quite equally. Of the cases of sarcoma of the small intestine collected by Libman, 15 were of the duodenum, 18 of jejunum and ileum, 14 of ileum, and 3 involved the entire small intestine. Sarcoma of the large bowel is most frequent in the rectum. Of Krueger's 37 cases of intestinal sarcoma, 16 were of the small bowel alone, and 16 of the rectum. The ileocecal region ranks next. Smoler mentions 13 cases involving the entire intestinal tract, of which 3 were of this region. Blauel, Engstroem, and R. Schmidt have each recently published 2 cases. I also have within recent years encountered 2 in the colon—1 each in the ascending and the descending. One was a lymphosarcoma.

Sarcomata usually attain a large size—even to the size of a child's head or twice that size. The neoplasmatic infiltration at times spreads over a large part of the intestine. Glinski describes one case thus: The entire large intestine, from the anus to the ileocecal valve, presents a rigid, patulous, thick-walled, rather wide, cylindric tube, and the lower 10 cm. of the ileum are also infiltrated.

The sarcomata most generally originate in the submucosa, although individual cases are reported from the mucosa. The musculature is early attacked, but the serosa is seldom involved. Histologically, they appear usually as small-celled round-celled sarcomata; in individual cases as spindle-celled. The sarcomatous growths of the rectum are remarkably often melanotic. As early as 1894 Strasburger, despite the limited number of sarcomata of the large gut, collected 9 melanotic ones of the rectum. Treves reports a case as primary melanosarcoma of the ileum.

[Jopson and White [1] were able to collect 22 cases of primary sarcoma of the colon, while Libman [2] has collected 59 cases of sarcoma of the small intestine. In Libman's cases of sarcoma of the small intestine

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[1] Jopson and White, *Amer. Jour. Med. Sci.*, 1901, p. 807.
[2] Libman, *ibid.*, vol. cxx., p. 309.

the oldest patient was seventy years and the youngest five days ; in 49 cases in which the sex was recorded, 35 were males and 14 females ; more than half the cases occurred between the ages of twenty and forty. In 3 of Libman's cases the clinical picture was that of appendicitis. Ablon[1] has brought together 10 cases of sarcoma of the intestine in children.

In Jopson and White's 22 cases of sarcoma of the colon, the sexes were about equally represented, the ages varying from two to over sixty years, but the first decade contained the largest number, the fourth coming next. In 20 cases 10 were round-celled, 9 were lymphosarcomata, and 1 was a spindle-celled sarcoma. In 10 out of 22 cases the bowel was resected, with 5 deaths from this operation. Babes and Nanu[2] have described a remarkable case of myosarcoma as large as two fists in the small intestine ; it showed transitional phases of smooth muscular fibers to sarcoma cells.

Cases of melanotic sarcoma of the rectum have also been described by Heaton,[3] Ball,[4] and De Buck and Vanderlinden.[5] Bland Sutton points out that melanotic sarcoma, often pedunculated and of considerable size, is not uncommon in white horses. I have examined one or two cases of melanotic sarcoma of the skin at the anal margin, which is normally pigmented, but this is different from a melanotic sarcoma of the bowel. It is not clear that this difference has always been kept in mind. In connection with primary melanotic sarcoma of the rectum or protodeum, it is interesting to note that exceptionally, as in Treves's[6] case of melanotic sarcoma of the hard palate, a similar growth may arise in the stomodeum. Sarcoma of the vermiform appendix is very rare indeed. Warren[7] and Paterson[8] have described a round-celled sarcoma, and Glazebrook[9] an endothelial sarcoma.—ED.]

Pic has collected a number of cases of primary lymphosarcoma of the duodenum from the literature, but calls attention to the fallacy of a statement that some authors have made—namely, that primary "cancers of the small intestine" are almost always lymphosarcomata. I think that Pic is right in this matter.

[It may be pointed out that a colloid carcinoma may present histologic appearances which may suggest a myxosarcoma. So eminent a pathologist as the late Professor Kanthack[10] was, for a time, inclined to regard a colloid carcinoma of the colon and lower part of the ileum in a boy aged seventeen as a myxosarcoma.—ED.]

Sarcomata of the intestine in the great majority of cases have their starting-point in the submucous layer of the bowel. According to some published observations, it appears that sarcomata may also start from

[1] Ablon, *Thèse de Paris*, 1898.　[2] Babes and Nanu, *Berlin. klin. Wochenschr.*, 1897.
[3] Heaton, *Trans. Path. Soc.*, vol. xlv., p. 2.
[4] Ball, *Diseases of Rectum and Anus*, second ed., p. 341.
[5] De Buck and Vanderlinden, *Belgique Med.*, November 9, 1899.
[6] Treves, *Trans. Path. Soc.*, vol. xxxviii., p. 350.
[7] Warren, *Boston Med. and Surg. Jour.*, Feb. 24, 1898.
[8] Paterson, *Practitioner*, vol. lxx., p. 515.
[9] Glazebrook, *Virginia Med. Monthly*, vol. xxii., p. 211.
[10] Kanthack and Furnival, *Trans. Path. Soc.*, vol. xlviii., p. 102.

the mucosa. The muscular coat is involved in the process early in the disease, whereas the serosa usually remains free. The sarcomatous tumors that have been examined histologically were found, in the majority of cases, to be small-celled round-celled sarcomata. In one or two instances the structure of the neoplasm was that of a spindle-celled sarcoma. Treves reports a tumor of the ileum that, according to his statements, was a primary melanosarcoma. The same kind of tumor has several times been found in the rectum.

Lymphosarcomata of the bowel always start from the lymphatic apparatus of the intestine, the solitary as well as the agminate lymph-follicles. The rule seems established that sarcomata as well as lymphosarcomata of the intestine are always primary in this situation.

[The term lymphosarcoma is used above as synonymous with lymphadenoma, which is not a sarcoma. Still it may be useful to draw attention to Newton Pitt's [1] observations on lymphadenoma of the intestinal tract. Lymphadenoma may occur in two forms : (1) Attacking the lymphoid structures in the mucous and submucous coats and thus producing tumors which project into the lumen of the bowel, but do not materially narrow its caliber, and usually do not ulcerate. The ileum near the ileocecal valve is chiefly affected. (2) It may start in the lymphatic glands of the mesentery and spread into the bowel by the subserous lymphatic vessels. The intestine is invaded from without and becomes inclosed in a lymphadenomatous sheath which invades the muscular coat, paralyzes it, and thus produces dilatation. Not being well supplied with blood-vessels, the growth, when it reaches the mucous membrane, is more liable than the first-described form to ulcerate. It does not specially attack Peyer's patches and the solitary glands.—Ed.]

It is true that sarcomata or lymphosarcomata may involve the intestine by contiguity from some neighboring organ ; a primary lymphosarcoma, for instance, of the mesenteric and retroperitoneal glands may extend until it involves the intestine, but secondary lymphosarcomata in the intestine due to metastasis are hardly ever found. Conversely, a primary sarcoma of the intestine very commonly produces secondary metastatic sarcomata in other organs. In the case of lymphosarcoma of the intestine it appears that metastases occur only in the regional lymph-glands.

The changes in the intestine at the site of a sarcomatous growth are very peculiar, being exactly the opposite of those produced by a carcinoma of the bowel ; the latter lesion, as we have said, in the overwhelming majority of cases, leads to narrowing and stenosis of the intestine. Sarcomata, on the other hand, do not produce stricture of the bowel. The sarcomatous neoplasm usually develops in a longitudinal direction along the intestine, and lymphosarcomata in the same way produce extensive infiltration of various intestinal coils over wide areas. In either case there is no stenosis of the intestinal lumen, but, on the contrary, a dilatation of the affected portion of the bowel, which in lymphosarcoma appears to be almost constant. In the 13 cases

[1] G. Newton Pitt, *Trans. Path. Soc.*, vol. xl., p. 80.

occurring in the Prague institute, Smoler found the intestine markedly dilated in all. Kundrat has described the appearance of the intestine in lymphosarcoma as spindle-shaped; Glinski describes a diffuse, cylindric, and Madelung-Baltzer speaks of aneurysmal, dilatation of the bowel in sarcoma. Occasionally this dilatation of the affected portion of the bowel attains almost monstrous dimensions. Bessel-Hagen has described a case which illustrates this excellently. His patient was a boy of seven years in whom, at the autopsy, a large portion of the jejunum was found to be infiltrated with sarcomatous tissue. In this part of the bowel there was an aneurysmal sac which was dilated so as to be as large as a man's fist. The cause of the dilatation is to be sought in the involvement of the muscular coat, and the form depends upon the degree of affection of the muscles. Frohmann, who describes a jejunal sarcoma with the intestinal lumen "of normal width throughout," finds, from the literature, that sarcoma of the small intestine did not cause stenosis in about 70 per cent. of the cases, it being only in rectal sarcoma, as in carcinoma, that stenosis is the rule. (For the general gross anatomic results of this form of tumor of the intestine the reader should refer to the section on Carcinoma, as the two are essentially alike in this respect.)

In speaking of the frequency of carcinoma of the intestine attention was called to the fact that cancer of the bowel is relatively more frequent in young persons than is cancer of any other portion of the body. This truism applies with still greater force to sarcoma of the intestine, for this lesion seems to predominate in the third and fourth decades of life. The oldest patient with a sarcoma of the intestine that is on record was seventy years old; the youngest, whose case was reported by Madelung, a boy of four years. Horn describes a case of congenital sarcoma of the descending colon with intussusception of the intestine that led to the death of an infant only three days old. [*Vide* Libman and Jopson and White's figures on p. 444.—ED.]

The clinical picture presented by sarcoma of the intestine differs in a marked degree from the clinical picture presented by carcinoma. Very early in the development of an intestinal sarcoma, particularly in the sarcomata of the small intestine, the general health of the patient becomes greatly impaired, whereas the local symptoms are quite insignificant, and at this time occupy a subordinate position in the general syndrome. This peculiar want of proportion between the impairment of the general health and the absence of local symptoms is always seen in sarcomata of the intestine. In carcinoma, as we have seen, local symptoms chiefly predominate in the early stages, and impairment of the general health is rarely seen until later in the course of the disease. Patients with sarcoma of the intestine, on the other hand, emaciate rapidly from the very beginning, become debilitated, feel very weak and tired, and present an anemic appearance. R. Schmidt lays stress upon the early occurrence, in one case of edema, which he thinks must be regarded as due to cachectic hydremia. These general symptoms may all appear before any digestive disturbances or pain is experienced.

The absence of abdominal pain often renders it very difficult in the early stages to recognize that the primary seat of the trouble is in one of the abdominal organs (Madelung). In reply to Engstroem's statement that in both his cases of sarcoma of the small intestine no appreciable loss of strength, and, even less, cachexia, existed, in spite of the fact that the presence of a tumor had been known for several months, I can but add that both cases were round-celled sarcomata, not lymphosarcomata. In some of the cases that have been studied the temperature was raised to 103° F. (39.5° C.) throughout the whole course of the disease.

It appears to be characteristic of sarcoma, in contradistinction to carcinoma, that the former does not produce symptoms of stenosis of the intestine. We have seen that in carcinoma symptoms of enterostenosis are very frequent, and usually occur early in the course of the disease. Treves was the first to call attention to this difference between the two forms of malignant tumor of the bowel. Madelung-Baltzer also investigated this subject very carefully, and fully corroborated Treves' observation. The stools rarely ever present any abnormal features. Occasionally there may be a slight and insignificant constipation that can, as a rule, be relieved without difficulty. Occasionally constipation alternates with diarrhea. In 4 sarcomata in my own experience—2 of the small intestine, 1 of cecum and ascending colon, 1 of descending colon (large-celled, not lymphosarcomata)—there was no constipation. In fact, in the last case there was decided diarrhea, despite the gigantic size of the tumor. Siegel believes, from the study of cases found in the literature, that in 55 per cent. of cases symptoms of stenosis appeared at one time or another, while Frohmann estimates that this occurs in about 75 per cent. (see his anatomic work). That stenosis has been observed in various cases is certain, but no one who has seen a number of cases of internal carcinoma, with its usual inexorable progress from stenosis to occlusion, and has, besides, seen some cases of sarcoma of the intestine, can avoid the conclusion that in the latter stenosis is comparatively very rare. The characteristic symptoms of stenosis, chiefly intestinal rigidity, commonly so marked in carcinoma, are only exceptionally met with in sarcoma, and when they are, there are generally to be found secondary consequences, as kinking, peritonitic incarceration, or complications, as their direct cause. Thus in my case, cited further on, there was tuberculous cicatricial stenosis, and in Schmidt's case the stenosis was not caused by the neoplasm (lymphosarcoma) *per se*, but by adhesion of two loops of ileum and slight kinking of the intestine at the point of adhesion. This important symptom is evidently in relation with the anatomic fact that the early involvement of further stretches of the muscular coat in the tumor-formation, and the consequent elimination of the mucosa, cause a distention of the intestinal walls by the contents, and the above-mentioned widening of the canal may take place. We might naturally expect ileus paralyticus in such cases, from inability of peristalsis to drive the intestinal contents on, and an interesting case of Kraus shows

that symptoms of obstruction may occur. The mechanism of these conditions will be more fully considered under the headings of Stricture and Paralysis of the Intestine.

Very little need be said in regard to the character and the constitution of the tumor. Sarcomata of the intestine frequently grow to a very considerable size. All the facts of importance as regards abdominal tumors in general have been touched on in the account of carcinoma of the intestine in the preceding paragraphs. As a rule, a sarcomatous growth of the bowel can be made out without difficulty; the neoplasm remains freely movable for a relatively long time; as a rule, the swelling is of hard consistence, but occasionally there is a soft or even a fluctuating area, usually in the center of the tumor. This form of tumor, as a rule, grows and develops with great rapidity, so that within a short time it may become as large as an adult's head.

There are also certain marked differences between sarcoma and carcinoma of the intestine as regards the total duration of the disease. In carcinoma, as we have seen, the course of the disease is usually slow, and a rapid course and termination may be considered exceptional. In sarcoma the reverse holds good, the disease usually running a very rapid course and terminating within a comparatively short time after the onset of the disease or the discovery of the first symptoms. The majority of subjects with sarcoma of the intestine die within nine months. There is only one case on record in which the disease lasted twenty-one months; on the other hand, a case is on record in which the patient succumbed to his malady within two weeks. Schmidt's observation, in which he believes that symptoms of sixteen years' duration were attributable to a lymphosarcomatous process, does not appear at all conclusive to me. Death usually results from general cachexia.

I am unable to state definitely whether or not the clinical picture presented by lymphosarcomata of the intestine is very different, because my personal experience of this form of neoplasm of the intestine is too limited. I remember, however, one case in particular which presented some features of interest and will be briefly described here. The case was one of lymphosarcoma of the jejunum. The patient was a woman of twenty-three years. The disease was not recognized as a lymphosarcoma of the jejunum during life, and the patient was treated for tuberculosis of the peritoneum and of the intestine. The duration of the disease was a year and a half, during which time there was no constipation, but, on the contrary, diarrhea was frequent. The dejecta were repeatedly examined for tubercle bacilli, but with a negative result. This patient suffered from violent abdominal pain from the very beginning, which is unusual in lymphosarcoma. Another interesting feature was that after the disease had lasted for almost a year, the patient was only slightly emaciated and developed only a mild degree of anemia, the percentage of hemoglobin at this time, estimated by Fleischl's hemoglobinometer, being 45 per cent. At this time there was no leukocytosis.

The subject was thirty-seven years old; had always been perfectly healthy until the onset of her present illness. The patient was admitted to the clinic on Novem-

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ber 30, 1895, and died on January 28, 1896. The woman stated that her illness had begun in May, 1895; at this time she began to suffer from pain in the abdomen for the first time; this pain was chiefly felt on the left side, and appeared almost without exception immediately after taking food. She never suffered from vomiting. The bowels had always been regular and had only been confined for a few days on two occasions. Very soon after the onset of the pain the patient noticed a peculiar phenomenon on the surface of the left side of the abdomen, which she described as " rounded elevations that rose up, seemed to move to and fro, and to disappear from one place only to reappear in another." In the course of the clinical observation of this patient distinct symptoms of stenosis of the small intestine appeared, stiffening of certain coils of intestine, and visible peristaltic movements of the bowel, remaining strictly limited to the left half of the abdomen; occasionally, but only rarely, these movements spread to the right side. About the level of the umbilicus there was a distinct feeling of resistance on palpation. The tumor in this area could not be distinctly circumscribed and was not very hard. The patient soon showed a moderately severe degree of anemia and began to emaciate considerably. While she was in the hospital the stools remained normal and the bowels were open fairly regularly every day. No bacilli or any other constituents characteristic of any form of intestinal disease were ever found in the stools. The temperature remained subfebrile throughout; in the morning it was normal; in the evening it usually rose to 100.4° F. (38° C.) to 102.6° F. (39.4° C.). The last rise occurred only once, and may be considered the maximum temperature. All the other organs were found to be free from any abnormality, and nothing noteworthy could be discovered anywhere. The lungs in particular were carefully examined and found to be free from disease. A tentative diagnosis was made either of sarcoma of the small intestine or of peritoneal tuberculosis with tuberculous strictures of the small intestine. Professor Weichselbaum performed the autopsy on this patient and found that both conditions were present. Throughout the whole small intestine he found numerous cicatrices of tuberculous ulcers of the bowel. The majority of these cicatrices were placed transversely to the axis of the bowel; they were very deeply pigmented, and varied in size from mere linear scars to large cicatricial areas that were 1 cm. broad. The tuberculous ulcers themselves also varied greatly in size: some of them were small, others were so large as to involve large areas of the circumference of the bowel. In some places there were annular ulcers involving the whole arc of the circumference of the bowel. In two places marked stenosis of the bowel had been produced, with its attendant result, namely, saccular dilatation of the portion of the intestine immediately above the stenosis. The cicatrices usually corresponded to Peyer's patches or the solitary follicles of the bowel. In addition, in the great majority of the cicatrices of these tuberculous ulcers nodular, grayish-white masses were seen; these resembled little tumors, and on microscopic examination were found to be lymphosarcomata. They varied in size, some of them being only one-half a centimeter in diameter, others again being several centimeters in diameter. In some of the cicatrices isolated little nodules were found either at the margin of the ulcer or at their base; in other cicatrices again the margin was formed by a continuous tumor-mass, so that the whole cicatrized area was surrounded by a ridge of lymphosarcomatous growth; again, in other parts, the whole cicatrized area was occupied by lymphosarcomatous tissue, so that the latter had, in a measure, replaced the cicatricial tissue in the affected areas of the bowel. In those portions of the bowel in which the largest tumors were found, the intestinal wall was greatly dilated; in some places, so as to form hemispheric diverticula. A few of the lymphosarcomatous masses in the bowel showed necrosis and softening. Only a very few of the cicatrices were found free from tumor growth, and, conversely, only one single tumor nodule was found in the uppermost portion of the jejunum, which was not definitely related to one of the intestinal cicatrices. In a number of places adhesions had formed between neighboring loops of intestine, and, in addition, evidence of a recent general fibrinopurulent peritonitis was found. Many of the mesenteric glands were as large as beans. On transverse section their cut surface appeared gray, with isolated grayish-white spots. The large intestine was found to be perfectly healthy. A careful examination of the lungs and of all the other organs failed to show any abnormalities whatever.

Libmann, who reports 5 cases of sarcoma of the small intestine from his own experience, states that 3 simulated remarkably closely the clinical picture of appendicitis. I must acknowledge that I did not get this impression from a study of his cases.

INNOCENT GROWTHS OF THE INTESTINE.

BENIGN NEOPLASMS OF THE INTESTINAL CANAL (Neoplasmata Benigna Intestini).

THE benign forms of neoplasm of the intestinal canal are usually relegated to the background in all clinical accounts of intestinal tumors. This is due to the fact that carcinomatous and sarcomatous neoplasms of the intestinal canal are so dangerous and so menacing to the life of the patient that they occupy the most prominent position in the pathology of abdominal diseases. Another reason why benign neoplasms of the intestine are not considered as important as the malignant forms is that they are relatively rare as compared to the latter. Moreover, their clinical significance is, as a rule, very slight. But in spite of all these facts, an account of these forms of tumors will be given in this section in order to round off this work and to make it complete. In addition, this form of tumor of the intestine, while it is usually unimportant and in the great majority of cases harmless, may occasionally produce severe symptoms, and in exceptional instances even cause the death of the patient.

ANATOMY.

Innocent growths of the intestine may be attached to the wall in two ways—that is, they may be connected with the bowel either by a broad base or by a thin stem or pedicle. The recognition of these two forms of attachment has led to a differentiation of benign neoplasms of the intestine on these grounds, so that polypous tumors of the intestine are spoken of. This name has absolutely nothing to do with the histologic structure of the intestinal neoplasm, and refers exclusively to the anatomic fact that it has a pedicle. There is another form of "intestinal polypi," which, however, are quite different histologically from these polypoid tumors of the intestine.

The proper classification of benign neoplasms of the intestinal canal is based on the tissue from which they originate. We distinguish, therefore, tumors arising from the glandular tissue, or adenomata; tumors derived from connective tissue, or fibromata, lipomata, or papillomata; and those which originate from the muscular coat, or myomata; and, finally, benign tumors of the intestine composed of bloodvessels, or angiomata.

Adenoma.—This is the commonest form of innocent growth of the intestine. The tumors arise from the glands of Lieberkühn, and in the duodenum from Brunner's glands. The histologic structure of

adenomata of the intestine is quite typical—namely, acinous. Different forms of adenomata are distinguished according to their shape and outline (Reinhardt, Luschka, Klebs)—namely, a flat form of adenoma attached to the bowel by a broad base, and consequently constituting a diffuse swelling of the affected portion of the bowel, and secondly, a polypoid form. The size of these tumors may vary greatly. As a rule, they are very small—that is, about as large as a pea and not larger than a cherry; occasionally, however, more voluminous growths are observed, reaching the size of a pear or even of an adult fist. The color of these neoplasms is usually red; they have a great tendency to bleed. Their consistence varies: as a rule, they are soft, but occasionally they are found to be more solid and resistant. The surface of these growths is usually smooth; occasionally it is uneven, and may have a cauliflower appearance. The favorite site for these adenomata is the rectum. Here they are found in both forms—namely, either as flat tumors or as pedunculated polypoid tumors. A sessile adenoma of the rectum is usually situated immediately above the anus, and occasionally forms an annular tumor involving the whole circumference of the intestine and narrowing the lumen of the bowel. Adenomata are rarely found in other portions of the intestine. Statistics show that this form of innocent growth of the bowel is specially met with in early childhood,—namely, between the fourth and the seventh year,—and that of the two forms of adenoma, the polypoid form predominates in children. The extreme infrequency of adenomatous rectal polypi may be seen from Bokai's statement that among 65,970 patients he found but 25 cases (16 in boys, 9 in girls). [Ball[1] suggests that the frequency of rectal polypi is associated with that of intestinal worms. —Ed.]

Many cases—to date about 20—of adenoma of the bowel have at one time or another been reported which are extremely interesting and remarkable, from the very extensive distribution of these tumors. Hauser has designated this condition as *polyposis intestinalis adenomatosa*. Luschka reports the case of a woman thirty years old, in whom the whole large intestine was found covered with adenomata. The colon, from the ileocecal valve to the end of the rectum, was covered with thousands of adenomata; they were more thickly disseminated toward the anal end of the bowel than toward the ileocecal end. Bardenheuer and Hauser have reported similar cases. The latter reports a still more remarkable case in an adult,—as a matter of fact, all the cases observed occurred in adults,—in which the adenomatous tumors involved the whole intestinal tract from the rectum to the pylorus; the growths were not uniformly distributed throughout the intestine, but were more numerous in some areas than in others. In the three last-named cases and in another case of Hauser's carcinomatous degeneration of these polypoid and verrucous growths was found in some parts of the bowel, especially in the rectum. Hauser has attempted to explain this peculiar carcinomatous degeneration of benign tumors of the intestine. He

[1] Ball, *Brit. Med. Jour.*, 1903, vol. i., p. 413.

investigated the subject very carefully, and arrived at the conclusion that these tumors are apt to degenerate into carcinoma merely on account of the structure of their epithelium and on account of the chronic irritation that they are exposed to by the bowel contents. In other words, that adenomata of the intestine, owing to these peculiarities, merely develop an increased predisposition to carcinomatous degeneration. He expressly negatives the proposition that these growths must, in a sense, be considered precursors of carcinoma, and that every adenoma of the intestine, provided it is allowed to grow for a sufficient length of time, will lead to carcinomatous infiltration of the tissues of the bowel. Hauser is very explicit in enunciating this view of the connection between multiple glandular tumors of the whole intestinal mucosa and the local development of carcinoma that occasionally follows these adenomata. Rotter describes a case of polyposis recti in a woman thirty-one years old. A palliative operative was performed, and an adenoma histologically malignant was found. Patient discharged as incurable. The process pursued a malignant course for some time, but finally spontaneous resolution occurred.

[In 42 cases of multiple polypi collected by Quénu and Landel,[1] carcinomatous change developed in 20; this change almost always came on some years after the polypi had given signs of their presence, and histologic examination shows that a number of polypi undergo this carcinomatous change, the adenomatous growth and the developing carcinoma being sometimes visible in the same polypus. In 36 cases there were data for estimating the number of polypi: in 3 there were between 15 and 30; in 5 there were 30 to 50; in 25 there were several hundred; and in 3, several thousands. More than half the cases occur between the ages of sixteen and thirty-one years. In 37 cases there were 23 males and 14 females. In only 2 cases were the polypi limited to the colon; they may occur throughout the whole length of the colon, small intestine and colon, but are never limited to the small intestine. In a case of innumerable polypi of the small intestine recorded by Kanthack and Furnivall[2] there were 4 polypi in the sigmoid flexure. Collier[3] described multiple polypi, varying in size from a pigeon's egg to a pea, in the small intestine and stomach. In 4 out of Port's[4] 13 cases the condition appeared to be hereditary. Niewack[5] has recently reported cases showing a family predisposition.—Ed.]

Fibroma, Lipoma, Papilloma.—Apart from the polypoid growths of the intestinal mucosa which are often met with as the result of the changes set up by chronic catarrh and dysentery in the intestine, neoplasms of the bowel derived from the connective tissues, especially from the submucosa, are comparatively rare. Histologically, these tumors are, as a rule, lipomatous in structure, and are only in rare cases genuine fibromata. The former often originate from the appendices

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[1] Quénu and Landel, *Rev. de Chirurg.*, April 10, 1899, p. 465.
[2] Kanthack and Furnivall, *Trans. Path. Soc.*, vol. xlviii., p. 83.
[3] W. Collier, *ibid.*, vol. xlvii., p. 46.   [4] Port, *Zeitschr. f. Chirurg.*, vol. xvii.
[5] Niewack, *Annals of Surgery*, July, 1902, p. 104.

epiploicæ. Lipomata of these appendages frequently project into the peritoneal cavity. Occasionally lipomata of this kind become detached from the bowel by twisting or rupture of their pedicle, and are sometimes found free in the peritoneal cavity. Connective-tissue tumors of this kind may be hard or soft. Their consistence depends entirely on the proportion of firm fibrous connective tissue that enters into their construction. They may be attached to the intestine either by a broad base or by a pedicle. They may be spheric, pear-shaped, or polypoid. They vary greatly in size: sometimes they merely form slight elevations and protrusions; in other cases they grow to be as large as an apple, and in rare instances attain very much larger dimensions. Link observed a lipoma of the descending colon as large as a man's clenched fist. They occur most frequently in the colon and rectum (in about two-thirds of all cases), less frequently in the jejunum, and still less so in the ileum. On the whole, lipomata are scarce. In 1899 Hiller could find but 23 cases in the literature, and subsequent years have added very few.

[Shattock[1] described polypi of lymphatic tissue which were removed from the rectum of a boy aged four and a half years, in whom no evidence of lymphadenoma existed. The polypi were multiple and were found to involve the mucous membrane of the rectum as far as the finger could reach. They were sessile. The boy suffered from threadworms. Ball also met with another case of polypoid lymphatic growths.—ED.]

Myoma, Fibromyoma.—Benign neoplasms of the intestine arising from the muscular coat of the intestinal wall are very rare. A small minority of these tumors appears to originate from the submucous layer of the intestinal wall, probably from the muscularis mucosæ (so-called muscular layer of Bruecke). The majority of myomata and fibromyomata originate from the outer muscular coat of the intestine, and chiefly from the longitudinal muscular coat of the intestine. Boetticher and Lode have collected most of the cases that have been reported with some detail in the literature, and the reader should refer to their monograph for the histologic details of myomata and fibromyomata of the intestine. According to these writers, two chief forms of myomata of the intestine must be recognized: one form originates from local proliferation of the unstriped muscle-fibers of the muscular coat of the intestine. These growths are more or less spheric nodules, and usually grow toward the mucous lining of the intestine. As they increase in size they push the mucosa in front of them, but do not form solid connections with this layer of the intestine. Sometimes the growths become pedunculated, and the resulting tumor is called a polypoid myoma. In the other form the tumor forms a broad thick layer in the intestinal wall, so that the neoplasm is really a circumscribed thickening of the bowel-wall. This form develops from local hyperplasia of the longitudinal and circular muscular coats of the intestine. Certain differences are observed between these two forms as

[1] Shattock, *Trans. Path. Soc.*, vol. xli., p. 137.

regards their connections with the mucous layer, for whereas in the former tumors the mucosa remains freely movable over the myoma, in the latter form the mucosa and the submucosa become thickened and tightly adherent to the neoplasm. Fleiner has reported a case of the latter kind with great care and detail. In a third category of cases, which are certainly very rare, myomatous tumors of the intestinal wall do not grow toward the lumen of the bowel, but toward the peritoneal cavity. This form pushes the serosa outward, just as the other two forms push the mucous membrane of the intestine inward. Lode has described a myofibroma of the bowel as large as an adult fist—this is the largest tumor of this kind that has so far been described. Lode's myofibroma originated from the outer coats of the ileum and was attached to this portion of the bowel by a pedicle, the tumor itself hanging into the peritoneal cavity.

[Steiner[1] has collected a number of intestinal myomata, among which there were 19 submucous and 16 subserous myomata.—Ed.]

Angioma.—Angiomata of the intestine are exceedingly rare. These tumors may either occur as telangiectatic tumor-like masses or may form flat, vascular tumors, involving more or less extended portions of the bowel-wall. Treves is of opinion that the majority of these tumors are probably only very vascular fibromyomata. His view, however, is extreme, for genuine angiomata of the intestine have been described by some authors.

[A remarkable case of extensive lymphangiectasis of the small intestine—chiefly of the duodenum and jejunum—has been recorded by Allchin and Hebb.[2] The mucous membrane showed myriads of coarse villosities due to varicose lymphatic vessels.—Ed.]

CLINICAL FEATURES.

Many cases of benign tumors of the intestine run their course without producing any symptoms whatever, and are discovered only by accident after death. In other cases they produce a certain train of symptoms which are so indefinite and are compatible with so many different conditions that the existence of a benign neoplasm may not even be suspected. In a third series of cases the tumors are diagnosed, even though they do not produce any definite group of symptoms.

In comparatively rare cases benign neoplasms of the intestine do produce certain functional disturbances which are usually extremely indefinite. The individual symptoms are in no way typical, and the symptoms, when viewed as a whole, are not such as to permit us to draw any conclusions as to the nature or the situation of the neoplasm that produces them. In benign forms of intestinal tumor, moreover, the functional disturbances produced are quite irregular and present no constant features. The only course, therefore, in describing the clinical features of this form of tumor is to deal *seriatim* with the symptoms

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[1] Steiner, *Beitrag z. klin. Chir.*, 1898, vol. xxii.
[2] Allchin and Hebb, *Trans. Path. Soc.*, vol. xlvi., p. 220.

which are occasionally observed. The leading symptoms are the following:

The most important sign is hemorrhage from the intestine, which may occur in all forms of vascular adenomata, fibromata, and myomata. The most copious forms of intestinal hemorrhage, however, are seen in angioma. Whenever an attack of enterorrhagia occurs in an individual who was previously in perfect health, who had never presented any symptoms of intestinal disease before this time, and in whom no manifest cause for this attack of bleeding can be discovered, it must always be borne in mind that an intestinal tumor of the kind described may possibly be the cause of the hemorrhage. This applies particularly to sudden hemorrhages from the bowels in children, in whom polypi are the most frequent cause of primary enterorrhagia. It need hardly be mentioned that when these hemorrhages are frequently repeated and are very violent, the well-known consequences of loss of blood will all supervene.

The authors who have reported cases of polyposis intestinalis adenomatosa all speak of diarrheic evacuations consisting of mucus and blood. So few cases of this disease are on record that it is quite impossible to decide to what extent the adenomatous growths themselves produce this form of diarrhea and are responsible for the appearance of blood and mucus in the stools. It is quite possible that in these cases certain catarrhal changes of the mucosa are present and produce the abnormal stools, or that the development of carcinoma—a frequent complication of these cases—has something to do with it.

A second group of symptoms is produced by obstruction or narrowing of the intestinal passage.

Afezou, quoted by Treves, reported a very interesting observation of this kind: The patient was a woman of eighty-two years, who for many years had suffered from indigestion, paroxysms of colic, and constipation with intercurrent attacks of diarrhea. Finally, after this condition had persisted for many years, the constipation became so obstinate that the bowels could be evacuated only by enemata. One day, immediately after an examination of the intestine had been performed, a soft, polypoid lipoma was passed in the stools. All the abdominal symptoms disappeared immediately, and the action of the bowels became perfectly regular.

The lumen of the bowel may be obstructed in a number of different ways: The tumor may be so large that it mechanically occludes the lumen of the intestine. This chiefly occurs when the tumor is situated in the region of the ileocecal valve. Sometimes a flat, annular adenoma involves the whole circumference of a certain portion of the intestine and produces stenosis of its lumen. This is most frequently seen in the region of the rectum. Finally, a pedunculated tumor of the bowel, or in rare cases a myoma attached to the bowel-wall by a broad base, may cause intussusception of the intestine. This accident occurs most readily in polypi of the small intestine, and very rarely in polypi of the large intestine. (The reader should refer to the section on Intussusception of the Bowel for the details of these interesting accidents.)

Benign tumors of the intestine produce constant and truly local

symptoms only when they are situated in the rectum, and particularly when they are situated in the lowest portion of this division of the bowel. In the first place, they may, like tumors situated in any other portion of the intestine, produce hemorrhages and the passage of mucus. The passage of mucus is a direct result of the catarrh produced by these tumors. They may also, like tumors situated elsewhere, interfere with defecation. In addition, however, they produce tenesmus, and may also produce the sensation of a foreign body impacted in the rectum. The last two symptoms will naturally lead the medical man to make a digital examination of the parts. Tumors of this kind, when situated low down in the rectum, can be diagnosed without difficulty. A direct and positive diagnosis is nearly always possible, for the tumor can either be felt or may occasionally even protrude from the anus, provided the pedicle is sufficiently long. Sometimes these tumors protrude spontaneously—namely, when the abdominal pressure is increased voluntarily by the patient. It is not necessary that fecal matter should be evacuated at the same time. A pedunculated tumor of the rectum which projects from the rectum in this way may either slip back spontaneously without external help or occasionally may be torn off. Only in very exceptional cases up to date have there been benign intestinal tumors that were palpable through the abdominal wall, like the lipoma described by Link. In this case the nature of the tumor remained in doubt, but its consistence and smooth surface, as well as the health of the patient, were strongly against malignancy.

COURSE AND TREATMENT.

Benign tumors of the intestinal canal may remain perfectly latent and give the patient no trouble during life. In other cases they may produce more or less disagreeable symptoms for a number of years, and then be passed by the rectum spontaneously. This occurs particularly in the pedunculated form of benign neoplasm of the intestine whenever the pedicle becomes too long and is consequently torn in two. Again, in other cases benign tumors of the intestine may produce severe and even fatal consequences, among which enterorrhagia, obstruction of the bowel, and intussusception may be especially mentioned. Finally, it must always be remembered that multiple adenomata of the intestine constitute a favorable soil for the development of carcinoma.

Direct treatment can be considered or even discussed only when the tumors are situated in the rectum ; whenever a benign tumor is found in this position it should be removed by surgical interference. In cases in which numerous tumors are discovered in the lower portions of the rectum, operative interference, to judge from past experience, offers very little chance of a cure, even though the tumors are readily accessible, for it is a well-known fact that these multiple tumors are almost exclusively adenomatous in character, so that the probabilities are that there are other tumors in other parts of the intestinal tract. The rectum, therefore, may be cleared of these tumors, but the existence of

many others higher up in inaccessible portions of the intestine must be expected. When no tumor of the intestine can be felt, or when its presence is merely suspected, the only available treatment is symptomatic —measures to combat individual symptoms like hemorrhage, etc. [After removing as many polypi as possible from the rectum, Quénu and Landel suggest that the ileum should be inserted into the rectum. Ritter von Karajan[1] records a case of a man aged twenty-three who had vomiting, irregularity of the bowels, and a number of tumors palpable in the abdomen. At a laparotomy one and one-half years after the commencement of the illness the small intestine was distended, an intussusception was found in the ileum, and tumors in the ileum, jejunum, and duodenum. The intestines were opened longitudinally in five places and eight polypi removed. The patient recovered perfectly.—ED.]

INTERNAL STRICTURE OF THE INTESTINE (Strictura Intestinalis Interna).

TREVES' definition of a stricture of the intestine is that form of narrowing of the bowel which is produced by changes in the layers constituting the intestinal wall.

Strictures of the intestine may be divided into two main groups : those due to cicatricial contraction and those due to neoplasms. The latter narrow the lumen of the bowel, either in the same way that cicatrices do or else by blocking it. Cancerous growths act in both ways, while benign tumors act only by blocking the lumen. Of late years attention has been directed to another form of stricture, of which I cited a typical case in the first edition of this work, under the title, " Stricture from Chronic Inflammatory Infiltration of the Intestinal Wall." It is similar to the process since frequently described under the name " cecal tuberculosis."

As carcinoma of the intestine, apart from producing the mechanical effect of enterostenosis also produces a series of other important phenomena, it has been already described in a special section, to which the reader should refer for a description of the special and characteristic clinical picture produced by this form of neoplasm. Enterostenosis produced by benign neoplasms obstructing the intestine has already been dealt with. It only remains, therefore, to consider the cicatricial form of stricture of the intestine, and, in addition, the form of stricture produced by chronic inflammatory processes going on in the intestinal wall.

CICATRICIAL STRICTURE.

Etiology.—Cicatricial stricture of the intestine is, in the overwhelming majority of cases, the direct result of some form of ulceration in the intestine. As a matter of fact, ulceration of the intestine may be regarded as almost the only cause of this form of stricture of the

[1] Ritter von Karajan, *Wien. klin. Wochenschr.*, March 2, 1899.

bowel. It is only in exceptionally rare instances that any other cause produces this lesion.

It was shown, in a preceding section, that though there are a great many different forms of ulcer of the intestine, only a few ever lead to the formation of cicatrices and secondary stricture of the intestine. The most important forms of ulcer in this respect are the tuberculous and the stercoral ulcers of the bowel. In rare instances stricture follows round ulcer of the duodenum, dysenteric ulcers of the intestine, catarrhal follicular forms of ulceration, and some syphilitic ulcers of the bowel. In exceedingly rare instances stricture of the intestine may develop after typhoid ulceration. I do not wish to maintain that it is impossible for other forms of ulcer of the bowel to lead to the formation of constricting cicatrices, but only to point out that no evidence is forthcoming that they do, and that if stricture ever does follow other forms of ulceration, it must be extraordinarily rare.

Further, before entering on the description, it may be pointed out that a stricture of the intestine is often found in the course of a post-mortem examination, in which even the most careful examination of the morbid conditions fails to explain its origin. This subject will be referred to again in a subsequent paragraph.

Formerly the belief was prevalent that cicatricial strictures of the intestine following dysenteric ulceration were the commonest. This can be readily understood when the special character of the morbid lesions of dysentery are borne in mind. Woodward, in his classic work on *Diseases of the Intestines*, first expressed the rather startling opinion that the facts of the case do not support this view. His position at the time was particularly surprising, as all the text-books repeatedly reiterated the older view given in the first sentence of this paragraph. Immediately after the American War of Secession an enormous number of cases of dysentery came under observation, and among all these cases not a single instance of stricture of the intestine following the development of dysenteric ulceration and cicatrization of the bowel was found. Woodward admitted that possibly an isolated case might have escaped detection here and there, but even granting this, dysenteric cicatrization of the bowel must be considered an exceedingly rare event as compared to the enormous number of cases of dysentery in which this sequel did not occur. Among the United States troops there were 287,522 cases of dysentery, and of these, 28,451 suffered from the chronic form of the disease. There were 9431 deaths in all, and of these fatal cases, 3855 were chronic cases. Woodward further studied the complete literature of the subject, and failed to find any appreciable number of cases of dysentery reported by other authors in which stricture of the intestine developed as a sequel. Woodward's excellent work is characterized by a degree of completeness and care that is almost surprising, and there can be no doubt that his investigations and his review of the literature were performed with the greatest care. Nevertheless, he places himself in direct opposition to the ordinarily accepted view, and makes the definite statement that

cicatricial stricture of the intestine from dysenteric ulceration of the
bowel must be considered a rare event. My own experience agrees
with Woodward's views. I can only remember one case under my
observation in which this occurred; here, moreover, carcinoma devel-
oped on the basis of the dysenteric scar. Treves, in his work, pub-
lished an illustration of another case of this character, and a search
through the whole literature of the subject only shows here and there
an isolated case of cicatricial stricture of the intestine following dys-
enteric ulceration which can be considered as satisfactorily established.
The extreme rarity of the cases makes it impossible to maintain the
statement that cicatricial strictures of the intestine frequently follow
dysentery.

I believe that stercoral ulcers of the intestine frequently undergo
cicatrization, and that a large percentage of cases of stricture of the
intestine are due to this process. From a study of the exact patho-
genesis of these ulcers and their consequences it can readily be under-
stood that they frequently produce stricture of the bowel. These
ulcers are usually annular and involve the whole circumference of the
bowel, and being benign in character, they have a great tendency to
heal, so that all the conditions are favorable for the production of con-
tracting cicatricial tissue.

There is a considerable diversity of opinion as to the rôle of tuber-
culous ulcerations of the intestine in the production of cicatricial stric-
ture of the bowel. Not very long ago the statement was universally
accepted that the stenosis of the bowel that follows healing of a tuber-
culous ulcer of the intestine only rarely leads to considerable degrees
of stricture, and only then when, at the same time, there is some con-
stricting peritonitic process in the area of the bowel corresponding to
the ulcer. This was the view expressed by Leichtenstern. Quite
recently, however, König has called attention to the fact that constrict-
ing tuberculosis of the intestine occurs much more frequently than is
ordinarily assumed. From personal experience I can indorse König's
view. I have often seen cases of enterostenosis in which examination
of the diseased portion of the intestine after excision by operation or
postmortem showed that the stricture was due to a cicatricial stenosis
of the intestine following tuberculous disease of the intestinal wall.
In many of these cases the complete clinical picture of enterostenosis
was produced. As an example, reference may be made to a case
described at great length on page 423. It is important to remember
that a careful study of the history of the illness, and even of the clinical
signs in many of these cases, may fail to direct our attention to tuber-
culous affections of the bowel. The case quoted above is a good example
of this. I have also seen a number of other patients who presented
the clinical picture of chronic stenosis of the small intestine, but in
whom no evidence whatever could be found of tuberculous involvement
of the intestine. In one of these cases, nevertheless, I found 3, in
another 5, and in another case 7 or 8 strictures due to tuberculous
cicatrices. In the last case the 7 or 8 strictures were distributed over

the jejunum and the ileum. Only 2 of them, however, were so pronounced that they led to secondary dilatation of the bowel. In addition to these cicatrices that led to stricture there were a number of other scars in the basis of tuberculous ulcers which did not produce stricture of the intestine. Litten reports a very remarkable case of stricture of the intestine following cicatrization of tuberculous ulcers. In his case he discovered 6 marked strictures of the ileum which were only $\frac{1}{4}$ to $\frac{1}{2}$ m. apart; between these six constricting bands the intestine was dilated into 5 large sacculated pouches filled to bursting with a large amount of semiliquid fecal material. Patients with stenosis of the intestine due to the cicatrization of some tuberculous lesion of the bowel are not necessarily always pale and emaciated—König, *e. g.*, found that this was not the case in the few instances that he observed. Another important point to remember is that tuberculosis may be completely absent in all other organs, or, if it is present, may be so slight that it cannot be detected and demonstrated. It is quite possible that many strictures of the intestine are in reality due to the cicatrization of tuberculous ulcers, and it is quite possible that many of the cases of enterostenosis whose etiology is considered uncertain or obscure belong to this category.

[C. Bernay [1] has collected 70 cases of tuberculous stricture of the small intestine. Tuffier [2] divides tuberculous stricture of the small intestine into three distinct forms : (1) The purely cicatricial form due to healing of a tuberculous ulcer ; (2) that due to contraction of tuberculous granulation tissue developing in the submucous coat without any ulceration of the mucous membrane : this leads to a diaphragm-like stricture. Tuffier believes that non-malignant strictures are usually tuberculous ; (3) the rare hypertrophic form of tuberculous stricture, of which Bernay has collected 8 cases. It is a fungous enteritis with hypertrophy and tuberculous infiltration of the muscular coat ; there is a secondary infection superimposed on the tuberculous ulceration. The thickening leads to stenosis and may be felt through the abdominal wall. In a case of Tuffier's a tumor as large as a tangarine orange was felt near the right iliac fossa, and a tuberculous tumor of the small intestine was successfully diagnosed and removed, with recovery.

Operative treatment of tuberculous strictures of the bowel has been performed in 45 out of Bernay's 70 cases, and in 37 of them the affected part of the bowel, nearly always near the lower end of the ileum, was removed. Erdheim [3] operated on 5 cases of multiple tuberculous strictures of the small intestine on adults affected with tuberculosis elsewhere—*e. g.*, phthisis. In a portion of ileum containing four tuberculous strictures removed by Mayo Robson,[4] a gall-stone was found entangled in a pouch between two of the strictures.—ED.]

It is a well-known fact that many forms of ulcer of the intestine do

[1] C. Bernay, *Thèse de Lyon*, 1898–99.
[2] Tuffier, *La Presse Médicale*, February 21, 1900.
[3] Erdheim, *Wien. klin. Wochenschr.*, June 25, 1900.
[4] Mayo Robson, *Trans. Clin. Soc.*, 1902, vol. xxxv., p. 61.

not lead to stricture of the bowel from cicatrization. In some of the forms of ulceration of the bowel this can readily be understood. In the septic, uremic, embolic-thrombotic, leukemic, and a number of other forms of ulceration of the intestine the primary diseases cause the death of the patient so soon that neither cicatrization of these ulcers nor stricture of the bowel can supervene. Typhoid ulcers only rarely produce stenosis of the bowel, and it is difficult to understand why ulceration of the bowel in typhoid fever should lead to enterostenosis only in such exceptional cases. On reviewing the literature of the subject, it will be found that only very few cases are on record in which it could positively be determined that the stricture of the bowel was due to the cicatrization of a typhoid ulcer. Klob, for instance, has reported a case of this kind. Hochenegg quite recently has reported another case, but it is not so well established as that of Klob, and must be considered very doubtful. In attempting to explain this peculiarity, for instance, that typhoid ulcers are frequently very small; that, as a rule, they develop parallel to the longitudinal axis of the bowel, and consequently involve only a very small portion of the circumference of the intestine in any one spot. This alone, however, does not explain the rarity of stenosis, for in many cases typhoid ulcers involve large areas of the inner surface of the bowel, extend deeper down into the tissues, and are circular or annular in position. Catarrhal follicular ulcers of the bowel also rarely lead to stricture of the intestine. The relative rarity of this sequel in ulcers of this kind may possibly be explained by the fact that this form of ulceration does not, as a rule, produce much loss of tissue, and that if it does, death rapidly supervenes. Peptic ulcer of the duodenum is comparatively rare in itself, so that it is not surprising that strictures of the duodenum are not very frequent after peptic ulcer of this portion of the bowel. As compared to the absolute frequency of this form of ulceration, stricture of the duodenum from peptic ulcer is not so very rare. (For stricture of the intestine following cicatrization of syphilitic ulcers the reader should refer to p. 263.)

Cicatrization of intestinal ulcers is, properly speaking, the only cause of stricture of the intestine; other etiologic factors that might produce this lesion need hardly be considered. It is true that Treves has collected 4 cases from the literature in which a very severe form of stricture (3 in the ileum, 1 in the duodenum) developed in a portion of the bowel that had at one time been incarcerated in a hernia. In all these 4 cases the cicatrix developed after ulceration or circumscribed gangrene of the strangulated loop of intestine had occurred. The stricture, particularly after the occurrence of inguinal and femoral hernia, produced symptoms of obstruction in from one month to several years after the operation for hernia. There is another class of cases that is very rare, in which a piece of intestine becomes intussuscepted during invagination, sloughs off, and in this way ultimately produces stricture of the bowel.

Finally, trauma, such as a blow, a fall, sudden impact on the abdomen, or being run over, may sometimes injure the intestine and

lead to cicatricial stricture. Schloffer has succeeded in finding in the literature up to 1900 only 10 undoubted cases, including 1 of his own. All cases involved the small intestine only. This coincides with the experience that subcutaneous intestinal lesions occur in the small intestine in most cases. The mode of origin is, doubtless, cicatricial stricture following a wound which did not entirely divide the intestine. Other methods are conceivable, for which we refer to Schloffer's careful experimental study on traumatic stenoses of the intestines. Pouzet, for instance, reports an interesting case of stenosis of the bowel due to circumscribed peritonitis following trauma. His patient was a man who had been perfectly healthy up to the time of the accident. Six months before his death he fell on the abdomen ; two months before his death he suddenly developed violent attacks of colic, and later paroxysms of vomiting. He died with symptoms of collapse. At the autopsy it was found that the jejunum was stenosed 15 cm. below the duodenum. The stenosis was produced by a whitish ring of connective tissue half a centimeter wide, which had produced such extreme narrowing that an ordinary sound could be passed through the stricture only with difficulty.

Another form of stricture of the intestine which requires special description here is stricture of the rectum. I think stricture of this part of the intestine should be considered separately, because the etiologic factors which cause stenosis of the rectum are somewhat different in character from those producing stenosis of other portions of the bowel. From an examination of the facts recorded as to the etiology of strictures of the rectum it appears that this condition is much more frequent in women than in men, in the remarkable proportion of 190 women to 25 men. It must, of course, be remembered that cicatrization of the bowel following dysenteric ulceration plays some part in the production of these strictures of the rectum, while tuberculous ulcers are very insignificant in this respect.

[It is quite possible that tuberculosis is a more frequent cause of stricture of the rectum than is usually believed ; it is probable that they are often regarded as syphilitic. Sourdille[1] showed that 5 cases of rectal stenosis, of the type usually spoken of as syphilitic, were really tuberculous, and Lapointe[2] has added 4 more, making 9 of stenosing tuberculous proctitis.—ED.]

Cicatrices developing from stercoral ulcers must also be considered, and, in addition, all the forms of stricture of the rectum that develop after cicatrization of hemorrhoidal ulcers, and, finally, those important forms of stricture which may follow two special traumatic causes. One of these causes are the very varied surgical operations performed for a variety of rectal diseases—for instance, removal of a prolapsed portion of the rectum, destruction of hemorrhoids, etc. ; the second causes are the manifold injuries to which the rectum is exposed from the patients' unskilful manipulation of the nozzle of enema-syringes. Included in the same category are those cases of trauma of the rectum produced by

[1] Sourdille, *Arch. gén. de méd.*, 1898.   [2] Lapointe, *Thèse*, Paris, 1897.

the intentional introduction of foreign bodies. Occasionally a periproctitic abscess may perforate into the rectum and thus lead to the formation of a scar, and subsequently a stricture.

[Obstetric trauma is a cause of importance : the child's head may lead to extensive damage of the mucous membrane of the rectum. F. C. Wallis[1] believes that the commonest cause of rectal stricture is septic ulceration, and has drawn attention to the fact that patients with rectal ulceration are especially prone to get attacks of acute synovitis.—Ed.]

Syphilitic strictures, or at least those forms of stricture that are considered syphilitic, are almost exclusively limited to the rectum. In order to avoid repetition I refer to pages 262 and 263 for a description of syphilitic ulcers of the intestine. Finally, in rare instances stenosis of the rectum may depend on excessive development of the plicæ transversalis of the rectum, or the folds of mucous membrane which are arranged in the same way as the valves of the veins. In cases of this kind they are apt to form pockets in which fecal matter accumulates; they thus become enlarged and cause narrowing of the lumen of the bowel.

We shall make only brief mention of congenital occlusions of the intestines, as they are of surgical or anatomic interest only. For further description we refer to Leichtenstern's work and the literature mentioned by him. We confine ourselves to the following remarks (from Leichtenstern) : Congenital occlusion of the rectum is the most frequent form, and occurs either as agenesia ani, in which there has been absolutely no anus-formation, or as atresia ani. These anomalies were observed three times in 66,654 new-born children. Congenital atresia of the colon or small intestine is still more infrequent; of 375 collected cases, 10 were of the colon and 74 of the small bowel. The atresias of the colon are almost exclusively situated in the iliac flexure; those of the small intestine at the ileocecal valve and duodenum. The most frequent cause of congenital atresia of colon and of the small intestine is fetal peritonitis.

Anatomy.—The most common seat of stricture of the bowel following cicatrization of an intestinal ulcer is in the large intestine. Strictures of this kind in the small intestine are comparatively rare. This account is limited to those forms of stricture that follow cicatrization of intestinal ulcers, as strictures due to any other factors are quite insignificant in comparison. (The carcinomatous form of stricture of the intestine, as has been already stated, is especially excluded from consideration here. Treves has calculated the relative frequency of cicatricial stricture of the large and the small intestine, and has estimated that the proportion is approximately as 6 is to 1. This preponderance of stricture of the large intestine is due to the causal factors responsible for this lesion. It has been seen that dysenteric, stercoral, and syphilitic strictures are exclusively localized in the large intestine; attention has also been called to the comparative frequency of traumatic strictures of the rectum and to the fact that catarrhal-fol-

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[1] F. C. Wallis, *Brit. Med. Jour.*, 1900, vol. i., p. 1002.

licular ulceration occurs quite frequently in the large intestine and may lead to stricture. Stricture of the intestine following tuberculous ulcers, on the other hand, in the great majority of cases, occurs in the small intestine, particularly the ileum. In fact, I might almost say that in any given case of chronic stenosis of the small intestine in which the general symptom-complex does not definitely and distinctly point to some other cause, stricture of the bowel following tuberculous ulceration and cicatrization should always be thought of first. Stricture of the intestine following cicatrization of a peptic ulcer naturally occurs only in the duodenum.

From a study of the frequency with which stricture occurs in different parts of the large intestine it appears that the rectum is the most frequent seat of stricture, and more especially the lowest portion, immediately above the anus. After the rectum, the sigmoid flexure is the most frequently involved, and next in their order of frequency the descending colon, the splenic flexure, and the hepatic flexure. Stricture of the ileum occurs almost exclusively in its lowest and middle portions.

From statistics collected by Reach we abstract the following data, but must first call attention to the fact that Reach's work includes multiple carcinomatous stenoses. He has collected 91 cases. A case of Homén showed 30 strictures, all in the small intestine, and of syphilitic origin; a carcinomatous patient of Küttner's showed 22—18 in the small, 4 in the large, intestine; Strehl's case, with 15, is next—14 in the ileum and 1 in the hepatic flexure of the colon, all of tuberculous origin. As regards localization, the small intestine was affected 55 times, the colon alone only 10 times. In the remaining cases both large and small intestines were involved. Etiologically, tuberculous cicatrization-stricture ranks first, with 42 out of 91 cases; this confirms my statement in the preceding edition, that multiple cicatricial strictures in particular are of tuberculous origin. Next come the carcinomatous cases, with 16, and the syphilitic, with 12.

The character of the stenosis varies greatly according to the extent of the ulceration and the direction in which the ulcer develops. When the ulcer grows in a direction parallel to the longitudinal axis of the bowel, and when its surface is irregular, the resulting cicatrix can at best produce an insignificant degree of stenosis. In many instances the scar tissue resulting from such a lesion merely leads to folding and distortion of the intestinal wall, without, at the same time, causing narrowing of the intestinal lumen or in any way interfering with the functional activity of the bowel-wall. The most severe degrees of stenosis of the bowel lumen are naturally produced by annular ulcers which involve the whole circumference of the bowel.

The length of the constricted portion of the bowel may vary: as a rule, the stricture is short. In cases of this kind the intestine, seen from the outside, presents a peculiar appearance, looking as if a ribbon or piece of string had been tightly tied around it in one place. In cases in which dysenteric ulcers of the bowel undergo cicatrization, the intestinal tube may be found rigid throughout long areas.

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The external surface of the intestine is often covered with deposits of peritonitic exudate, so that the whole bowel is thickened. In many other cases there are adhesions between the affected loop of intestine and other loops of the bowel, or with adjacent organs, as the spleen, the liver, etc. These peritoneal adhesions often cause additional constriction of the bowel from without, and thus stenosis, produced, in the first instance, by the scar on the inner surface of the bowel-wall, is further accentuated.

Histologic examination of the mass of cicatricial tissue shows that it consists essentially of connective tissue. The color of these scars is usually slate-gray, owing to the presence of pigment. In many cases the stricture produced by the cicatrix is not in itself very marked, but the caliber of the bowel may be greatly narrowed by secondary processes. Folds of mucous membrane in the immediate vicinity of the cicatrix that have remained intact, or excrescences of hypertrophic mucous membrane in the form of polypous protrusions, may cause valve-like closure of the stenosed portion of the bowel lumen. This peculiar secondary form of enterostenosis is called valvular stricture, and has been described particularly in cases of stricture of the intestine due to cicatrization of dysenteric ulcers. Associated with strictures due to cicatrization of tuberculous, catarrhal, follicular, and dysenteric ulcers, other parts of the intestine often contain ulcers of the same nature, but of later date. They must be carefully distinguished from the secondary ulcers frequently seen in the dilated loop of intestine immediately above the stricture. These secondary ulcers are decubital in character, and always develop later in the disease—that is, after cicatrization and stricture of the bowel have occurred. Bayer operated for a stenosis in the middle of which a deep, circular constriction was found; this narrowed the intestine to the size of a quill. It was surrounded by hard, knobby protuberances, so that the condition at first simulated carcinoma, but examination revealed tuberculosis.

The important changes which occur in the intestine immediately above the stricture have been described in a previous paragraph (p. 345), to which the reader should refer for the details. Attention may again be called to a peculiar phenomenon that is occasionally seen in multiple tuberculous strictures of the small intestine—namely, the peculiar sacculated dilatation of those portions of the small intestine that are situated between the different stenoses.

Clinical Features.—It is quite unnecessary here to enter into a detailed description of the symptoms produced by stricture of the bowel *per se*, as a very complete description has been given in two preceding sections—namely, on the symptomatology of carcinoma of the intestine and on the symptomatology of stenosis of the bowel in general.

In any given case of stenosis of the bowel it remains to be determined whether or not the symptoms are due to a stricture (according to the definition formulated above). Sometimes this diagnosis is quite easy; in other cases the diagnosis is only tentative, and remains uncertain, and again in other cases it is simply impossible.

It is, of course, easy to recognize the existence of the various forms of strictures in the lowest parts of the rectum. All that is needed is a digital exploration of the parts, and, as a rule, the medical man will thus be able to determine the exact character of the process.

In any given case of stricture of the bowel situated high up, the first point to determine is whether or not it is due to carcinoma. The chief guides to the diagnosis of the carcinomatous character of an intestinal stricture are, in the first place, the history of the case and, most important, the presence or absence of a tumor. The reader should be on his guard against possible errors in this respect (compare the case quoted on p. 423). Other clinical features which may be of use in the diagnosis of carcinoma are the general constitutional condition of the patient, and, in individual cases, the character of the stools.

The only means by which any definite and direct information as regards the existence of a cicatricial stricture of the bowel can be obtained is careful study of all the points in the history of the patient. Stress need hardly be laid on the fact that the history of any given case may be very misleading and lead to conclusions which are entirely erroneous. A diagnosis made from the history is, at best, a lucky guess. In an exceptional case a speculative diagnosis of a stenosis of the duodenum following an ulcer may turn out correct, or it may be rightly assumed that the case is one of stenosis of the intestine following cicatrization of a dysenteric, tuberculous, or decubital ulcer. Even in cases where a diagnosis of stricture following cicatrization of an ulcer has been made, operation or autopsy may show that while such a scar was actually present in the bowel, it had undergone changes and given rise to the development of carcinoma in the affected area. Again, a patient with symptoms of stenosis of the bowel and a history of long-standing constipation may be diagnosed as a case of stricture of the bowel due to the cicatrization of a decubital ulcer. But, as the result of treatment, it may become quite clear that in reality the patient is suffering from nothing more nor less than simple fecal accumulation. Let me again emphasize the great importance of visible abnormal peristaltic movements of the bowel, and the rigidity of certain loops of intestine visible through the abdominal wall, which have already been described at great length in the general part of this work. Whenever abnormal peristalsis and stiffening of certain loops of intestine are visible through the abdominal walls, a genuine stricture is usually present; while in their absence the diagnosis should incline toward coprostasis. The reasons for this differentiation will be found in the section alluded to.

As regards the diagnosis of stricture of the intestine from cicatrization of tuberculous ulcers, the reader should refer to what has been said before. Let me repeat here that in any case of stenosis of the small intestine in which no satisfactory cause for stenosis is forthcoming, cicatrization following tuberculous ulceration of the bowel should always be thought of. The diagnosis of cicatricial stenosis following a strangu-

lated hernia or external trauma can usually be made without difficulty from the history of the case.

Another very difficult matter to decide is whether the stricture of the bowel is the result of some abnormal process going on in the intestine or whether it is due to constriction of the bowel from without by peritoneal adhesions. Fortunately, this differential diagnosis is not of the least importance so far as the actual treatment of these cases is concerned.

TUMOR-LIKE TUBERCULOSIS OF THE CECUM.

As already stated, tuberculous processes show a preference for certain parts of the intestine—the lower part of the ileum and cecum. This is doubtless due to anatomic and physiologic peculiarities of the parts. It is here that prolonged stagnation of contents begins. Therefore, if the contents contain (swallowed) tubercle bacilli, ample opportunity is afforded them to exert their action on the intestinal wall. To this we should perhaps add peculiarities in the circulation and in the histology of the mucosa, which here forms a transitional stage between the villous formation of the small intestine to that of the colon.

Tuberculous ulceration in the intestinal parts just mentioned has been recognized for many years. But it is only since about 1890 that intra-abdominal surgery directed attention to a particular form, in which the tuberculosis may appear in the cecum. Durante, Billroth, and Hartmann and Pilliet, followed by numerous other investigators, stated definitely that tumor-like growths occur in the cecum, simulating carcinoma both clinically and macroscopically, but that, upon careful histologic examination, these proved to be tuberculous formations. In 1898 Conrath was able to collect 81 cases operated upon. As illustration I cite the following case from my own experience : A colleague of mine had had no intestinal trouble during the many years I knew him. About a year and a half before his death constipation set in, which he was compelled to relieve artificially. If he allowed constipation to occur, three or four severe colicky attacks appeared simultaneously, of a nature suggesting enterostenosis. Despite exhaustive examination, no tangible result was reached. Only about two months before death a round, hard tumor about as large as a walnut was felt in the ileocecal region. The growth was painless and readily movable. Although nutrition was but slightly impaired, all the patient's colleagues who examined the growth considered it to be in all probability a carcinoma. The rest of the body showed nothing save two or three hard growths the size of a bean, which had existed for many years. Operation was performed, but, unfortunately, the patient died three weeks later. The part of intestine removed by the operation, embracing the region of the ileocecal valve, showed a very hard tumor about the size of a small hen's-egg. It was funnel-shaped, turned downward ; the lumen of the resected part was narrowed to the size of a lead-pencil, and showed no sign of ulceration. The pathologic anatomist at first sight considered the growth a carcinoma, but histologic examination yielded

the startling result that the characteristics of a malignant growth were entirely lacking. The appearance was more of a chronic inflammatory proliferation, in which giant-cells and tubercle bacilli were demonstrated.

Anatomy.—The affection appears tumor-like, and, at times, as in my case just cited, may convey, macroscopically, the clinical and anatomic impression of ordinary malignant neoplasms. But usually the growth consists of nothing more than the greatly thickened, hard, rigid wall of the cecum. When of the former sort, the tumor is sharply confined; when of the latter, it generally disappears gradually toward the ascending colon and the ileum. In both forms stenosis of the lumen may occur. In Billroth's and in my case the lumen was narrowed to the size of a quill. The stenosis is usually greatest at the ileocecal valve.

To repeat, the intestine may sometimes show a circumscribed swelling; at others, a series of irregular, segmented convolutions, villous or knotty, and again a more regular, diffuse thickening. The mucosa over the circumscribed tumor may be in a perfectly normal condition, but generally it has become affected by new growths. Closer examination reveals numerous small or minute, confluent tubercles, which have developed chiefly in the mucosa and subserosa. They are characterized by a compact, small-celled infiltration, resulting sometimes in a very close simulation of sarcoma. Occasionally a few giant-cells and even tubercle bacilli are found. We cannot go further into the histology of the subject, and will only say that the true tumor forms show the characteristics of tumor-like tuberculosis, as described by Askanazy in various organs—larynx, nose, muscles, heart, lymphatics— viz., complete absence of any tendency toward fusion and firm consistence in consequence of fibrous metamorphosis.

The regional lymphatics are usually also diseased; in consequence of adhesive peritonitis, coalescence with various juxtaposed organs occurs.

Clinical History.—The first symptoms of cecal tuberculosis are frequently of a very general nature, such as functional digestive disturbances, anorexia, belching, constipation or diarrhea (the last at times bloody), or both alternately. Sometimes painful sensations early direct attention to the ileocecal region. At other times patients are suddenly attacked with pain, such as may occur in gradually progressing stenosis, which may come on in the midst of an apparently normal condition of the intestines. Then, in the majority of cases, the stenotic symptoms develop gradually more and more.

At a stage favorable for diagnosis the malady is evidenced first by the appearance of a palpable swelling in the ileocecal region. It is to be remembered that this focus of resistance or this tumor may be readily discernible on palpation, even at the time of the first paroxysm of pain; in other words, it had already existed for some time, but without causing any functional disturbance. As soon as the ileocecal tumor has become palpable, the question of its nature arises. A large number of tumors will come up for consideration: carcinoma, sarcoma,

perityphlitic exudate remnants, invagination tumors, actinomycosis. The most natural thought on the part of the diagnostician will be carcinoma, with sarcoma next. The differentiation between cecal carcinoma and tumor-like cecal tuberculosis is by no means easy, often remains undecided, and often is impossible. Age is of subordinate importance, as carcinoma of the intestine occurs not infrequently in youth. More important in the diagnosis is the existence of pulmonary tuberculosis and the previous history or observation of a preceding diarrhea. The absence of both, of course, in nowise contraindicates tubercle. Obrastzoff attaches great importance to the presence of tubercle bacilli in the stools (for this see p. 270). He adduces also the following differential points : (1) In carcinoma the intestines themselves are not palpable, only the tumor being felt, with edges sharp, as if cut off. On the other hand, in cecal tuberculosis the intestine itself is in its greatest part palpable ; the walls appear thickened and infiltrated, the infiltration disappearing very gradually, but the latter occurs also in the sarcoma. Conversely, cecal tuberculosis, as in my case, already cited, may be felt as a sharp, circumscribed tumor. (2) In carcinoma stenosis occurs early, but it may also occur early in cecal tuberculosis. For the rest, the question of differential diagnosis, though of great theoretic value, is as yet in an unsatisfactory state. In cases in which our only therapeutic agent—operation—can be considered—that is, where there exists no advanced tuberculosis—the question is most quickly decided by operation.

[Lartigau [1] suggests that most of the cases of resection of the cecum for malignant disease in which no microscopic examination was made and remained cured were in reality chronic hyperplastic tuberculosis of the cecum. Conrath [2] and Itié [3] have given comprehensive accounts of the surgical aspects of chronic hyperplastic tuberculosis of the intestine.

The editor has given a description of hyperplastic tuberculosis of the intestine in the section on Tuberculous Ulceration of the Intestine.—ED.]

EXTERNAL PERITONITIC CONSTRICTION AND ADHESIONS OF THE BOWEL (Constrictio et Adhesio Intestinalis ex Peritonitide).

UNDER this heading all those cases of constriction of the bowel are grouped in which the passage of the intestinal contents is interfered with or obstructed by changes in the lumen or in the normal course of the intestinal canal, depending on chronic local peritonitis without strangulation.

It is only occasionally that a process other than peritonitis causes constriction of the intestine. For example, Küttner describes multiple

[1] Lartigau, *Jour. Exper. Med.*, vol. vi., p. 23.
[2] Conrath, *Beitrag z. klin. Chirurg.*, 1898, vol. xxi., p. 1.
[3] Itié, *Thèse de Montpelier*, 1898.

enterostenoses from carcinomatous peritoneal metastases. At the inser-
tion of the mesentery to the whole of the small intestine there were 99
shriveled stenotic, cancerous foci, and 56 on the lateral aspect of the
mesentery and the convexity of the intestine. The primary tumor was
a cylinder-celled carcinoma of the sigmoid flexure.

Peritonitis plays a very important rôle in the pathology of the
permeability of the bowel. The various forms of peritonitis may act
in many different ways in this respect. Acute and diffuse peritonitis,
for instance, produces a condition of the bowel called ileus paralyticus,
in which the passage of the bowel contents is interrupted. The chronic
form of peritonitis frequently leads to the formation of fissures, holes,
and bands in the peritoneal cavity, which are frequently in their turn
the structural basis of acute occlusion and strangulation of the bowel.
These two possibilities will be discussed in another part of this book.
Chronic peritonitis *per se*, however, may, in addition, lead to simple
interference with the permeability of the intestinal lumen in another
way and without producing any disturbance in the circulation of the
blood in the bowel-wall and without producing symptoms of strangula-
tion of the intestine. This mode of production is the one which chiefly
concerns us here. Chronic peritonitis may act mechanically on the
intestine in many different ways. Since the final effect of this mechanical
action, however, is essentially the same, the sequelæ of chronic peri-
tonitis that pertain to the permeability of the intestinal canal will all
be dealt with in one section, so as to make the description more com-
prehensive and complete.

Here we are concerned only with the description of anatomic and
mechanical relations, and cannot enter into a consideration of the etiologic
relations of the forms of peritonitis connected with this subject. We
must, therefore, confine ourselves to the pathology of peritonitis.

Treves has written a most excellent monograph on the effect of
chronic peritonitis on the permeability of the intestinal lumen. He has
shown in an admirable manner how narrowing of the bowel lumen
can be brought about in many different ways by chronic peritonitis.
Some of the cases that he has collected may be considered anatomic
rarities ; others again are relatively frequent.

(*a*) Peritoneal scar tissue may surround the whole or a large part of
the circumference of the bowel like a ring or girdle, and thus produce
stenosis of the bowel lumen. Such annular strictures from without
appear only singly and may only be partial. In describing internal
strictures of the bowel due to cicatrization of ulcers of the bowel-wall
the formation of these external peritonitic strictures in the affected area
has already been referred to. In these cases they are the direct result
of an ulceration of the intestine, and augment the internal stenosis of
the bowel by exercising constriction from without, so that the lumen of
the bowel is stenosed both by an internal and an external cicatrix. These
double strictures appear both in the small and in the large intestine, and
are dependent, of course, upon the seat of the ulcer. There is, in addi-
tion, another form of stricture due to local peritonitis, in which, how-

ever, there is no primary disease of the bowel-wall itself. This form is most frequent in the large intestine, and chiefly in the region of the flexures of the colon. One form of this constricting, circumscribed peritonitis of the colonic flexures is particularly interesting; it is called partial hypochondriac peritonitis, and was investigated by Virchow, who was specially interested in the condition. This form of peritonitis is most frequent in the region of the hepatic, splenic, and sigmoid flexures and the cecum; it is occasionally seen in other portions of the large intestine. The process leads to the formation of adhesions between the affected portion of the colon, on the one side, and neighboring organs, on the other. The hepatic flexure, for instance, may become attached to the under surface of the liver, to the gall-bladder, the mesentery, the pylorus, or the duodenum; the splenic flexure may become attached to the spleen, the mesentery, or the diaphragm. This partial form of peritonitis may be due to a variety of primary causes. It may either have its starting-point in some organ in the immediate vicinity of the intestine, thus, from pericholecystitis (in gall-stones), from a perihepatitis fibrosa, a condition that is occasionally seen in cirrhosis of the liver and in women with a "corset liver"; it may start from perisplenitis, from perityphlitis, and from some chronic inflammatory process involving the female sexual organs or the tissues in their immediate vicinity.

[The subject of gastrocolic adhesion has been studied by Bouveret;[1] it may be due to chronic peritonitis of tuberculous or carcinomatous origin, or, more often, to ulcer and carcinoma of the stomach setting up perigastritis. The transverse colon may become constricted by adhesions or bent at an acute angle. Bouveret considers that this form of stricture is as common as carcinomatous stricture of the colon, and accounts for the development of intestinal symptoms, especially peristaltic contractions, in cases of gastric ulcer or carcinoma. The left side of the abdomen is unaffected and forms a contrast to the heaving, distended right side.—ED.]

Another very frequent cause of this form of peritonitis is fecal accumulation. In the account of the decubital ulcers and cicatrices which may depend on fecal impaction attention was called to the fact that partial peritonitis frequently accompanies cicatrization of these ulcers. It appears, however, that local peritonitis may develop in the region of the ulcer, even though the ulcer itself does not produce any great damage to the inside of the intestinal wall. Virchow believed that the local peritonitis in these cases may be solely due to the irritation of stagnating fecal matter in the region of the decubital ulcer. There seems to be, in addition, a congenital form of local constriction of the bowel from without that is due to the development of intra-uterine peritonitic processes. Willard, for instance, reports a case of this kind in which a peritonitic band situated in the splenic flexure of the colon had produced constriction of the large intestine. Partial constriction of this kind produced by peritonitic processes, without internal lesions of the

[1] Bouveret, *Rev. de Méd.*, April 10, 1899, p. 323.

bowel-wall, is much less common in the small than in the large intestine. In the small intestine it is probably most frequently seen when strangulated hernias have been reduced or cured by operation.

This constricting form of partial peritonitis has the same effect on the intestinal lumen as a stricture due to cicatrization of an ulcer, and the structural changes in the intestine above the stricture are the same as in those described in chronic stricture of the intestines, so no detailed account need be given here, as the reader can refer to the general anatomic description of chronic stenosis of the bowel. The clinical course of external strictures of the bowel from local peritonitis is also identical with the course of internal strictures—in other words, the clinical picture is the same as that described in the section on Gradual and Slowly Progressive Stenosis of the Bowel.

(b) Occasionally cases of constriction of the intestine are due to the following factor: As a result of the intestine becoming adherent to some other organ by one small portion of its lateral surface, the bowel becomes more or less kinked, and thus chronic stenosis, or even acute occlusion of the intestinal lumen, results. The intestine may become adherent to the abdominal parietes or to some other loops of intestine that are not anchored and fixed so tightly by peritonitic adhesions; it may happen that the transverse colon becomes adherent to the uterus, or the ileum to the tissues surrounding the femoral ring, etc. The causes that produce adhesions of this kind are manifold—for instance, traumatism of the abdominal wall, tuberculosis, and carcinosis of the peritoneum, pericholecystitis of the gall-bladder, pelvic peritonitis and other forms of localized inflammation, particularly those localized peritonitides due to hernias. A loop of intestine that becomes strangulated in a hernial sac and is reduced after it has been severely inflamed frequently forms partial but firm adhesions with neighboring parts. Adhesions may form between neighboring organs and one or more points of the surface of the intestine, so that the bowel may become knuckled and bent in one or more places. The angles thus formed are frequently very acute. It is important to remember that the bowel may become kinked in several different places; in other words, that in the same individual the intestine may be constricted or kinked in a number of places by peritoneal bands, and in operations on these cases for the relief of the stenosis or the occlusion of the bowel a careful search should always be made for other points of knuckling or occlusion, even after one has been relieved by surgical interference. I have repeatedly seen surgeons perform a single operation on one of these knuckled or occluded portions of the bowel without, in reality, curing the disease or relieving the acute condition, simply because they did not examine the rest of the intestines, and consequently failed to discover the presence of bands constricting other portions of the intestine. As a result, the operation was futile and the occlusion of the bowel continued. Sometimes laparotomy has to be repeated, the second adhesion carefully looked for, and relieved.

Treves has studied the methods by which this form of partial peri-

tonitis interferes with the permeability of the intestinal lumen, and has found that it may vary in different cases. In some cases portions of the intestinal wall become fixed and anchored in one or several places, which alone may interfere seriously with the propulsion of the feces, though the continuity of the intestinal lumen is not really interrupted; the condition resembles a partial enterocele (so-called Littré's hernia). In the same way as in Littré's hernia, some accidental factor, such as trauma, or some unknown cause, may produce an acute inflammation in the region of one of these old points of adhesion. Whenever this occurs, the interference with the motility of the intestine is greatly increased, so that complete obstruction of the intestinal lumen may rapidly develop. In other instances an attack of acute diarrhea or of colic with increased intestinal peristalsis may lead to rapid kinking of the bowel. The same effect may be produced if the intestine becomes acutely and greatly distended. Again, in other cases, the permanent kinking of the bowel is so great and the adhesions are so extensive and so rigid that they act in the same manner as circular internal or external strictures. In these cases true stagnation of the bowel contents is brought about, so that the typical anatomic consequences are seen in the portions of the bowel above the adherent area.

Adhesions of this kind are quite compatible with prolonged freedom from any bad symptoms. In some instances this condition never produces any symptoms whatever during life, and is only found as a surprise at the autopsy. Attention has already been expressly called to this fact in the section on Habitual Constipation. (See p. 107.) When disturbances appear, the course and the clinical picture presented by the disease may vary greatly. This is quite natural in view of the variations in the finer mechanism of these processes, which have been briefly described in the preceding paragraph. Sometimes the disease presents the same appearances as in genuine internal strictures; in other words, there are visible peristaltic movements of the bowel and rigidity and stiffening of certain loops; the patients suffer from constipation, which may alternate with diarrhea, or the latter may appear alone. Again, in other cases, the scene is terminated by the occurrence of acute occlusion of the bowel with all its distressing sequelæ. Finally, in some cases the whole syndrome appears suddenly and acutely.

(c) In a third group of cases chronic peritonitis produces narrowing and occlusion of the bowel in the following way: The serous surfaces of the intestine in some circumscribed area of the bowel become adherent to the serous surface of some other loop of intestine in the neighborhood.

In the case of the small intestine a descending branch of a certain loop occasionally becomes adherent to the ascending branch of the same loop, either in one spot or along the whole lateral surfaces of the two pieces of intestine that are in contact. In the former instance a round loop is produced with a circular lumen; in the latter case the two pieces of the intestine become tightly adherent to each other, so that they cannot be separated. In the latter case, moreover,

a very acute sharp angle may be formed at the apex of the loop—namely, where the one branch curves over into the other one. It can readily be understood that this acute bend constitutes an obstacle to the propulsion of the bowel contents, so that this particular form of adhesion between two loops of the small intestine may produce the same functional and anatomic consequences as an internal or an external stricture of the bowel. As a matter of fact, all the characteristic symptoms of stricture of the intestine are occasionally produced in this way. When the ascending and descending branches of the intestinal loop are merely adherent in one spot, the condition may remain quite unrecognized and not produce any symptoms; on the other hand, it may occasionally lead to very severe symptoms of acute occlusion of the bowel. In this instance the acute occlusion is produced either by volvulus of the intestine or by internal incarceration. The primary cause of adhesions that form between the two branches of the same loop of the small intestine may be either ulceration of the intestine or disease of one of the mesenteric glands. (Treves reports two cases of this kind in which he found a breaking-down gland in the angle of the loop.) This form of adhesion may also start after the reduction of a hernia, especially when the strangulated loop has been exposed to severe compression in the hernial opening.

The formation of local adhesions between the peritoneal surfaces of two different parts of the large intestine can occur only when the colon is dilated, elongated, or displaced. This can readily be understood if the exact anatomic position of the colon is kept in mind. There is only one portion of the colon in which peritoneal adhesions can form between two pieces of the large intestine in the absence of displacement; this is the sigmoid flexure, between the loops of which adhesions occasionally form. Whenever any part of the colon is abnormally long or displaced, an attempt must be made to discover whether these abnormalities are congenital or acquired. This refers particularly to the comparatively frequent form of displacement and bending of the transverse colon in which this portion of the bowel becomes a V-shaped or U-shaped tube. This condition is quite frequently acquired as the result of habitual constipation. The mechanism of this displacement and distortion is simple, for the bending of the transverse colon is simply due to the accumulation of fecal matter in this portion of the bowel. As soon as large quantities of the contents of the bowel accumulate in the transverse colon, this piece of intestine will naturally drop downward, especially when the mesentery attached to it is sufficiently long. In cases in which such acquired or congenital displacement or elongation of the colon exists, adhesions may form between the colon and normally distant organs. In isolated cases a still more interesting condition may be brought about—namely, the formation of partial adhesions between the surfaces of two portions of the colon that are normally far removed from each other. Leichtenstern and Curschmann have given a good description of the various possible forms of adhesions which may form in connection with the colon, and of the peculiar elongation of this portion of the bowel

that has been observed in many cases. These local adhesions may be produced by catarrhal and ulcerative processes in the colon. The condition may remain long or even permanently latent, so that, clinically, the existence of adhesions may remain entirely unrecognized. In some instances the only symptom produced is constipation. When the sigmoid flexure becomes adherent to neighboring organs, or its two branches become adherent to each other, volvulus is readily brought about. Volvulus may also occur when adhesions form in other parts of the colon, but kinking is more frequent.

(*d*) In another group of cases wide-spread peritonitis may lead to matting together of numerous intestinal loops and to adhesions between the intestine and the parietal peritoneum, and also with those portions of the bowel which are normally bound down to the back of the abdomen. This condition is particularly frequent in tuberculous and carcinomatous peritonitis, where loops of intestine are often found to be tightly adherent and fixed to each other. In these adherent intestinal coils kinking and twisting of the bowel may occur. This interesting and important condition will be discussed in detail in a subsequent section.

Treves is inclined to distinguish another form of local peritonitis which produces similar results in circumscribed portions of the bowel. After describing the result of wide-spread peritonitis which has just been referred to, he goes on to speak of the development of adhesions between limited portions of the small intestine that occasionally develop as the result of more or less localized peritonitis. The different loops of intestine that are thus adherent to each other may either become fixed to the abdominal parietes or may remain free. Sometimes only short pieces of the bowel are involved in the process; in other cases nearly one-half of the ileum has been found rolled up and converted into a solid ball. In cases of the latter kind it may happen that this ball of intestinal loops is felt through the abdominal walls during the life of the patient, and gives the impression of a diffuse abdominal tumor. In one case an ovarian tumor was diagnosed and a laparotomy performed for its removal.

[Even when the abdomen is opened and the adherent intestinal coils are exposed to view, the appearances may be so like a cyst or tumor that there is considerable risk that an incision or puncture may be made by the surgeon. Malcolm [1] records an instructive case where the matted intestines looked like a broad-ligament cyst, and it was only the absence of intestines above the supposed new growth which led to the supposed tumor being percussed, when it was found to be resonant and to be adherent intestine. He refers to cases in which adherent intestine had been cut into and aspirated.—Ed.]

The form of peritonitis leading to these intestinal adhesions is usually produced by some surgical operation—for instance, herniotomy or ovariotomy. In other cases the peritonitis is not traumatic, but merely remains localized. The symptom-complex presented in cases of pronounced peritoneal adhesions of the bowels varies greatly, particularly

[1] J. D. Malcolm, *Lancet*, 1901, vol. ii., p. 76.

as regards those symptoms that depend on the permeability of the intestine. In many of these patients autopsy shows the most complicated lesions, although the subjects may have suffered no appreciable inconvenience during life, and often have had no intestinal symptoms whatever. In the great majority of cases, it is true, constipation supervenes, while in others constipation alternates with diarrhea; in a third class of cases the whole symptom-complex of a gradually progressive stenosis of the intestine (with visible stiffening and rigidity of certain loops of intestine) develops, and death occurs, with symptoms of occlusion of the bowel.

(*e*) Peritonitis starting from the mesentery or from mesenteric glands is of particular significance and importance. Local chronic mesenteric peritonitis (Virchow) specially occurs near the last lumbar vertebra, usually somewhat to the right and near the attachment of the mesentery to the cecum, and still more frequently on the left side of the mesentery of the sigmoid flexure. In mesenteric peritonitis the mesentery becomes hard and shortened in certain directions. This shortening naturally impairs the normal motility of the corresponding intestine, and consequently the propulsion of the bowel contents in this part of the intestine is also impaired. A specially important form of mesenteritis is *mesenteritis sigmoidea*, which may also occur during fetal life. This inflammation of the mesentery causes approximation of the ends of the sigmoid flexure, and consequently constitutes the most frequent immediate cause of volvulus of the sigmoid flexure (compare also the section on Volvulus of the Intestine). *Peritonitis mesenterica chronica* also occurs in the diffuse form, and may result in the whole of the small intestine being drawn toward the spinal column. In many cases it is impossible to discover the etiologic factors of this disease. Sometimes chronic mesenteritis occurs together with universal peritonitis involving the bowels, and is due to the same causes. Some writers claim that *peritonitis mesenterica chronica* may be due to chronic venous engorgement of the portal area.

Disease of the mesenteric glands is of considerable importance in producing conditions which interfere with the passage of food through the intestine, for, in the first place, a diseased mesenteric gland may be the direct cause of universal chronic mesenteritis; in the second place, an isolated adenitis meseraica—that is, the involvement of one or two glands only—may lead to serious results, as described under (*b*). The factor of importance in determining the results is the exact situation of the affected gland or glands. If they are situated as in paragraph (*b*), they may lead to local adhesive inflammation of the peritoneum of two branches of a loop of intestine, and may, in this way, produce all the sequelæ already described; in other cases they may lead to the development of localized inflammation of the serous membrane of a loop of intestine, on the one hand, and some fixed organ, on the other, and in this way cause anchoring and fixation of an intestinal loop. In other instances the diseased mesenteric gland may lead to the formation of adhesions between a free diverticulum or a free portion of the mes-

entery with the intestine or the abdominal parietes, or may favor the development of cords and bands which, in their turn, may cause strangulation of the bowel.

Peritonitis chronica omentalis also plays an important rôle in the pathology of permeability of the intestine, but this condition is important only in the development of acute occlusion of the bowel. (Compare the section on Internal Incarceration of the Intestine.) A detailed discussion of this condition and of its results will, therefore, not be given here.

(*f*) Finally, another variety of stenosis of the bowel must be mentioned, which was described by Treves, who explained its mode of origin. Stenosis of the bowel may be the result of traction exercised on the bowel by a diverticulum. In Treves' cases the intestine was much stenosed at one spot, and the mucous membrane of the intestine above the stricture was covered with decubital ulcers. There were no dense, short, peritoneal adhesions, as in the group of cases described under (*b*), but he found that the distortion of the intestinal wall was due to the dilatation of certain portions of the wall resembling diverticula. In one case there was a true diverticulum. These diverticula, in their turn, became adherent to other portions of the bowel by peritoneal adhesions ; in some instances they were adherent to the mesentery or to the abdominal parietes.

The mechanism of this process, as Treves has correctly shown, is the following. These band-shaped diverticula interfere with the function of the bowel directly the slightest traction is exerted, by fixing the intestine to a slight degree or by bending it somewhat. In this sense they act similarly to ordinary fixed peritoneal bands. They, therefore, constitute a chronic obstacle to the passage of the bowel contents, and may produce all the anatomic and functional sequelæ of this condition. As a matter of fact, the case reported by Treves presented the syndrome of chronic stricture of the small intestine during life.

COMPRESSION OF THE INTESTINE (Compressio Intestini).

In its wider sense, the term compression of the intestine includes a great variety of conditions. It means more than simple compression of the bowel by a tumor, by a displaced abdominal organ, or by other tumor-like swellings of the abdomen. Properly speaking, all the forms of constriction of the bowel brought about by the development of peritoneal bands and all cases of internal strangulation of the intestine, invagination, the formation of knots in the intestine, and similar processes belong to this category ; strictly speaking, the intestine, in all these conditions, is occluded or narrowed by agencies acting from without—namely, by compressing its external surface. I will conform with general usage by limiting the term " compression " to those cases of stenosis or occlusion of the bowel lumen mentioned in the first sentence of this paragraph—namely, those cases in which the intestine is

really compressed by some force that acts in a coarse, mechanical manner. It must be understood that this force is operative directly upon the intestinal tube, and that the intestine is not merely strangulated or invaginated, and that it is not knotted.

In this limited sense compression of the intestine in the great majority of cases occurs through the agency of new growths. Examination of the literature shows that tumors arising in a number of organs situated in immediate proximity to the intestine, such as the liver, spleen, pancreas, certain lymph-glands, the omentum, the mesentery, and the bones of the pelvis, may compress the intestine; no detailed description of all these possible factors is necessary, since no definite principles or points of importance would result from such a review. The tumors which most commonly compress the intestine in this way are new growths of the ovaries, which, from their anatomic relations, are particularly liable to cause compression of the rectum and the sigmoid flexure; moreover, the hard and unyielding character of the bony walls of the pelvis further assist in the process of compression of the sigmoid flexure and rectum by ovarian tumors. Finally, when the rectum and sigmoid flexure are fixed by peritoneal adhesions and are thus prevented from moving away from the tumor, which is encroaching on their lumen, compression is still more liable to occur; the same applies when peritoneal adhesions form between the neoplasm and the bowel-wall. Tumors of the pancreas may and often do produce compression of the duodenum. (Compare for these tumors and their effects the section on Stenosis of the Duodenum.)

Occasionally compression of the bowel is brought about by a displaced normal organ, such as a wandering spleen or a floating kidney. Again, in other cases, part of the bowel becomes enormously distended with accumulated fecal material and presses on some other portion of the bowel. All these conditions, however, are rare. Compression of the bowel by the displaced uterus, however, is common, while a pregnant uterus is specially liable to compress the intestine.

[A sacculus connected with the urinary bladder, especially when the communication between the sacculus and the bladder is so small that the sacculus remains tense, has been known to press on the small intestine and to give rise to obstruction (T. Bryant,[1] Harrison [2]). It has been suggested that a greatly dilated stomach may mechanically press on and obstruct the duodenum (Box and Wallace [3]).—ED.]

In rare instances an inflammatory swelling or an abscess in the abdominal cavity, such as a paratyphlitic, psoas, or spinal abscess, may compress the bowel.

Another form of true compression of the bowel depending on the action of one portion of the intestine on another requires special discussion. In these cases the mesentery of the small intestine leads to constriction of the pedicles of certain knotted and twisted portions of the bowel in volvulus and in knotting of the intestine. According to

[1] T. Bryant, *Medico-Chir. Trans.*, vol. lxxxv., p. 37.
[2] R. Harrison, *ibid.*, vol. lxxxv., p. 42.   [3] Box and Wallace, *Lancet*, 1901, vol. ii.

Leichtenstern, this compression of the bowel by the mesentery occasionally comes on slowly and produces chronic and long-continued constipation.

The rectum is the portion of the bowel most frequently exposed to compression. This is chiefly due to two factors : in the first place, this portion of the intestine is anchored and fixed ; in the second place, it is situated within the pelvis, where there is less room and where the bony walls offer a certain amount of resistance to any compressing agency. Portions of the bowel situated outside of the pelvis are able to give because the walls of the abdomen are soft, but portions of the bowel like the rectum, within the pelvis, cannot yield because the walls of the pelvis are rigid. The presence of the female genital organs is another factor which frequently plays a part in producing compression of the rectum. Next to the rectum, the descending colon, with the sigmoid flexure, and the lowest portions of the ileum are most frequently compressed ; of the other portions of the bowel which are rarely compressed, the duodenum is, relatively speaking, most often, while the jejunum and the transverse colon are hardly ever, affected. It will be seen that the frequency with which different portions of the bowel are compressed is directly proportional to the movability of these portions, those parts of the intestine that are tightly fixed and anchored being more liable to compression from without than those that are not attached so firmly.

The clinical picture of compression of the intestine varies. In some cases the picture is similar to that of a gradually progressive enterostenosis, with its prolonged chronic course and its complete symptom-complex. In other cases acute occlusion of the bowel occurs. As a rule, in this instance the symptoms of strangulation are insignificant or absent, whereas the symptoms of simple interruption of the bowel passage are more conspicuous, consequently the course of this form of acute occlusion of the bowel is less violent and the pain and collapse are less severe than in other forms. These differential points, however, apply only in a very general sense. Baimbrigge, for instance, reports a case of acute compression of the colon by a floating spleen in which death occurred within twenty-four hours after occlusion of the bowel occurred. Such acute occlusion results either from a sudden change in the position of some tumor or from the sudden dislocation of a movable organ, or, finally, from the sudden strangulation of a loop of intestine underneath the tumor, the swelling, or organ.

Occlusion of the intestine at the duodenojejunal margin by the superior mesenteric artery deserves special consideration. It has been called also " intestinal strangulation," but this condition had best be included with occlusions from compression on account of its pathogenesis. Albrecht, who contributes 2 cases of his own, published a very complete description of the subject, based on all cases reported up to 1899. He states that Rokitansky described and correctly interpreted the condition. Subsequently Kundrat worked on the anatomy. Schnitzler was the first to consider it clinically.

In these cases constriction of the duodenum at its point of junction with the jejunum is caused by the mesentery of the small intestine or by the trunk of the mesenteric artery and vein. The latter compress the duodenum lying behind them into a cord. The duodenum and the stomach become greatly distended with their contents, so that the latter may extend almost to the symphysis. The small intestine, however, is empty, and is almost wholly situated within the true pelvis. The end of the enormously distended duodenum presents a great contrast to the empty and contracted portion of the jejunum.

Constriction of the end of the duodenum by the mesentery or by its vessels may occur when the empty small intestine has passed entirely within the limits of the true pelvis and thus causes traction on the mesentery. Among conditions favoring this descent Kundrat speaks of a peculiar conformation of the mesentery, powerful contraction, or complete emptiness of the small intestine and relaxed abdominal walls. Schnitzler added to these a comparatively low position of the duodenum and marked lordosis of the lumbar spinal column. Albrecht, on the contrary, attaches greatest importance to the distention and dilatation of the stomach, which has been shown by every autopsy. This condition produces the displacement of the intestines by forcing them out of the upper and middle parts of the abdominal cavity lower and lower down into the pelvis. This pressure, moreover, prevents the re-ascent of the intestines out of the true pelvis, and renders strangulation at the duodenojejunal junction by the mesenteric vessels inevitable. Thus the strangulation itself leads to further distention of the stomach and duodenum.

The clinical picture is at times so extraordinary that the diagnosis would seem to be possible, and, as a matter of fact, it has been made by Schnitzler. The cases usually ran a course similar to that of acute strangulation, in which, however, pain was usually absent. There were also local manifestations, such as are characteristic of duodenal obstruction—*i. e.*, retention of stools and flatus, profuse biliary vomit, distention of gastric region, slight meteorism of the intestines. Noticeable distention of the stomach, when existing alone and of acute nature, is a most important symptom. But, as Albrecht justly says, in making a diagnosis we must carefully consider the possibility of previously existing abnormalities in the position of the stomach. Albrecht contended that those cases of " acute gastric dilatation " running a course that ends in death belong to the above-described cases of strangulation of the intestine by the mesenteric artery ; but von Herff has recently advanced a new theory : he believes that the distention of the stomach and the weakening or complete paralysis of its muscular coat are the chief features in the clinical picture, and that the acute gastric distention is only of secondary importance, in that it produces closure of the duodenum by stretching the mesentery. Doubtless, impairment of the nervous apparatus of the stomach underlies this acute gastric paralysis. This impairment may perhaps be due to injury of the spinal cord or to an acute gastric distention. Again, it may be due to the toxic action

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of chloroform or to toxins formed in the stomach. For many of the cases no explanation is possible. In view of my entire lack of personal experience in the matter I must content myself with merely recording the experience of others.

[Narrowing of the third part of the duodenum by the pressure of the superior mesenteric vessels is, according to Byron Robinson,[1] particularly likely to occur in visceroptosis when the patient lies on the back.—ED.]

INTERNAL OBTURATION OF THE INTESTINE (Obturatio Intestini).

OBTURATION of the intestine is the occlusion of the bowel by some body situated within its lumen. Obturation, moreover, usually signifies complete occlusion, not merely stenosis, of the bowel lumen. It may be produced by the following different kinds of solid bodies :

(*a*) By gall-stones.
(*b*) By enteroliths.
(*c*) By foreign bodies.
(*d*) By masses of fecal matter.

(*a*) **Intestinal Obstruction Due to Gall-stones.**—An overwhelming majority of the gall-stones which enter the intestine through the natural passage—*i. e.*, through the common duct—pass through the bowel without difficulty and are evacuated with the feces. Calculi of this character are, as a rule, much smaller than a small hazel-nut. Occasionally the passage of a somewhat larger gall-stone through the intestine produces transitory disturbances, such as colicky pain, nausea, vomiting, and meteorism. In exceptional cases very large stones have been known to pass through the intestine without producing any further symptoms ; a case is, for instance, on record in which a gall-stone measuring $9\frac{1}{2}$ cm. in its largest diameter was passed with the feces without having produced any symptoms of intestinal obstruction. (For the literature on this subject the reader should refer to Leichtenstern's dissertation, to reports of cases in various "Jahresberichte," and particularly to an excellent monograph by Naunyn on cholelithiasis, and to a dissertation on the same subject by Schüller.) In a certain number of cases somewhat larger stones, and some of exceptional size, become impacted in the bowel ; when this occurs, they form a plug that occludes the lumen of the intestine, and in this way produce the clinical picture of occlusion of the bowel. According to Naunyn, very few calculi measuring more than 3 cm. in their smallest diameter pass through the bowel without producing symptoms of intestinal occlusion. In exceptional cases it may happen that a gall-stone originally of small size gradually enlarges in the intestine ; for instance, it may become lodged in a diverticulum of the intestine, and, under these circumstances, phosphates are frequently deposited on the surface of the gall-

[1] Byron Robinson, *American Practitioner and News*, August 15, 1900, p. 125.

stone; later it may reënter the main canal of the intestine in its enlarged form and then produce obstruction of the bowel lumen. A few cases are also on record in which large masses of small concretions produced occlusion of the intestine.

[Treves [1] removed a calculus with a diameter in its long axis of $1\frac{1}{2}$ inches from the ileum of an old lady who, for many years, had taken carbonate of magnesia daily. The nucleus was a small gall-stone, and its large size was due to layers of magnesia and fecal matter. —ED.]

It is very doubtful whether the largest of the calculi occasionally found in the intestine ever enter the intestine through the natural passage—viz., the common duct. Almost without exception—I might almost say always—some fistulous connection is first established between the gall-bladder (less frequently the common duct) and the duodenum, through which the large gall-stone travels. In rare instances there is a fistulous canal between the gall-bladder and the colon. In the latter instance the gall-stone naturally enters the colon direct, and the danger of obstruction is much less than when the gall-stone enters the small intestine. The only portion of the large intestine in which a gall-stone might possibly be retained under these circumstances is the lowest portion of the rectum.

As a rule, then, the gall-stones enter the duodenum; they may either become impacted in this portion of the bowel or they may be propelled downward as far as the jejunum, or even as far as the lowest portion of the ileum or the ileocecal valve. When the gall-stone succeeds in passing the ileocecal valve, the greatest danger is usually over, for it is a well-known clinical fact that gall-stones are most liable to become impacted in the ileocecal valve. After they enter the colon, they are usually propelled onward without any more difficulty. The only point, as I have said above, at which they may again be arrested, is that portion of the rectum that is situated immediately above the anus. Gall-stones are only exceptionally retained in the colon or in the upper and middle portions of the ileum. The various positions mentioned above are the narrower portions of the bowel, in which, consequently, the gall-stones naturally tend to become impacted. In addition, of course, there may be a great many possible abnormal points of stenosis in the bowel, in any one of which the stone may be arrested; I refer, for instance, to stenosis of the intestine from cicatrization of some ulcer, to neoplasms, to peritonitic contractions or knuckling of the bowel. As these lesions may affect any portion of the intestine, it is, as a matter of fact, impossible to formulate any definite statements in regard to the portion of the intestine in which a gall-stone may become impacted.

[In a case of multiple tuberculous strictures of the ileum in which the affected area of intestine was excised by Mayo Robson,[2] a gall-stone was found in a pouch between two of the strictures.—ED.]

Gall-stones which become impacted in the intestine are not necessarily

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[1] Treves, *Intestinal Obstruction*, ed. 1899.
[2] Mayo Robson, *Trans. Clin. Soc.*, vol. xxxv., p. 58.

always of considerable size. Israel reports a case in which he performed a laparotomy for the relief of symptoms of occlusion of the bowel. On opening the intestine a gall-stone was found that measured only 2 cm. in its largest diameter. This gall-stone was situated in the ileum, 20 cm. above the ileocecal valve. The stone, moreover, was freely moveable in all directions, so that it did not really mechanically obstruct the propulsion of the bowel contents. This small stone was the only abnormal condition found to account for the occlusion of the bowel. Israel believes, therefore, that in cases of this character there is a form of occlusion of the bowel which he proposes to call " dynamic." He argues that probably the stone irritates the intestinal wall, and that reflexly it produces local muscular contractions of the bowel. Naunyn, Körte, Rehn, and Heidenhain agree with Israel; I believe that their explanation is correct, particularly as it seems to explain the clinical picture presented. It must be remembered that the absolute size or the absolute weight of the stone is of subordinate importance in producing mechanical obstruction. The most important feature is the form of the stone. A long stone with a small circumference may occasionally pass through the intestine without any difficulty, whereas a smaller spheric stone with a large diameter may become impacted in the bowel.

[According to Mayo Robson,[1] intestinal obstruction may be brought about by gall-stones—(1) By mechanical obstruction of the bowel; (2) as the result of local acute peritonitis around the gall-bladder setting up paralysis of the bowel in the neighborhood—these cases recover without operation; (3) volvulus of the small intestine, which may depend—
—(a) on the intensity of the nervous disturbance during acute biliary colic; (b) on excessive peristalsis accompanying the passage of a calculus along the intestine—this form is closely related to the mechanical form of obstruction; (4) as a late result of cholelithiasis due to strangulation by adhesions or bands formed in connection with the gall-bladder or from cicatricial contraction in connection with fistulous passages between gall-bladder and duodenum or colon.—Ed.]

If a gall-stone remains in one place for a long time, it may produce irritation of the mucous membrane of the intestine, followed by ulceration of the bowel-wall, peritonitis, and even perforation of the intestine. Treves reports two cases in which an impacted gall-stone produced circumscribed purulent inflammation of the bowel-wall and finally led to the formation of an intestinal fistula that perforated the abdominal walls, through which the gall-stone was finally evacuated. In another case there was a fistulous opening at the right side of the back, from which a gall-stone was discharged. In some instances the stone, after lying for some time in one portion of the bowel, produces a diverticulum of the intestinal wall.

Clinical Features.—Obstruction of the intestine by gall-stones is a comparatively rare disease. It is more frequent in women than in men. This is, of course, due to the fact that cholelithiasis is more frequent in the female than in the male sex. In Naunyn's 127 cases of intestinal ob-

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[1] Mayo Robson, *Medico-Chir. Soc. Trans.*, vol. lxxviii., p. 117.

struction produced by gall-stones, only 34 were in men. This form of intestinal occlusion seems to be more frequent in the second half of life than in the first. This, again, is due to the greater predominance of cholelithiasis in this period.

[Morestin [1] has collected a total of 242 cases of intestinal obstruction due to gall-stones. In eight years, at the London Hospital, there were 360 consecutive cases of intestinal obstruction, and of these, 8 were due to gall-stones, or in the proportion of 45 cases of obstruction due to other causes to 1 due to gall-stone obstruction (Barnard).[2]—Ed.]

The symptoms of obstruction of the bowel are, in a number of cases, preceded by attacks of colicky pain which point with more or less probability to the existence of cholelithiasis. The history, therefore, of paroxysmal attacks of pain may indicate the correct diagnosis, especially when the attacks of pain are characteristic of gall-stone colic and are accompanied by definite icterus. In many cases however, there is no evidence of this kind in the history of the patient, and there is nothing whatever to point to the existence of gall-stone colic. Occasionally more or less definite symptoms raise the suspicion that something is wrong in the liver region. The patient, for instance, may complain of spontaneous pain in this portion of the body, or the region of the liver may be tender on pressure. In many cases, however, there are no symptoms of gall-stones before obstruction of the bowel occurs. This absence of attacks of gall-stone colic can be readily explained when it is borne in mind that calculi large enough to obstruct the lumen of the bowel rarely leave the gall-bladder through the common duct, but, in the great majority of cases, pass through a fistulous communication that is slowly and insidiously formed between the gall-bladder and the intestine. In this way it may occur that intestinal obstruction may thus develop suddenly in an individual who was apparently healthy up to the onset of the symptoms of occlusion.

When a calculus which has got into the intestine is not too large, mild symptoms suggesting a temporary and transitory occlusion of the bowel occasionally appear. These symptoms of obstruction may either be due to an actual temporary occlusion of the bowel or may possibly be due to reflex spasms of the intestinal wall produced by the presence of the stone. Again, in other cases, mild symptoms of occlusion may develop when the passage of the bowel contents is impeded by narrowing of that portion of the bowel lumen in which the stone happens to be situated. The transitory symptoms that some patients complain of are attacks of colicky pain, constipation, and vomiting. Paroxysms of this kind may recur at frequent intervals until, finally, the stone is evacuated through the anus and the paroxysms cease.

In addition, other cases present the complete and pronounced picture of acute occlusion of the bowel—namely, violent pain, violent vomiting that frequently becomes feculent in a short time (77 times in 120 cases, according to Schüller), constipation, meteorism, and collapse.

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[1] Morestin, *Bull. Soc. Anat.*, Paris, 1900, p. 196.
[2] Barnard, *Annals of Surgery*, August, 1902, p. 161.

The pain is usually paroxysmal and colicky in character and becomes more severe with each paroxysm. Constipation may be absent. The great majority of observers, however, lay special stress on the fact that in spite of frequent vomiting—which may even be feculent in character —meteorism never becomes very pronounced. Naunyn, in fact, re- marks expressly that in cases of this kind he has frequently been struck with the apparent contradiction that the patients vomit feculent mat- ter and at the same time expel gas and flatus from the rectum. Other observers have noticed that patients suffering from fecal vomiting may from time to time pass feces by the bowel. In rare instances a tumor or a hard resistant swelling may be felt in a situation corresponding to the pain. This, as a rule, is the impacted gall-stone. As the stone is propelled onward, the area of palpable resistance alters its position. In many instances, however, palpation yields absolutely negative results ; under very favorable conditions—that is, when the abdominal walls are very thin and the disease runs a prolonged course—increased peristaltic movements of the bowel may, in exceptional cases, be seen through the abdominal wall. In addition, all the symptoms of severe collapse from occlusion are usually marked.

These symptoms may persist and remain unchanged until death. The patients usually succumb from collapse, with convulsions and coma ; occasionally peritonitis terminates the scene. As a rule, death occurs between the fifth and the tenth day after the occlusion, rarely sooner or later. Death on the fifteenth day after occlusion has been reported, but this is exceptional. Isolated cases have been recorded in which death occurred still later. The statements made by different authors as regards the mortality of this disease vary greatly. Most investigators find that the mortality is somewhere about 50 per cent. (44 to 56) in all cases of occlusion of the bowel by gall-stones that are not operated upon.

When the condition undergoes a spontaneous cure,—that is, if the obstruction is removed,—the patients feel relieved almost instantly ; in other words, if the impacted gall-stone is propelled onward through the occluded area of the intestine so that the bowel becomes patent and the passage of bowel contents is no longer interfered with, all the bad symptoms seem to vanish at once. This sudden transition from great suffering to comparative relief is especially seen in those cases in which the stone is forced through the narrow inferior part of the ileum and the ileocecal valve into the cecum. The average duration of symptoms of occlusion in these favorable cases is from two to ten days ; a few cases are on record, however, in which the obstruction remained *in situ* for more than three weeks before it was moved and the patient recovered. Sands reports a still more remarkable case as follows : The patient was a woman who was attacked with intestinal obstruction due to an im- pacted gall-stone. On the third day, fecal vomiting began and persisted for over three weeks. There was absolute constipation for weeks. At the end of this time, however, the bowels acted after the patient had taken a large number of laxatives and had received a great many

enemas.   Seven days after this a gall-stone that measured $7\frac{1}{2}$ cm. in diameter was passed with the stools, and the patient recovered completely.

One point must be remembered—namely, that the passage of the gall-stone through the anus does not necessarily always coincide with the cessation of all the symptoms of intestinal obstruction.   Cases are on record in which all the symptoms of occlusion disappeared and in which the stone did not appear in the stools for weeks or even months afterward.   There can be no doubt that in these cases the gall-stone was originally impacted in the ileum, either near the cecum or higher up, and that it was then forced through the ileocecal valve into the cecum and remained in the large intestine until it was finally expelled.

Another peculiar phenomenon is sometimes seen in these cases : occasionally a group of symptoms pointing to intestinal obstruction recurs again and again at long intervals.   The symptoms may be more or less severe, but are always the same.   The different attacks may even occur at intervals of several months ; finally, one of these attacks of occlusion terminates fatally or terminates with the cure of the patient, brought about in the latter case by the propulsion onward and the evacuation of the gall-stone.

The symptoms produced by obstruction of the bowel by a gall-stone vary greatly according to the exact seat of the stone.   (For the differences thus produced the reader should refer to the sections on the Localization of Stenosis and Occlusion of the Bowel, for the results of occlusion and of stenosis of the intestine are the same, whatever the nature of the obstruction may be.   Attention may be especially directed to the fact that obstruction of the lowest portion of the duodenum or of the first portions of the jejunum produces the characteristic symptom of occlusion of the duodenum—namely, profuse vomiting of bilious material.

Concretions formed in the intestine itself (*lithiasis intestinalis*) are distinguished according to size as intestinal sand or gravel and intestinal calculi, just as they are when they occur in the biliary and urinary ducts.   Intestinal sand never causes stenosis, yet a few words concerning it will not be out of place here, although our knowledge of the subject is as yet very scanty.

Eichhorst, who lays stress on the extreme infrequency of intestinal sand, describes it as a very fine powder, grayish-yellow to light brown, but with occasional granules of a light-green color.   The granules, measured with the ocular micrometer, vary from 0.16 to 0.28 mm. They are of stony hardness, and are composed of an organic basic substance which, according to Eichhorst, consists of bacteria, needle-like crystals (fatty substances?), and a homogeneous, transparent mass (mucous or albuminous substances?).   The mineral constituents consist chiefly of calcium and magnesium salts.

The clinical relations of intestinal sand are still uncertain.   Its occurrence has been noted most frequently or perhaps exclusively in enteritis membranacea (see p. 236).   Dieulafoy speaks of a " lithogenous catarrh "

in connection with it, which may have originated primarily or from bacterial activity. The belief of the same author that lithiasis intestinalis occurs in gouty constitutions and is a clinical manifestation of arthritis urica is still hypothetic.

(*b*) **Obstruction by Enteroliths.**—Intestinal calculi (enteroliths) must be clearly distinguished from fecal concretions, for the latter are nothing more than inspissated thickened fecal material. Enteroliths themselves are rarely pure calculi—that is, are rarely entirely of a mineral nature. As a rule, large or small quantities of organic substance are mixed with the mineral constituents that compose the stone. Occasionally they contain pieces of organized structures. In general, however, the mineral substances that compose them preponderate so greatly that the designation " intestinal calculus " seems justified.

I do not consider this the proper place to describe all the different varieties of enteroliths that have been occasionally observed and that are reported in the extensive literature on this subject. All that is necessary is the consideration of a few general points of view. In the following account I shall follow Leichtenstern's views. This author distinguishes three large groups of enteroliths :

1. Hard and heavy calculi. Their structure may be uniform, or irregular both on the surface and on transverse section ; occasionally they show a concentric arrangement. They are usually of a dark-brown color on the outside and a dirty gray on the inside, but they may present different shades of color. They are usually fairly spheric in shape ; sometimes they are more egg-shaped or elongated and elliptic in outline. When several calculi are present, their surfaces are faceted. They are usually of moderate size—from 2 to 3 cm. in diameter, but sometimes they assume enormous dimensions ; calculi have been discovered that were as large as 23 cm. in diameter, and that weighed as much as 4 pounds. As a rule, there is only a single one in the intestine ; occasionally, however, there are a large number. Cases are on record in which 32 distinct concretions were found in the bowel.

The chemic composition of these calculi also varies. Ordinarily they consist of carbonates and phosphates, as the phosphate of calcium, the phosphate of magnesium, and ammoniomagnesian phosphate. In addition they usually contain certain organic substances, and occasionally other inorganic salts, as sulphate of calcium. Frequently it will be found that these inorganic salts have become deposited around a definite nucleus. This nucleus may either be some small foreign body that was swallowed by accident, or something taken with the food (a particle of bone, a shred of vegetable fiber, a fruit-stone, or the like). Schuberg believes that, with the exceptions noted further on, enteroliths are always formed from such foreign bodies or indigestible food-remnants.

2. In a few isolated cases enteroliths have been discovered that originated from the prolonged and continued medicinal use of mineral drug preparations. Bamberger, for instance, describes an intestinal calculus that contained 80 per cent. of carbonate of calcium. His

patient had been in the habit of taking chalk for a great many years. Hutchinson describes an enterolith consisting of magnesia and iron salts, and another observer reports an intestinal concretion consisting of pure magnesia compounds. Kiär found a calculus of 9 grams in a cecal hernia. It consisted chiefly of bismuth, and Leo found concretions consisting of salol in the stools.

3. There is a third group of intestinal concretions that is occasionally included under the category of intestinal calculi. These, too, contain mineral substances,—as a rule, phosphates,—but the inorganic salts constitute only a small portion of the concretion. The greater portion of the stone consists of thickly interlaced masses of indigestible vegetable fibers and membranes. In Scotland, for instance, so-called oat stones (avenoliths) are frequently observed, particularly in subjects who eat large quantities of coarse oatmeal. The "stones" of this group usually have very low specific gravity, since they are composed in great part of vegetable material. These stones, moreover, are usually porous, and contain large vacuoles and cavities ; most of them, therefore, float on water. They are frequently irregular in outline and may occasionally attain considerable dimensions.

[Salol, which is frequently taken as an intestinal antiseptic, may, when taken in the form of tabloids, give rise to the formation of enteroliths. C. R. Marshall[1] has studied the formation of the salol calculi in the gastro-intestinal tract, and points out that in order to avoid this salol should be rubbed up with some innocuous powder or given, as Sahli suggests, in the form of an emulsion. Robin[2] has described small salol calculi, and Treves, in 2 cases of colotomy for carcinoma of the colon, came across calculi of salol which, in one instance, were as large as the tip of the finger and resembled amber in appearance. It does not appear that salol calculi have given rise to obstruction, but it is conceivable that this might occur especially if the bowel was already narrowed by growth or stricture.—ED.]

In the great majority of cases enteroliths develop in the large intestine, particularly in the cecum. They are also often found in the haustra of the colon. Sometimes they can be felt in the ampulla of the rectum. In very rare instances they form in the small intestine, and, as a rule, will then be found in parts of the intestinal wall resembling diverticula. Enteroliths require a long time for their formation, but it is quite impossible to make any definite statements as to the time required for the formation of an enterolith of considerable size, as it is quite possible that they may be present in the intestinal canal for a long time, but remain quite latent and give rise to no symptoms of their presence.

The symptoms of enteroliths only rarely appear acutely, or present the picture of occlusion of the intestine. This may, however, occur when a large enterolith which has developed alongside of the main axis of the intestinal canal—for instance, in a diverticulum—suddenly

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[1] C. R. Marshall, *Brit. Med. Jour.*, 1897, vol. ii., p. 78.
[2] Robin, *Acad. de Méd.*, March 30, 1897.

enters the lumen of the intestine, becomes impacted, and thus causes occlusion of the bowel. In the great majority of cases enteroliths produce chronic symptoms which persist for many years, are indefinite, and of various characters, such as loss of appetite, digestive disturbances, constipation, occasionally very obstinate, and abdominal pain. In a few cases well-marked signs of chronic stenosis of the intestine developed, with all its characteristic symptoms, such as violent intercurrent attacks of colicky pain, vomiting, and complete constipation. In some instances an enterolith situated in the cecum has been known to produce typhlitis and paratyphlitis. In some of the latter cases the stone could actually be felt in the cecal region.

The termination of the intestinal affections produced by enteroliths is either a cure, when the enterolith is gradually moved onward and finally passed by the rectum, or death from peritonitis or from complete intestinal obstruction. In other cases the vague and undefined symptoms due to the presence of a stone in the intestine may persist for a long time until death occurs from some intercurrent disease. In these cases the enteroliths may be merely an accidental and unsuspected finding on autopsy.

The diagnosis can be positively made only in those cases in which the enterolith can be felt in the rectum. In all other instances the diagnosis is at best a probable one. The presence of an enterolith may sometimes be suspected from a study of the patient's mode of life and history. In the absence of more or less definite evidence of this kind of the presence of an enterolith, it is impossible to form a more definite diagnosis than habitual constipation, fecal accumulation, or intestinal catarrh. It is, moreover, possible to confound the symptoms produced by the impaction of an enterolith with the symptoms produced by other chronic forms of stenosis of the bowel. In cases, for instance, in which the patient is very much reduced and in which the stone can be palpated as a hard tumor in the abdomen, the disease may very easily be confounded with a carcinoma of the bowel. This error has, in fact, been made several times.

[A. D. Whiting[1] reports a case of an enterolith in a woman aged sixty-eight which was felt *per vaginam* as a hard, freely movable mass in the right iliac fossa, and was thought to be an ovarian tumor. Radiographic examination was negative. Acute obstruction supervened, and laparotomy was performed, the enterolith being removed from the lower part of the ileum.—Ed.]

(c) **Obstruction by a Foreign Body.**—Foreign bodies—that is, substances possessing no nutritive value and not acted upon in any way by the digestive secretions or by the digestive process in general— may enter the intestine either by mouth or *per anum*. Foreign bodies are introduced into the bowel under a number of conditions : in the first place, children, in their ignorance, insane persons, hysteric subjects, and idiots may all inflict this injury upon themselves. Malice, a desire to "show off," or vanity—*i. e.*, performing certain tricks—

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[1] A. D. Whiting, *Annals of Surgery*, August, 1902, p. 297.

may lead persons to introduce foreign bodies into the rectum. Occasionally certain callings, accidents, sexual perversions, and many other factors lead to the same result. The strangest and the most wonderful things have been found in the intestine—substances of every imaginable shape and form, size and dimension, surface and consistence, chemic and physical constitution; pins and needles; bones and glass beads; dagger-blades and whetstones; spoons, knives, forks, pieces of iron and bits of wood; a small flute; pieces of porcelain; nails and screws; keys and hooks; false teeth and false palates; a stomach-tube; hat-pins; belt-buckles; reels of thread and cotton; neckties and suspenders; masses of hair and wool; fruit-kernels; pea-pods; peels and fibers of a great many different plants in enormous numbers. Cases are on record in which the following articles were introduced into the rectum through the anus: A bottle and a glass; a small pot; a small porcelain dish for mixing paints; a pair of pincers; a pestle; a wooden nine-pin; the tail of a pig; and numerous other things. It is not my intention to give a complete catalogue of all the curiosities that have been found in the intestine. I will limit myself, therefore, to report the following case as an illustration. It shows to what extent the intestinal canal can suffer the presence of foreign bodies. This case is reported by Schmucker and quoted by Esmarch. The patient was insane. In the motions that he passed the following articles were found in the course of eight months: 157 pieces of sharp angular glass, the largest piece being 6 cm. long; 102 brass pins; 150 rusty iron nails; 3 large hair-pins; 15 pieces of iron of different size; a large piece of lead; half of a brass shoe-buckle; 3 tent-hooks. The patient recovered physically, but remained insane.

[The "Human Ostrich," a man aged forty-three, who gained his living by swallowing coins, sovereign cases, pieces of tin, tin tacks, nails, etc., died with perforation and intestinal obstruction. The lower end of the small intestine was mechanically obstructed by an enormous number of foreign bodies (Eve).[1] In describing a case of intestinal obstruction by impaction of a gall-stone in the small intestine J. Hutchinson, Jr.,[2] pointed out that it was practically the same size as the Murphy button used for intestinal resection of the small bowel, and that, as foretold by Chaput, this form of Murphy button, when it becomes free, has, unfortunately, proved fatal in several instances.—Ed.]

At present we are specially concerned with those articles that are introduced into the intestine *per os*—in other words, that are swallowed. It is a well-known fact that in the majority of cases foreign bodies that are swallowed are evacuated through the natural passages and passed with the dejecta. This is true not only of small and round foreign bodies, but also of larger foreign bodies with angles and points. It is often a matter of surprise how such objects can pass through the whole length of the intestine without doing any harm or becoming

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[1] Eve, "Human Ostrich," *Brit. Med. Jour.*, 1894, vol. i., p. 963.
[2] J. Hutchinson, Jr., *Trans. Path. Soc.*, vol. xlvii., p. 94.

impacted anywhere. It is frequently impossible to understand how such bodies can pass through the lowest portion of the ileum and through the ileocecal valve. Sometimes foreign bodies that are swallowed travel through the intestine at the same rate as normal food. This applies particularly to small round bodies. They may appear in the stools at the same time as other digestible normal constituents of the food, and without producing any symptoms whatever.

Larger and more irregularly formed objects may produce a great variety of symptoms. In the first place, they usually remain in the intestine for a longer time than the other articles of food or smaller objects. They are especially likely to come to rest in certain portions of the bowel—namely, the duodenum, the lower portion of the ileum, the rectum, and, most frequently of all, the cecum. According to their size, they produce the symptoms and signs either of complete occlusion of the bowel or only of moderate stenosis of the intestine. In the latter case the symptoms they produce may remit and intermit for weeks and months. During all this time the foreign body is present in the intestine until, finally, it is propelled onward and removed in the natural way. When this occurs, the patients, as a rule, recover. In other cases, again, the presence of a large and irregularly shaped foreign body in the intestine produces severe secondary symptoms and occasionally death. Apart from the symptoms of occlusion *per se* that may result from the impaction of a large and irregularly shaped body in the intestine, parts of the bowel may become ulcerated when the foreign body remains too long in one place.

This ulceration of the intestine may lead either to perforation of the bowel followed by acute peritonitis or to the formation of a peritoneal abscess. Cases are on record in which the latter produced a fistulous opening through the abdominal wall so that the pus was evacuated through the skin, or in which fistulous passages were established with other portions of the bowel or with the vagina, so that the pus was evacuated either into the intestine or into the vagina. Small pointed bodies, like needles, frequently bore their way through the intestinal wall, and occasionally appear under the skin without ever producing an abscess. Even in those cases in which the foreign body, after the lapse of a long time, is at last evacuated *per anum*, the disease is not by any means necessarily cured, for a variety of disagreeable consequences produced by the presence of this foreign body in the intestine may persist for some time. Sometimes, in fact, the passage of the foreign body through the intestine leads to permanent changes, such as cicatrization of parts of the bowel due to ulceration produced by the foreign body.

[**Foreign Bodies in the Appendix Vermiformis.**—In 1400 cases of appendicitis Mitchell[1] found about 7 per cent. of true foreign bodies ; 45 per cent. of the described foreign bodies are fecal concretions. In addition to various kinds of worms, shot, fish-bones, pins, pips and seeds of fruit have been found. Mitchell has collected 33 cases of pins

[1] J. F. Mitchell, *Johns Hopkins Hosp. Bull.*, March, 1899, p. 196.

in the appendix and 2 cases of pins in the cecum ; it is remarkable that in only 1 of the 33 cases was there a history of a pin having been swallowed. In no less than 8 of these cases there was secondary suppuration in the liver. As a curiosity, reference may be made to a piece of pure anthracite coal $\frac{1}{4}$ inch in length and $\frac{1}{16}$ inch thick, thought to be due to agglutination of coal-dust, found in the appendix of a young miner by Lathrop.[1]—ED.]

The accumulation of large numbers of intestinal parasites, particularly of ascarides, was formerly thought to be a cause of obstruction of the lumen of the intestine, and was spoken of as *ileus verminosus.* Leichtenstern was the first to express serious doubts in regard to the possibility of such an occurrence. Some recent observations, however, quoted by Mosler and Peiper, including one of Mosler's own, seem to show that this old view was correct after all and that blocking of the intestinal lumen by masses of intestinal worms can occur. These authors believe that occlusion of the bowel and incarceration and volvulus of the intestine can very well be brought about by the presence of enormous numbers of ascarides.

[In a girl aged three years who died with peritonitis there were two perforations in the ileum, $17\frac{1}{2}$ inches from the cecum, through one of which 25 round-worms projected. The ileum between this point and the cecum, the cecum, and the ascending and transverse colon were completely filled with round-worms. Five hundred round-worms were found in the body (Zotoff).[2]—ED.]

Thanks to the Röntgen-rays, the diagnosis of foreign bodies in the intestines is at present not only possible, but easy. Formerly, of course, it could be made only from the history.

(d) Blocking of the Intestine by Fecal Masses.—The subject of fecal accumulation has already been considered in other sections, particularly those dealing with constipation and paresis of the bowel. In the following paragraphs the subject will again be considered from a somewhat different point of view, but for many details the reader should refer to the sections mentioned, as this section will deal only briefly with the subject. It has already been shown how the accumulation of fecal matter in the large intestine is brought about in habitual constipation ; how fecal tumors which may attain enormous dimensions are formed ; how these act upon the intestine ; the nature of the structural changes produced by their presence in the bowel ; and, finally, how they mechanically obstruct the lumen of the bowel. In the section on Paresis of the Bowel the pathogenesis of occlusion of the intestine by fecal masses is also described ; here, therefore, a general summary of the interrelationship existing between accumulation of fecal matter in the bowel and occlusion of the bowel is all that is necessary.

Accumulation of bowel contents in habitual constipation is usually due to sluggish innervation of the intestinal wall, and less frequently to

[1] Lathrop, *Phila. Med. Jour.*, July 7, 1900.
[2] Zotoff, *Gaz. de Bothine*, 1897, Nos. 11 and 15; also *Rev. de Méd.*, November 10, 1898, p. 919.

congenital or acquired weakness of the intestinal musculature. The greater the accumulation of fecal material in the large intestine, the more serious the results. The whole sequence of events constitutes a vicious circle; the interference with the innervation of the intestine or of the action of the intestinal musculature leads to the retention of fecal material; the accumulation of fecal material, on the other hand, produces disturbances in the innervation of the bowel and interference with the action of the intestinal musculature. When they have once started, these two processes can hardly be arrested, and eventually the picture of the most severe form of occlusion of the bowel is apt to develop. This final issue may be brought about in a number of different ways. Occasionally the quantity of fecal material that accumulates and stagnates becomes so enormous that it occludes the intestinal lumen in a purely mechanical way. In cases of this kind the clinical picture is often that characteristic of slowly progressive stenosis of the bowel, when suddenly all the symptoms become acute and complete occlusion of the intestine supervenes. Again, in other cases, the loops of intestine containing the stagnating fecal matter become so heavy that they sink down in the abdomen and become displaced; in this way knuckling of the bowel, or volvulus, is produced; in other cases where the intestine is distended by the stagnating mass of fecal material the functional powers of the intestine, which, under these conditions, are usually sluggish from the first, become more and more feeble, until, finally, the intestine is no longer capable of performing its normal functions. Ultimately, then, a condition of complete motor paralysis of the bowel is brought about, with all its necessary consequences; in other words, the clinical picture is that of occlusion of the bowel.

The diagnosis of this form of occlusion depends on the presence of palpable fecal tumors. (As the most important points to be considered in arriving at this conclusion have been dealt with, the reader should refer elsewhere for the details.) It is necessary to draw attention again to the fact that it is not always possible to feel the fecal tumors, especially when there is meteorism. In cases of this character the diagnosis must be made chiefly from the history of the case, especially from the fact that the subject has been a sufferer from habitual constipation for many years. Unfortunately, this guide to the diagnosis may frequently be misleading, for fecal tumors may be felt in the intestines of some patients whose bowels act regularly every day.

INVAGINATION OF THE INTESTINE (Invaginatio s. Intussusceptio Intestini).

INTUSSUSCEPTION is that condition in which one segment of the intestine is inserted into a neighboring segment, so that the latter forms a sheath for the former. This condition is one of the most prolific causes of narrowing and occlusion of the bowel. The exact mechanism of this morbid process is not easy to understand. The pathogenesis of

the condition, moreover, is peculiar and by no means simple, consequently it will be well to begin the section on Invagination of the Intestine with a brief consideration of the—

ANATOMY OF INVAGINATION OF THE BOWEL.

The invaginated portion of the intestine consists of three cylinders or tubes—one within the other. The accompanying illustration (*a*) shows a diagrammatic section through such an invaginated area, the section being carried transversely to the long axis of the intestine; (*b*) illustrates a section through the intestine, the section being carried vertically and parallel to the longitudinal axis of the bowel. In (*b*), moreover, the exact relative position of the different canals and of the different anatomic layers concerned in the invagination is shown.

The three portions of the bowel forming the three cylinders have been called by different names, but the outer tube is generally called
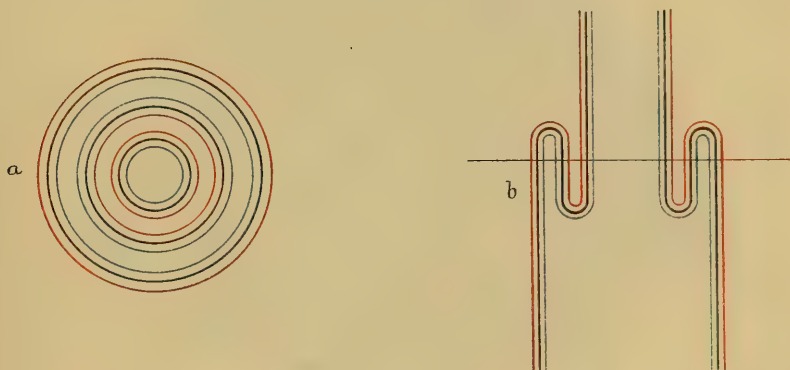
Fig. 4.

the sheath or the *intussuscipiens;* the middle and the inner tubes together are called the *intussusceptum.* The two tubes constituting the intussusceptum are again designated by different names, the innermost, situated nearest to the central lumen of the intestine, being called the entering ("eintretend") tube, the middle one, the returning ("austretend") tube. The upper part of the invaginated area—that is to say, the part of the bowel-wall in which the middle returning cylinder bends over into the sheath—is called the *neck* of the invagination, and usually looks toward the stomach. The lower portion of the invagination, namely, where the internal (entering) cylinder bends over and merges into the returning cylinder, is called the *head* or the apex of the invagination, and it usually looks toward the anus.

In illustration *a* and *b* it will be seen that the external tube is covered by the serous coat on the outside, and that the internal tube is covered by the mucous membrane on the inside. Where the sheath and the returning tube of the invagination are in contact, they are both

covered by the mucous membrane ; where the returning and the entering tube are in contact, they are covered by the serous coat.

This description applies to the ordinary form of intussusception, which consists of three layers of the intestine. A more complex form, the so-called double invagination of the bowel, is occasionally seen, and
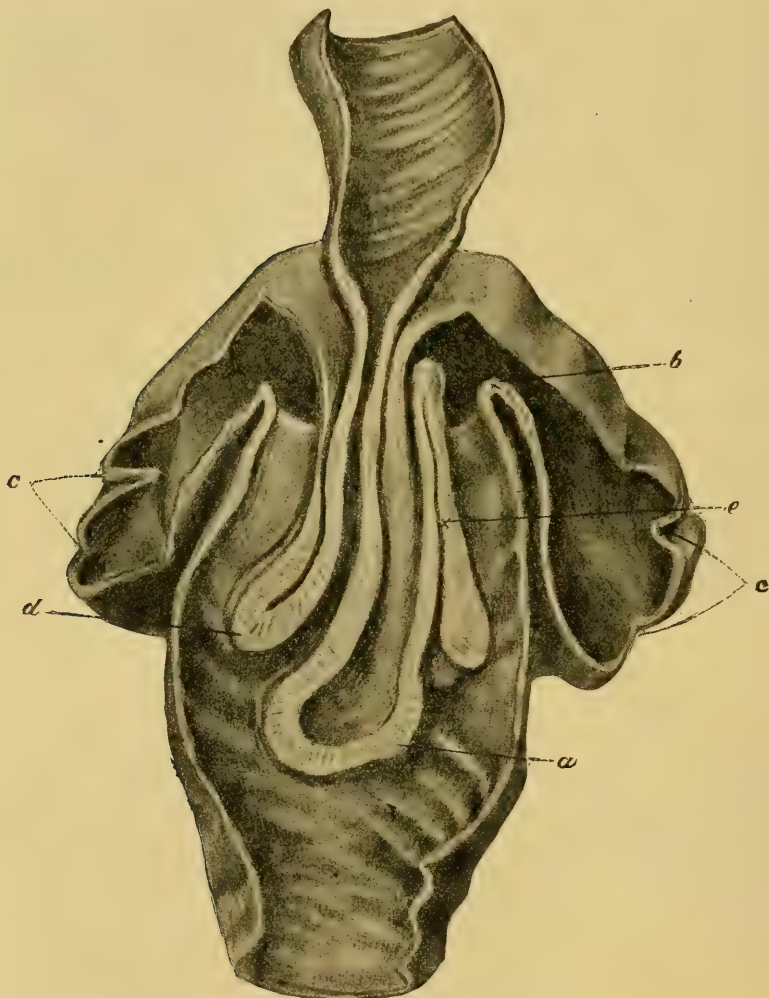
FIG. 5.—Double intussusception (D'Arcy Power).

it consists of five layers or cylinders of intestine. [A good example of such a double intussusception is shown in the annexed drawing.[1] The drawing is semidiagrammatic, and is a longitudinal section through a descending ileocecal intussusception (a) complicated by a retrograde or

[1] Borrowed by permission of the Council from a paper by Mr. D'Arcy Power, in *Trans. Path. Soc. of London*, vol. l., p. 123.

ascending invagination (*b*). The puckering at *c* and *c* was more marked in the first specimen and gave the appearance of an additional or third intussusception ; *d* is the thickened ileocecal valve ; *e* is the vermiform appendix.—ED.] In rare instances a triple invagination consisting of seven layers of intestine is found. The double form of invagination is more frequent in physiologic or agonic intussusceptions than in the pathologic form, which comprises all intussusceptions due to inflammatory or obstructive processes. I shall return to the definition of these different terms in a subsequent paragraph. Leichtenstern has made a very careful study of invagination and distinguishes several different modes of origin. His views as to the origin of double invagination in certain inflammatory conditions are as follows : " If the intussusceptum is held fast in the cecal ostium by an iliac invagination that has entered into the cecal ostium (for instance, from swelling of this ostium or from a spasm of the cecal sphincter), violent peristaltic movements of this portion of the bowel may result. When this happens, a double invagination may occur, for the whole invagination, including the sheath,— and not only the incarcerated intussusceptum,—may become invaginated into the colon. A double invagination may also be brought about by a portion of the ileum becoming intussuscepted ; this intussusception may reach as far as the cecal ostium. It can only go as far as this point because the tissues surrounding the cecal ostium are swollen ; consequently it pushes the whole cecum into the colon in such a manner that the ileocecal ostium is pushed onward and the other portions of the cecum follow ; in this way a double invagination is produced, consisting of ileum, cecum, and colon. A third method in which double intussusception may be produced is the following : Occasionally the sheath of an ileocecal invagination becomes relaxed and folded so that it is pushed upward toward the neck of the primary invagination—in fact, it may become inverted along its whole periphery. (See Fig. 4.) In this way a double invagination is brought about, for the sheath folds upon itself and upon the intussusceptum. Double invagination may finally be brought about in such a way that a portion of the ileum situated above a primary invagination is forced into the canal of this invagination."

Invaginations may be classified in another way. In the first place, there is the ordinary complete, total, or central form of invagination just described ; in addition, however, there is an incomplete lateral or partial form of invagination in which only a circumscribed portion of the intestinal wall is pushed inward toward the lumen of the intestinal canal. This condition is exactly the reverse of local diverticulation of the intestinal wall. This condition has been known for a long time ; in fact, the authors of the eighteenth century mention the occasional occurrence of this lesion. Quite lately a number of authors, especially Duchaussoy, Fleiner, Böttcher, and others, have paid particular attention to this subject and have described cases of this kind. [Eve has drawn attention to this method of commencing intussusception [1] and says that the invagination begins at the caput cæci, or free end of the

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[1] *Brit. Med. Jour.*, 1901, vol. ii., p. 582.

cecum, instead of at the ileocecal orifice. In other cases the apex of
the intussusception is formed of an inverted Meckel's diverticulum, and
it has been known to start from a tuberculous ulcer.—Ed.] Properly
speaking, however, this lateral form of invagination does not strictly fit
in with our clinical conceptions of genuine invagination of the bowel.

Although the purely clinical aspects of invagination of the bowel
will be described in a subsequent paragraph on the origin of this condi-
tion, the account of the anatomy and etiology of invagination must be
prefaced by a few general clinical remarks. This is more especially
necessary when discussing the different kinds of invagination. Writers
on this subject are in the habit of distinguishing, on the one hand, a
so-called agonic form of invagination; on the other hand, a vital, or
persistent, inflammatory (Leichtenstern) or obstructive (Treves) form
of invagination. It will be seen that this juxtaposition of the two
different varieties of invagination clearly expresses the clinical differ-
ences that exist; at the same time, this method of differentiation cannot
be considered to be absolutely correct; it certainly does not exhaust the
possibilities, as will be seen later on. I would suggest, therefore, that
these two different forms should be described by two other names—
viz., that one variety should be called physiologic intussusception, and
the other, pathologic intussusception.

A *physiologic* intussusception produces no symptoms during life.
It is frequently multiple, and in examining dead bodies it will often be
found that the bowel is invaginated in three, five, or even more places.
These "agonic" invaginations occur almost without exception in the
small intestine; the invaginated area is generally small, being rarely
longer than 5 cm. The invagination, moreover, can usually be reduced
by exerting very slight traction on the bowel. The intestine is other-
wise perfectly normal, and no anatomic changes can be discovered in the
intestinal wall, the peritoneum, or the mesentery. The direction of the
invagination in this form may vary greatly. In the great majority of
cases it is ascending; occasionally, however, it is descending; the mean-
ing of this is the following: the piece of intestine that forms the intus-
susceptum may be either formed by a portion of the bowel which is
situated higher up than the sheath; in other words, that a higher por-
tion of the intestine has become invaginated into a lower portion; or
occasionally the reverse, namely, that a piece of intestine forms the intus-
susceptum that is situated lower down (that is, nearer to the rectum)
than the piece of intestine which forms the sheath of the invagination.

In this section we are chiefly interested in the true or *pathologic*
form of invagination. Pathologic intussusception is almost without
exception single. A careful review of the literature on this subject
fails to reveal more than a few cases of multiple pathologic invagination.
Quite recently D'Arcy Power has reported a case of pathologic invagi-
nation of the bowel in a boy of five months. Here two intussuscep-
tions were found: one of them was about two inches long and was situ-
ated in the ileocecal region; the other one was only one inch long and
was situated in the transverse colon. As the serous surfaces of these

invaginated areas were adherent to each other and were covered with lymph, it was certain that this pathologic intussusception had existed for some time during the life of the patient. Another characteristic feature of pathologic forms of invagination of the bowel is the fact that their length may vary greatly; cases are actually on record in which the ileocecal valve appeared in the anus. Pathologic invagination of the bowel may, moreover, involve almost any portion of the bowel; it is true that certain parts of the intestine are affected more frequently than others; in the pathologic form, moreover, all these important disturbances of the circulation in the bowel-wall and the other structural changes appear which are now recognized as characteristic of intussusception. This form of invagination, therefore, produces a group of very severe symptoms. The direction of the pathologic form of invagination of the intestine is almost always descending; only a very few well-established cases of ascending pathologic invagination of the bowel are on record. Leichtenstern, who has collected exhaustive statistics on this subject, studied 593 cases of pathologic invagination of the bowel, and among this large number found only 8 ascending cases. Even in these 8 cases he argues that the term "ascending invagination" is not strictly justified, and he criticizes those authors who advocate the employment of this term. He says that, anatomically speaking, ascending forms of invagination undoubtedly occur—that is, the lower portions of the bowel undoubtedly become invaginated into higher portions of the bowel; physiologically speaking, however, these cases are altogether different from ordinary intussusception of the bowel; in other words, it is not correct to speak of both these conditions as intussusception, because the factors producing the latter form are so radically different from those responsible for the former: they are really two distinct conditions which lead to the same anatomic lesions. He shows that all these 8 cases of so-called ascending "inflammatory" invagination of the bowel originated at a time when the normal peristaltic action of the intestine had ceased, and when antiperistaltic action had already been started. The inhibition of normal peristalsis was in all these cases due to peritonitis followed by paresis of large segments of the bowel. Here, then, the intussusception really occurred in the direction of the peristaltic movements of the bowel, just as in the descending form. There are, however, I believe, a few cases on record in which this form of invagination of the bowel is really due to an ascending retrograde insertion of a lower portion of the intestine into a higher portion, while the peristaltic movements of the bowel remained normal. Besnier reports such a case in a woman of twenty-two years who was ill for nine days and then died. At the autopsy a small and simple intussusception was found, consisting of the sigmoid flexure invaginated into the descending colon. The invaginated portion of the intestine was anchored and fixed by firm adhesions and formed a kind of valve in the intestinal lumen. The intestine above the invaginated area was covered with ulcers. In D'Arcy Power's case, quoted above, there was a double invagination: the one situated in the transverse colon was an ascending invagination,

for that portion of the intestine situated nearer to the cecum formed the sheath for another portion of the intestine situated nearer to the rectum. Jones reports the case of a patient who was ill for eight weeks and in whom, at the autopsy, an ascending intussusception was found, the low portion of the descending colon being invaginated into the transverse colon. Hektoen has recently reported a very remarkable case of quadruple invagination of the ileum. He states that all the intussusceptions were of the ascending type.

As any portion of the bowel may become inserted into that in its immediate neighborhood, three main groups of invagination of the bowel may be made:

(*a*) *Ileo-ileac Intussusception or Invaginatio Enterica.*—In this form one portion of the small intestine becomes invaginated into another portion of the small intestine.

(*b*) *Colocolic Intussusception or Invaginatio Colica.*—In this form one portion of the large intestine becomes invaginated into another portion of the large intestine.

(*c*) *Ileocecal Intussusception or Invaginatio Ileocœcalis.*—In this form a portion of the small intestine becomes invaginated into a portion of the large intestine.

Other subdivisions are, of course, possible; some authors, for instance, speak of the following forms: *Invaginatio duodenalis, duodeno-jejunalis, jejunalis, jejuno-ileaca, ileaca-ileocolica, colica, colicorectalis, rectalis.* All these different terms and the conditions they are intended to designate are self-explanatory. A few remarks will be made with regard to the ileocecal form and the subforms of this condition, termed the ileocolic and ileaco-ileocolic forms, which have been specially studied by Leichtenstern. In the ordinary ileocecal intussusception form portions of the ileum and the cecum become invaginated into the colon in such a way that the valve advances first. In this form of invagination the ileum forms the internal cylinder, the colon the sheath, and the ileocecal ostium, with the valve, the apex. This is the form which occasionally involves long tracts of the intestine; the ileocecal valve may advance through the whole length of the large intestine, and may appear at the anus, or may even protrude through the sphincter ani. In all these cases, however, the ileocecal valve remains the apex of the invagination. In the ileocolic form the conditions are somewhat different. Here the lowermost portion of the ileum prolapses through the ileocecal valve into the colon, so that the ileocecal valve itself and the cecum remain in their normal position; this condition of affairs is maintained until the two invaginated cylinders become adherent to each other. If the process of invagination continues after the two cylinders have become adherent, the cecum and the colon are inverted and form the middle cylinder, since, as the invaginated area becomes elongated, it must involve longer portions of the sheath of the invagination—namely, the colon. In this form, then, in contradistinction to the form described above, the apex of the invagination is always formed by that piece of the ileum that originally became prolapsed. Finally, in the ileaco-

ileocolic form, the first step is a simple ileac invagination which gradually advances until it forces the intussuscepted portion through the ileocecal valve into the colon.

A careful study of all the statistics on invagination of the bowel shows that the different portions of the intestine are involved with equal frequency in the different forms of invagination. The most important and the most frequent form of invagination is invagination of the small intestine into the large intestine. This condition seems to occur at all ages; according to Leichtenstern's statistics, 52 per cent. of all the cases of invagination are cases in which intussusception of the small intestine into the large intestine occurs. He found, moreover, that this particular form is still more frequent in childhood, particularly in the first year of life, for here he found that 70 per cent. were of the ileocolic form. Of the 52 per cent. of cases of invagination, he found 44 per cent. to be of the ileocecal form and 8 per cent. of the ileocolic. Next in order of frequency comes the purely ileac form of invagination—of this, there was 30 per cent.; next the colic form of invagination—of this there was 18 per cent. It will be seen from these statistics that the ileocecal form seems to predominate greatly; this is due to the fact that this particular form of invagination is so extraordinarily frequent in childhood. The statistics of invagination of the bowel in adults show that the ileac and the ileocecal form occur with about equal frequency. For completeness and comparison with the older statistics of Leichtenstern, we give a summary of 321 cases collected by Weiss from the latest literature (1894–98): In the new-born and sucklings there were ileac, 24 per cent.; ileocecal, 42 per cent.; ileocolic, 10 per cent.; colic, 24 per cent.; in those of childhood to puberty—ileac, 23 per cent.; ileocecal, 43 per cent.; ileocolic, 14 per cent.; colic, 26 per cent.; in the adult—ileac, 29.5 per cent.; Meckel's diverticulum, 4.5 per cent.; ileocecal, 34.5 per cent.; ileocolic, 4.5 per cent.; colic, 27 per cent. In the ileum the process usually involves the lowermost portion; in the colon, usually the sigmoid flexure. The rectal form of invagination is rare, and the pure duodenal form very exceptional. Intussusception attains the greatest length in the ileocecal and the ileocolic form, and then in the ileac form. [D'Arcy Power expressed his belief in the Hunterian Lectures, delivered in the year 1897, at the Royal College of Surgeons of England, that intussusception is due to the swallowing of one piece of intestine by a neighboring portion, the part swallowed being usually constricted, owing to local or other causes producing a powerful contraction of the circular layer of muscle. An intussusception, therefore, is generally spasmodic in origin. In children an intussusception is most common at the junction of the small with the large intestine; in adults it is commonly in the colon, or more rarely in the small intestine. An intussusception is generally sudden in its onset in children, and no definite cause can be found, though a polypus, ulcer, or other gross lesion is sometimes present to account for it; in adults, on the other hand, the cause is generally obvious—a stricture, cancerous or otherwise, when it occurs in the large intestine, or a polypus or new growth when it is found

in the small intestine.[1]—Ed.] Colic invaginations are usually short, and rectal invaginations still shorter.

[Intussusception of the vermiform appendix is usually understood to mean its inversion into the cecum, which is followed by invagination of the cecum into the ascending colon; there is thus a double intussusception. Corner[2] has collected 16 cases of this kind, in none of which the appendix had sloughed; this is probably due to the facts that its circulation need not be seriously interfered with, and that its inverted position no longer offers a nidus and culture-medium for micro-organisms. All the cases except one occurred before the age at which appendicitis is common; appendicitis would probably leave behind conditions such as fibrosis, stricture, or adhesions, which would tend to prevent inversion. Corner also refers to two cases of prolapse of the mucous membrane of the appendix into the cecum without intussusception of the cecum into the colon.

Intussusceptions associated with Meckel's diverticulum fall, according to Corner, into two groups:

I. When the diverticulum opens at the umbilicus as well as into the small intestine.

    (*a*) An enteric intussusception formed above the process traverses it and is extruded at the umbilicus.

    (*b*) The ileum opposite the internal opening of the diverticulum becomes protruded at the umbilicus—(1) as the result of prolapse of the diverticulum; (2) primary extrusion of this part of the ileum.

    (*c*) The intussusception begins in the diverticulum itself.

II. The inversion of the diverticulum in acute cases is associated with an enteric intussusception; the gut grasping the process is forced onward—propulsion. In chronic cases the inverted process inverts the ileum behind it by dragging—traction. In acute attacks following a chronic both these, traction and propulsion, enteric invaginations may be present.—Ed.]

In the preceding paragraphs an attempt has been made to give a more or less comprehensive review of the general anatomic conditions in invagination of the bowel. In the following paragraphs a description will be given of the more important processes that occur after intussusception of the bowel has taken place. It can readily be understood that, as soon as invagination of a portion of the intestine occurs, the mesentery becomes greatly distorted and twisted. The most important factors in determining the subsequent course of invagination of the bowel are these changes in the mesentery.

It can readily be understood why, as soon as one piece of the intestine becomes inserted into another piece, the mesentery belonging to the invaginated portion naturally follows its corresponding segment of bowel

[1] *Some Points in the Anatomy, Pathology, and Surgery of Intussusception,* by D'Arcy Power, F. R. C. S. Eng., London, 1898, The Rebman Publishing Co.
[2] E. M. Corner, *Annals of Surgery,* November, 1903, pt. cxxxi., p. 708.

and becomes inclosed in the sheath of the invagination; in other words, it forms part of the invagination and lies between the entering and the returning tube of the intussusceptum. It usually forms a long-drawn-out, and occasionally compressed, conic mass of tissue. The base of this cone is situated at the neck of the invagination, the apex at the end of the invagination—that is, at the lower angle where the bowel folds upon itself. The further the invagination progresses and the longer the invaginated portion of the bowel, the greater the tension exercised on the mesentery. It can easily be imagined that in cases of ileocecal invagination in which the ileocecal valve advances as far as the rectum the traction exercised upon the mesentery must become enormous. Treves, however, remarks that in reality this tension is not so great as it might seem, for the invaginated portion of the intestine describes a circle in traveling from the cecum to the anus. The root of the mesentery is the center of this circle, and the mesentery itself constitutes a radius, so to speak, of this circle.

The mesentery naturally exerts traction on the intussusceptum and this part of the bowel becomes curved in consequence, and usually forms an arch, the concavity of which points toward the mesenteric attachment. In a slighter degree tension is exerted on the sheath, so that this portion of the intussusceptum also becomes curved; in other words, the whole swelling formed by the invagination of the bowel frequently assumes a peculiar arched form, the concavity of which is always directed toward the mesentery. These peculiarities are most pronounced in the case of ileocecal intussusceptions, while in the ileac and colic forms this arching may frequently be absent, and in the purely rectal forms it is always absent.

The arching of the swelling formed by the invagination produces certain sequelæ that are of immediate clinical importance. The bending of the invaginated area may be so considerable that true angular kinking of the bowel is produced and the lumen of the intestine is thereby diminished. This is always a serious event, for stenosis and occlusion of the bowel may thus follow a simple invagination; as a matter of fact, excessive arching of the invaginated portion of the bowel is the most prolific cause of occlusion or stenosis of the affected area. Another important sequel of invagination of the bowel which is also directly due to the traction exerted upon the affected portions of the bowel by the mesentery is the following: In cases in which more traction is exerted on the intussusceptum than on the intussuscipiens the axes of the two cylinders may become displaced in their relation to each other in such a manner that the axis of the intussusceptum comes to occupy an eccentric position. When this occurs, the lumen of the intussusceptum is also distorted, so that it forms a narrow slit which is no longer directed straight toward the lumen of the bowel below, but more toward the wall of the intussuscipiens. (See Fig. 4.)

The circulatory disturbances that develop in the invaginated portions as a direct result of traction and compression of the mesentery are of **fundamental** importance in determining the clinical course of a case of

invagination of the bowel. The intensity of the resulting circulatory disturbances is directly proportionate to the rapidity with which the invagination develops. Another factor which, of course, determines the intensity of the circulatory disturbances is the degree of compression to which the mesentery is exposed after it becomes included in the invagination. In physiologic intussusceptions no anatomic alterations are seen, for the traction and compression of the mesentery in these cases are either very slight or absent. In the pathologic form of invagination, however, somewhat the same sequence of events is observed as in a strangulated hernia, and they are described further on.

The changes are not, however, identical in intussusception and in strangulated hernia, for the tissues in children are more elastic and gangrene is consequently less common than in adults. In the majority of cases the strangulation and compression of the mesentery are not at first very severe, so that the veins are compressed more than the arteries. As a result, dark, bluish-red and blackish-red hyperemia of the mesentery and the invaginated portion of the bowel develops. This hyperemia is rapidly followed by exudation, so that the invaginated portion of the bowel becomes edematous and swollen. If this condition of stasis and congestion continues, hemorrhages occur both into the parenchyma of the intestinal wall and into the internal and external surfaces of the intestine.

The anatomic picture presented by the intussusceptum is very typical. The development of this picture can readily be explained when we consider what must be the direct results of the processes just described. Definite anatomic changes are produced in the intussusceptum both in the acute and the chronic forms of invagination; usually the lesions are more severe in the acute form. In the intussusceptum in a case of invagination of the bowel which has run either an acute or a chronic course the following conditions are found: The swelling and thickening of the bowel are more pronounced on the convex surface of the invagination than on the concave surface; this is due to the same hemodynamic causes that determine the greatest amount of swelling at the apex of the intussusceptum. This part of the intussusceptum may constitute a nodular tumor and may be very much thickened— in fact, so much so that the general appearance presented by this tumorlike swelling, which is perforated by a canal with a narrow lumen, recalls the appearance of the os uteri. Of the two cylinders forming the intussusceptum, the middle one is more involved than the internal one, and always shows more pronounced anatomic changes. The inner cylinder, in fact, is occasionally completely contracted, and forms a band or ribbon-shaped mass; at the same time the middle cylinder may be very much thickened, so that it measures as much as 1.5 cm. in diameter. This hyperemia from stasis and edema may either involve the whole intussusceptum or chiefly its apex; at all events, it constitutes another frequent cause of obstruction of the bowel; it also seriously interferes with the reduction of the invaginated portions of the bowel.

If the strangulation does not become excessive, the whole swelling

may partially recede, and in this way permeability of the intestinal lumen
may be reëstablished.   In some cases the intestinal lumen never becomes
absolutely occluded during the whole course of the disease ; occasionally
permeability for liquid bowel contents is reëstablished, while solid mate-
rial cannot pass.

In cases in which the strangulation of the mesentery reaches ex-
treme degrees the arterial and venous circulation of the bowel-wall
is seriously interfered with or completely interrupted, so that gangrene
of the intestine may supervene.   This strangulation of the mesen-
tery may occur at once, at the onset of the disease, or may develop
gradually.   Gangrene of the bowel is more frequent and more pro-
nounced in cases of invagination of the bowel that run a very severe
and acute course.   It usually involves chiefly the neck of the invagi-
nated area, and is frequently followed by sloughing of the invaginated
cylinder.   Sloughing of this portion may be either complete or nearly
complete.   The development of gangrene is not so rapid in intussus-
ceptions which run a chronic course, and in these cases, too, the apex
of the invagination is more apt to be involved than the neck.   Feigel
described two cases of invagination of the bowel in which ulcers devel-
oped at the apex of the intussusceptum.   The destruction of the bowel
is more gradual in chronic intussusception, so that those portions of the
intestine that slough off and are eliminated appear as shred-like struc-
tures in the feces.   All statistics seem to point to the fact that sloughing
of the intussuscepted portion of the bowel occurs more frequently and
more rapidly in those cases that run an acute course than in those that
run a chronic course.   Leichtenstern, who has gone into this matter
very carefully, finds from his statistics that among 125 cases sloughing
occurred 94 times before the fourth week, 3 times after the sixth month,
and only in isolated cases later.   The length of the piece that is passed
per anum may vary greatly—sometimes it is only a few centimeters
long ; in other cases it may be as long as 3 meters (the case of Cruveil-
hier).   Sometimes long pieces of intestine are evacuated through the
rectum in a perfectly intact state ; their structure may be quite normal,
and it may be easy to determine the exact seat of the invagination from
the examination of the piece of bowel that is passed.   In other cases
nothing appears in the motions but shred-like pieces of bowel tissue
which show no definite histologic structure.

The middle cylinder of the invagination is, as a rule, the part most
seriously involved—both in the hyperemic swelling just described and
also, as a rule, in the gangrenous changes.   Occasionally, however, the
reverse holds good—namely, that the gangrenous process involves the
inner cylinder principally.   In the ileocecal form of intussusception the
innermost cylinder of the invaginated area is usually involved (Treves).
In this form it occasionally happens that the portion of the intussus-
ceptum formed by the small intestine loses its vitality sooner than the
portion formed by the large intestine.   In cases of this kind the most
remarkable conditions are often seen ; it has been found, for instance,
that the piece of intestine that sloughs off was apparently inverted,

so that the inner surface was directed outward. Treves, again, has called particular attention to this phenomenon and has furnished a very lucid description of these anomalies. He describes the occurrence of the apparent inversion of the intestine just mentioned as follows: In the ileocecal form of invagination, particularly if peritoneal adhesions form at the neck of the invagination, the innermost cylinder, consisting of the ileum, may be liberated, while the middle cylinder, consisting of the colon, is not freed. When this occurs, the former rolls over downward in such a manner that the mucosa of the inner cylinder (ileum) becomes turned toward the mucosa of the sheath (colon). Finally, this piece of intestine (after its companion, the middle cylinder, has become loosened by the gangrenous process) sloughs off and is passed by the rectum. Naturally it is evacuated in the inverted position, the genesis of which has just been described.

The sheath of an invagination is less frequently affected by hyperemic changes or gangrenous processes. When processes of this character do involve the sheath, they are usually less intense than when they involve other portions of the invaginated area. It happens quite frequently that the sheath becomes slightly infiltrated and thickened, and in exceptional cases gangrene may supervene or perforation may occur.

[D'Arcy Power gives the following history of the case of a patient who had passed a portion of intussuscepted intestine:[1]

"The Museum of the Royal College of Surgeons of England contains (No. 2715) a piece of bowel measuring 40 inches in length with a polypus attached to its upper end. The piece of bowel was passed by a lady aged thirty-two, who was suddenly attacked with vomiting and abdominal pain on December 28, 1863. The symptoms recurred at intervals for two or three weeks, and at the end of this time she had complete intestinal obstruction, lasting for two or three days. The bowels then acted regularly and without the passage of blood. The piece of intestine was passed through the anus, enveloped in fecal matter and without any blood, on the eighteenth day after the supervention of the severe obstruction and fifteen days after the resumption by the bowel of its normal habit. The patient died July 30, 1889, from some lung trouble. She became very thin before her death, and at times was subject to constipation, but she never again suffered from obstruction of the bowels. No postmortem examination of the body was made."

Mr. D'Arcy Power made microscopic sections of a portion of the exfoliated bowel and reported that "the specimen consists of the whole thickness of the intestinal wall, for portions of the mesentery are still attached to it and the mucous membrane is seen covering its inner side. The microscopic sections show that all the coats of the intestine have undergone sclerosis. The line of the mucous membrane is distinct, though none of the details of its structure can be seen, but the submucous coat is indistinguishable from the muscular, and the muscular layers from each other, or from the serous coat. The whole thickness of the

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[1] *Op. cit.,* p. 23.

section consists of dense fibroid tissue, like that found in an old scar. This tissue is a little more cellular and rather more vascular just beneath the mucous membrane and along its outer border; but otherwise it is a dense hyperplasia of the connective tissue containing a few large vessels."—Ed.]

A study of the intestine above the invagination shows that in cases of intussusception of the bowel that run an acute course no appreciable changes or alterations in the structure of this portion of the intestine can, as a rule, be discovered. In cases, on the other hand, that run a chronic course all the sequelæ which usually follow in cases of chronic stenosis of the intestine in general may develop in a very typical and marked form; in other words, dilatation of the intestine with hypertrophic changes in the muscular layers of its wall, and occasionally hypostatic ulceration.

Peritonitis is another important complication of intussusception. It usually begins on the second or third day, or rather it would be more correct to say that the first symptoms of peritonitis become noticeable on the second or third day after invagination. In the sections on Peritonitis the details of the etiologic factors concerned in the production of the peritonitis which complicates invagination of the bowel will be more carefully gone into. Nothing more will be done here beyond mentioning some peculiarities of the peritonitic processes that are seen in intussusception. Inflammatory processes involving the peritoneum are almost constant in the chronic form of invagination; in the acute form they are also very frequent, occurring, according to Treves, in more than one-half of the cases. In Rafinesque's statistics of the peritonitic complications of intussusception there were 23 cases of chronic invagination, in 4 of which there was no evidence of peritonitis whatever, although the invagination lasted for a very long time—in one for ten, and in another for thirteen, months. In the paragraphs on chronic invagination (*vide* p. 538) a case analogous to these and running a very peculiar course is described.

The peritonitic process may involve large or small areas of the invaginated portion of the bowel. Sometimes the peritonitic process is limited exclusively to the neck of the invagination; in other cases it is found only at the apex; the latter is very much less commonly involved than the former. Most frequently the peritonitic process produces adhesions between the serosa of the inner and the middle cylinder, so that these two portions of the invaginated area become tightly adherent to each other along their whole length. The constitution and the character of these adhesions vary according to the duration of the peritonitic process. If the local peritonitis is of short duration, only a few fibrinous adhesions, that are readily torn, may be found; if the peritonitis persists for a long time, very tough and firm membranes may be formed between the different portions of the bowel. In very rare cases the peritonitic process leads to the pouring-out of a liquid exudation.

The prognostic significance of the peritonitic processes that complicate invagination of the bowel may vary greatly. When peritonitis

develops very early in the course of the disease, the prognosis is influenced unfavorably, since the formation of adhesive bands between the invaginated portions of the bowel interferes with the spontaneous separation and reduction of the intussuscepted pieces or even makes complete recovery impossible. Very delicate and fragile adhesions, of course, do not necessarily interfere with the reduction of the invaginated portions of the bowel, because these delicate adhesions can be torn apart. If, however, gangrene begins, particularly before peritonitic adhesions have formed at the neck of the invagination, diffuse perforative peritonitis may occur as soon as the invaginated piece of bowel sloughs off ; in this way a fatal issue may readily be precipitated.

Finally, another sequel which may occasionally follow sloughing of the intussusceptum must be dealt with. It must always be remembered that the process of sloughing *per se* is really one of ulceration, for there is always ulceration of the invagination where separation of the invaginated piece of intestine occurred. As has been shown above, this generally occurs at the neck of the invagination. The ulcer left at the neck of the invagination, after the invaginated portion of the bowel has sloughed off, usually undergoes cicatrization, and as it is generally annular and involves the whole circumference of the neck of the invagination, a circular scar is produced. In this way internal stricture of the bowel may develop and in its turn may lead to all the results of this condition which have already been described. The stenosis of the bowel may be further increased by the traction exerted on the cicatrized portion of the bowel by the adhesions that develop near the neck of the invagination as the result of an adhesive peritonitis. In this way stenosis of the bowel may occasionally develop long after the invagination of the intestine itself has healed spontaneously.

[D'Arcy Power [1] reports the various histologic changes exhibited by the portions of intestine involved in an intussusception. The result of his examination shows that any part of the intestinal wall may be affected, but that one portion usually suffers more than others, and the stress of the affection falls most often upon the submucous tissue and upon the circular layer of muscle. The mucous membrane, too, may be seriously injured, but the longitudinal layer of muscle and the serous coat are the least often affected. The earliest histologic changes are correlated with an effusion of blood, but the amount of the extravasation varies greatly—at one time so slight as hardly to displace the tissues, at another time so considerable as utterly to destroy them. The seat of the extravasation, too, varies. It may be in the mucous membrane and it seems that this occurs in the most acute cases ; it is usually in the submucous coat, though it may be in the muscular layers of the serous coat. The extravasation is followed by inflammatory changes, in which the submucous tissue and the circular layer of muscle are chiefly involved. These changes terminate in a hyperplasia of the connective tissue, leading to sclerosis ; in a tryptic (pancreatic) digestion, leading to the disappearance of every cellular element in the wall

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[1] *Op. cit.*, pp. 8–33, and *Jour. Path.* and *Bacteriol.*, vol. iv., p. 484.

of the bowel and the conversion of its connective tissue into reticulin ; in diffuse suppuration or in sloughing of the inflamed bowel, which is then separated and cast off by the ordinary process of ulceration.—ED.]

PATHOGENESIS AND ETIOLOGY.

For a long time all attempts to explain the pathogenesis of invagination of the bowel were based exclusively on hypothetic speculations. The different writers on the subject indulged in many theories in regard to the pathology, physiology, and anatomy of this condition, and based all their ideas on a very limited number of clinical observations. They soon discovered the peculiar fact that in some instances the same clinical observation could be utilized in support of theories diametrically opposed. It is quite unnecessary to give a critical review of all the hypotheses which have from time to time been formulated by different authors. It will be sufficient to give a concise and brief exposé of those facts and theories which at present have a direct bearing on the pathogenesis of invagination of the bowel.

No direct proof of the various assertions that have been advanced as regards the factors determining the development of invagination of the bowel can be made on the basis of pure speculation ; it is necessary to perform a number of experiments, and in this way to demonstrate directly *ad oculos* the earliest stages of invagination and the phenomena which develop consequent to this event.

From a resumé of the various facts known as to the histogenesis of invagination of the bowel it appears that many points are still obscure, and that a satisfactory and convincing explanation is still required.

A priori it must be granted that invagination of the bowel may be produced by spontaneous movements of the intestine itself. The first question to be answered is, What are the factors which alter the otherwise regular peristaltic action of the bowels in such a way that invagination of adjacent portions of the intestine can occur?

In one of the preceding paragraphs a distinction was drawn between physiologic and pathologic intussusception. The very fact that such a classification is recommended and that a physiologic form of invagination is mentioned shows that, in my opinion, invagination of the bowel may be produced by the normal movements of the intestine.

Starting with this premise, it remains to be shown whether the pathologic intussusception owes its origin to some perversion of the normal movements of the bowel, and whether this perversion is a difference in degree or in kind.

Finally, an investigation into the causes determining intussusception must settle the question whether invagination of the intestine can ever occur without the intervention of any external, or so-called causative, factor, or whether some abnormal condition that can cause a perversion of the peristaltic action of the bowels is necessary.

For many years authors have been speaking of agonic intussusception as a condition occurring *in articulo mortis.* Agonic invagination

is frequently found in examining children postmortem, particularly those under ten years old. It should, however, be expressly mentioned that agonic invagination of the bowel also occurs in adults, although much less frequently than in children. Professor Kolisko, who has made a careful study of this subject in the postmortem material at his disposal in our Pathologic Institute here at Vienna, tells me that the proportion of cases of agonic invagination found in adults and in children is as 1 is to 50. On p. 498 a brief description of the anatomic picture presented by agonic invagination was given. It has not been definitely made out why agonic intussusception is so very much more frequent in children than in adults. The most plausible hypothesis for this phenomenon is that the bowel of a child is very much more irritable than the bowel of an adult. This explanation, however, remains an assumption and hypothesis, for there are no facts to explain this phenomenon satisfactorily. It must always be remembered, too, that the intestine of an adult is not in this respect radically different from the intestine of a child.

[The cause of spontaneous intussusception is unknown, but D'Arcy Power[1] has shown that the width of the large intestine at birth is only a few millimeters greater than that of the small intestine. Before birth its diameter is usually the same or even a little less, while at the age of fifteen years it is from two and one-half to three times as large. The colon begins to grow in girth directly after birth, though it remains for a time almost stationary in length. The ileum, on the other hand, grows both in length and in breadth. The ileum, however, rarely doubles its diameter in the course of its growth, but the large intestine not only often doubles its size, but may even treble or quadruple it. These facts seem to have an important bearing upon the question of the origin of intussusception in young children. The colon is growing in width rapidly and continuously from birth onward, but at about the age of four months—the exact time when spontaneous intussusception becomes common—it also begins to grow in length. The small intestine has grown steadily in length and breadth from the beginning, though the increase in its circumference is less rapid than the increase in its length. During the early months of a child's life, therefore, there is a rapidly increasing disproportion between the transverse diameters of the large and small intestines, and physiology teaches that too rapid growth is often associated with perversion of function, especially when, as in this case, the increased rate of growth affects both the muscular and the nervous tissues. Unduly rapid growth of the large intestine may even allow the end of the ileum to become prolapsed into the colon, and, under suitable conditions, such a prolapse may serve as the starting-point of an intussusception. When an intussusception has once been started, the anatomic peculiarities of the individual alimentary tract are of paramount importance, for they determine the character of the intussusception. In the ileocecal forms a

[1] D'Arcy Power, *Some Points in the Anatomy, Pathology, and Surgery of Intussusception,* London, The Rebman Publishing Co., 1898.

wide colon with few and simple ileocolic folds devoid of lymphatic glands will allow the intussusception to run a chronic course, even though the amount of bowel invaginated is very great. Complex fossæ with numerous lymphatic glands at the ileocolic angle and prolongations of mesentery along the wall of the ileum will, no doubt, so far steady this portion of the small intestine as to render its invagination less likely, though, should it occur, the additional amount of tissue invaginated will render the impaction peculiarly tight, so that if gangrene be not produced at once, early adhesions will be formed and the intussusception will soon become irreducible.—ED.]

I have performed a large number of experiments on the healthy intestine of living rabbits, in the course of which it was shown that complete short invaginations of the bowel (0.5 to 2.0 cm. long) may occur in portions of the intestine that are lying perfectly still. This phenomenon may be observed as soon as the abdomen is opened. In addition, I was often able to watch the production and resolution of invaginations. In all these experiments the abdomen, after it was opened, was filled with warm normal salt solution; this method differed from that of previous experimenters who did not immerse the intestine in a bath of normal salt solution, but exposed the bowels to the irritation of the atmospheric air.

It may be remembered that A. von Haller, under these conditions, also observed and described the formation and the resolution of intussusceptions. In my cases irritation by the air was excluded, and the finer mechanism of the formation and resolution of invagination of the intestine could be carefully studied. An invagination occurs as follows: A piece of the intestine undergoes a very active contraction, and, so to speak, inserts itself into an adjacent portion of the bowel, which is either not in so vigorous a state of contraction or is even lying perfectly still. The contracted piece of intestine is always inserted from above downward; thus the intestine lying near the stomach becomes invaginated into a piece of intestine situated nearer the rectum. This invagination may be released at once; in some instances, however, I have seen it persist for as long as ten minutes. In the latter instance the contraction of the upper portion of the bowel ceases, and a flaccid segment of intestine is seen lying within another flaccid portion of the bowel. I imagine that in this way agonic invagination occurs. This peculiar phenomenon is quite frequently seen in the small intestine when the peristaltic actions of the bowel are lively; it is much less frequent in the large intestine, though it also occurs in this portion of the bowel.

[D'Arcy Power[1] performed a series of experiments on the artificial production of intussusception by increasing the peristalsis of the intestine by injecting turpeth mineral (yellow sulphate of mercury), eserin, and barium chlorid. The general result of the experiments was to produce a series of "agonic" intussusceptions, but in no case to give rise to a true pathologic intussusception. It was possible, on the

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[1] *Loc. cit.,* p. 45.

other hand, in a cat to produce an artificial intussusception which became progressive, for in the course of a week the intussusception had doubled its original length.—ED.]

It appears, therefore, that this form of invagination of the bowel is a physiologic occurrence in living healthy rabbits. Elsewhere I have pointed out that the same may possibly occur in the normal intestine of human beings ; in other words, that the formation of small invaginations in the human intestine which spontaneously become released and lead to no further deleterious consequences is quite a probable state of affairs. This view seems probable, as the peristaltic action of the human intestine is very much more like the peristaltic action of the intestine of a rabbit than of dogs and cats. Cruveilhier long ago took this view, which was also shared by Rafinesque. Treves has recently advocated the same view ; he, too, considers the frequent occurrence of slight degrees of invagination which may be considered transitory and of short duration, a very possible event in the human intestine. Cruveilhier and Treves go still further, and apparently feel justified in assuming that many attacks of colic, particularly those following the ingestion of indigestible food, may be due to temporary invagination of the bowel. They adduce, in support of this view, a number of clinical phenomena which they think prove that temporary and transitory invagination of the bowel is, in fact, the anatomic basis of many paroxysms of colicky pain. Joseph Bell has reported a very remarkable case that may possibly belong to the same category. His patient suffered from internal strangulation of the intestine due to the presence of a band composed of peritoneal adhesions. Bell performed a laparotomy, and on opening the abdomen found, in addition to a band, an invagination of the bowel 10 cm. long which he was able to reduce without any difficulty. The question arises, Was this too a physiologic process ?

Personally, I believe that the formation of small intestinal invaginations is a physiologic occurrence in man. Such invaginations are without danger and quite harmless ; they produce no symptoms, and, owing to the fact that they are so small, can readily undergo spontaneous cure. I think, therefore, that these physiologic forms of invagination are different from the pathologic forms in degree, but not in kind.

The question remains to be answered, What are the factors that determine the development of those extensive and wide-spread forms of invagination which produce the extremely grave symptoms so often seen ? In other words, what produces the pathologic form of invagination ? From a review of the numerous theories that have been advanced in explanation of the pathologic form of invagination it appears that a number of conflicting views have been advocated. The various explanations which have been offered can be divided into two distinct classes—according to one group, invagination is due to spasm, while the other group regards paralysis as the underlying factor—so-called *invaginatio spasmodica* and *invaginatio paralytica*. The most distinguished advocates of the spastic theory are Dance, Cruveilhier, Brinton, Bristowe, and Rafinesque. Brinton attempts to explain the

pathologic form of intussusception in the following manner : He supposes that some part of the bowel suddenly undergoes a violent annular contraction ; at the same time there is a less marked contraction of the longitudinal muscular coat of the same portion of the bowel ; these changes necessarily lead to elongation of this segment of the bowel. He believes that when this occurs the contracted and elongated segment of bowel must naturally be forced into the adjacent intestine which is lying still and quiescent. Rafinesque expresses himself in the same terms.

Other authors have warmly advocated the paralytic theory, which is particularly popular among German authors, as will be seen from an examination of the German literature on the subject. Leichtenstern has formulated the position taken by writers as follows : " Paresis of a limited area of the intestine, together with intense peristaltic action, which may be due to a variety of causes, constitute the conditions that are essential for the production of invagination." Besnier's theory is slightly different from these. He believes that neither spasm nor paresis of the intestine is necessarily the primary factor in the production of invagination ; and that, on the contrary, purely mechanical processes may readily lead to invagination of the bowel. He argues that as soon as a portion of the bowel undergoes even moderate contraction it may drop into an adjacent portion of the bowel by reason of its weight, especially when the portions of the bowel that are situated above the contracted area contain a large amount of heavy material. He imagines, therefore, that the contracted portion slips downward into an undilated portion which is ready to receive it.

Personally I am of the opinion that clinical experience in the majority of cases militates directly against the assumption of a primary paresis of the bowels, and that, in the majority of cases, it must be admitted that spasm is the primary condition or that conditions exist which favor the mechanical invagination of the bowel described by Besnier. Experimental investigations and observations are opposed to the paralytic theory and almost force us to accept the spastic theory.

The question arises, How is invagination of the bowel brought about? The only way to decide this point is by the experimental production of artificial invagination of the intestine in animals. Experiments of this kind usually fail, it is true, in one respect—viz., they cannot exactly imitate the pathologic processes met with in the human body, nor can they fully elucidate all the factors concerned in producing the pathologic changes in question. Experimental work, however, is of definite value in making it possible to study the actual onset of an invagination, and to follow its subsequent course. G. Leubuscher, at my suggestion, has performed experiments of this kind. His results, it is true, were quite negative. Subsequently I carried out another series of experiments on the same lines, but employed a somewhat different technic. My results were positive, and led to the production of artificial invagination of the bowel in the animals experimented upon. This is not the proper place to reproduce the details of my ex-

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periments ;[1] but the more important results and a brief summary of the conclusions based on my experimental data may be given.

It is possible to produce artificial invagination of the bowel in living rabbits by the application of a faradic current to the intestinal wall. This sets up tetanic contracture of a limited portion of the intestine, which is the starting-point of the invagination. In these experiments the application of the faradic current to a segment of the intestinal wall produces tetanic contracture of the lower end of this portion. At this lowest point, then, there are in close proximity to each other one segment of intestine greatly narrowed by contracture and another (nearer the anal end of the bowel) with a normal diameter and lumen. Where these two portions of bowel merge into each other a very small invagination is first formed. The mechanism of this process is simple : the portion of the bowel whose lumen is normal is pushed over the lower end of the contracted portion like a sheath—in other words, it rolls over the narrow piece of bowel. The subsequent development of the intussusception proceeds as follows : The piece of intestine that is situated below—this is, toward the anus—forms the sheath of the intussusceptum, and the whole invagination is produced at the expense of this lower portion. So far as I have been able to study the finer mechanism of the process the circular muscular coat of this portion of the bowel undergoes contraction below the point of irritation, and this contracted portion becomes the "entering" cylinder of the intussusceptum. The "returning" cylinder and the intussuscipiens are formed by the portion of the intestine which is situated still lower down, for in the latter piece of bowel the longitudinal musculature undergoes contractions, so that the bowel is pushed upward by wave-like movements. It is not impossible that the application of the electrodes to the intestine mechanically prevents the lower piece of intestine from moving upward over the whole piece of the upper piece of intestine that is contracted. It is quite possible that in the absence of the electrodes the upper contracted portion would become inserted into the lower portion throughout its whole length, and in this way constitute a much longer "entering" cylinder, consisting of the whole contracted segment of bowel. As it is manifestly impossible to modify the technic of these experiments, I am unable to affirm or to negative this proposition ; this much, however, may be considered established, that the portion of the intestine situated above the contracted portion is in no way concerned in the formation of the invagination.

My experiments supply no evidence whatever in favor of the view that the invagination is in any way paralytic. I have never succeeded in producing intussusception of the bowel after causing paralysis of a circumscribed piece of intestine, even in cases where visible and vigorous peristaltic movements were going on in the portion of the intestine that was situated between the duodenum and the paralyzed area. Leubuscher, whose experiments were practically on the same lines, also failed to produce this result. I may add that paralysis of a circumscribed

[1] *Loc. cit.*, pp. 42–50.

piece of bowel was usually produced by crushing. *A priori* I expected to see that the paralyzed portion of the bowel would be carried into portions of the intestine that were situated lower down by the energetic peristaltic wave coming from above, but, as a matter of fact, I was never able to verify this preconceived idea, and in no instance was the paralyzed piece of intestine inserted into the normal portion situated below it. The phenomena observed under these experimental conditions were entirely different from those which I felt justified in anticipating from the theories advanced by numerous authors. This matter will be dealt with in detail in the section on Paralysis of the Intestine. I do not in any way wish to deny the existence of a paralytic form of invagination—the *invaginatio paralytica* of the clinical physician ; all that I maintain is that so far no experimental evidence has been adduced in support of the view that this form of invagination can occur when limited portions of the intestine become paralyzed ; on the other hand, I maintain that my experiments undoubtedly show that the spasmodic form of invagination can be brought about by the factors employed in my investigations.

On the basis of my experiments I feel justified in enunciating the following views in regard to the genesis of the pathologic form of invagination of the intestine. I believe, moreover, that the explanation which will be offered holds good for the great majority, if not for all, of the cases seen in practice.

The first event in the formation of an invagination of the bowel is an energetic annular tetanic contraction of some portion of the intestine. This contracted area constitutes the fixed point from which the invagination develops. The invagination, however, is not produced in such a manner that the contracted portion of the bowel is forced into the normal intestine situated below this spot by the peristaltic waves coming from above ; on the contrary, the invagination is primarily produced by the action of the musculature of the normal portion of the intestine situated below the contracted spot. It is probable that the longitudinal muscles of the intestinal wall of this piece of intestine are chiefly active, and that they pull the normal intestine situated below the point of spastic constriction upward over the contracted piece of bowel.

In the experimental form the invagination increases in extent exclusively at the expense of that portion of the bowel which is situated toward the anus. Some authors use the expression " at the expense of the sheath " to designate the development of the invagination at the expense of this outer and lower portion of the bowel. In other words, new areas of the bowel situated below the constricted area continuously roll over it and in this way aid in forming the intussusceptum.

When the process of invagination has once started, and particularly when it exceeds physiologic limits, it is enforced and reinforced by the same factors that initiated it. It is quite possible that the head of the invagination, after it becomes tightly wedged into the sheath, constitutes the primary irritant which causes further spasmodic constrictions of portions of the bowel situated above it, or causes violent peristaltic move-

ments of this portion of the bowel, which, in its turn, carries the incarcerated head downward just as it would propel any other constituent of the bowel contents onward.

[This explanation is borne out by D'Arcy Power's case of progressive intussusception produced artificially. The details of the experiment are as follows: The specimen drawn in the annexed illustration (the specimen is in the Museum of the Royal College of Surgeons of England, No. 2726 e) was taken from a cat in which the ileum was invaginated into itself and was pushed into the colon, where it was secured with three interrupted sutures of silk. The intestine was replaced in the peritoneal cavity and the abdomen was closed. The cat took its food and behaved exactly like a healthy cat, except that it would not jump and got down steep places by scrambling. Eight days after the first operation it was again anesthetized and a second incision was made to the right of the first one, which had almost healed. The intussusception was easily found and was secured by cutting through the intestine above and below the tumor. The ileum and colon were then

Fig. 6.—D'Arcy Power's case of artificial progressive intussusception.

joined by Maunsell's method, but the cat died the next day. The invaginated portion of the intestine bears out the theory that intussusception is due to the swallowing of a piece of constricted intestine by a neighboring portion whose peristalsis is still active. The invaginated intestine has been divided longitudinally (Fig. 6). The sutures are seen in position, but, instead of being at the neck of the sac, where they were inserted a week previously, they are now situated in the very middle of the intussusception, at a distance of 32 mm. from its beginning, as measured along the lesser curvature, or 42 mm. measured along the great curve. The whole length of the intussusception was 75 mm., and as the sutures mark the original neck of the sac, the invagination has advanced a distance of 30 mm. in a week—that is to say, it has doubled itself in this time. The intussusception is pervious throughout, a fact which explains the freedom of the cat from any symptoms of intestinal obstruction. The invagination is of the chronic form, as the apex of the invaginated ileum is alone congested. The intussusceptum, as is usual, is curved toward the side of the mesenteric attachment. The

two opposed serous surfaces can be separated from each other as low as the point of suture, but below they are blended by inflammatory adhesions. The adhesions are much better marked along the concave surface, where the mesentery is crowded into a small space, than along the greater curvature, where the two serous surfaces can be separated in their whole extent, except at the point where they have been sutured.—ED.]

The results obtained from experimental work are more or less corroborated by the study of the pathologic anatomy of invagination of the bowel in man. The process of invagination which can actually be seen to develop in animal experiments is also illustrated by anatomic studies in human beings. The part of the intestine which is the primary starting-point of the invagination always remains as the apex of the invagination. For the sake of convenience this segment will be called " C." The whole invagination, however long it may be, subsequently develops from intussusception of the portion of the bowel that is situated below " C," whereas the portion situated above " C " is in no way concerned in the development of the invagination. That is why, for instance, the ileocecal valve may appear at the anus in the ileocecal form of invagination ; why in this form the whole large intestine is invaginated ; and why the ileum, with the exception of the lowest portion that was primarily contracted, is found outside of the invagination proper.

Treves has accepted the views that I have just enunciated on the initial processes which inaugurate invagination. Leichtenstern, who still adheres to the view that paresis of a limited portion of the intestine is the primary causative factor in a large number of cases of invagination, nevertheless emphasizes the fact that in the most frequent form of invagination—namely, the ileocecal form—a spasm of the sphincter of the ileocecal ostium occurs, that this spasm is of the character of tenesmus, and that, in addition, a number of other well-recognized anatomic factors are concerned in producing this lesion. Leichtenstern, moreover, lays stress on the fact that in the ileocolic form of invagination, in which a large portion of the ileum passes through the ileocecal valve into the colon, genuine prolapse of the ileum into the colon occurs—at least to start with—in a manner identical to the complete prolapse of the rectum through the anus (Rokitansky's invagination without a sheath). Later, then, according to Leichtenstern, this abnormal condition is modified in the sense of a true invagination at the expense of the colon.

Let me repeat that I do not deny the occasional occurrence of *invaginatio paralytica*—that is, the formation of an intussusception by " paresis of a limited portion of the bowel." This event undoubtedly does occur in certain cases. I merely wish to emphasize the fact that we are not in a position to describe the mechanism of this form of intussusception on the basis of direct experimental observations, and that if this form of intussusception ever does occur, it certainly constitutes the great minority of the cases seen in practice. This statement is based on a careful study of the clinical factors responsible for the primary development of an intussusception of the bowel.

Predisposing Causes.—If peristalsis, as described above, is the

immediate cause of invagination, then certain anatomic and embryologic facts formed predisposing factors or conditions favorable for the development of invagination. D'Arcy Power has recently carefully studied these factors. He attaches great importance to the relative diameter of the ileum as compared with that of the colon. At birth their lumina are very nearly equal, but in the first few months of life the colon develops far more rapidly. Thus it may often occur that the end of the ileum, already physiologically prolapsed into the colon, may be the starting-point for invagination. These conditions are, in fact, calculated to throw new light on the great frequency of invagination during the first year of life. D'Arcy Power also emphasizes the importance of abnormal length of the mesentery, which was noticed also by the earlier observers. Measurements showed that the proportionate length of mesentery to the entire body in babes is greater than in children and adults, thus giving the greatest degree of movability to the intestine of the nursling.

Direct or Causative Factors.—It is interesting to compare the so-called etiologic factors which have been credited with causing invagination of the bowel, and the factors which experiments show actually do produce this condition. A review of the almost endless number of cases published in the course of years reveals some very interesting facts. A summary of Leichtenstern's extensive statistics based on the notes of some 593 cases shows:

CASES.

1. Absence of any statements in regard to the condition of the patient before the disease 267
2. "Sudden" onset of the disease in perfectly healthy individuals 111
3. Intestinal polypi 30
4. Carcinoma of the intestine and stricture of the intestine 6
5. A history of diarrhea preceding the onset of the invagination 21
6. Other symptoms of perverted intestinal functions 25
7. Ingesta 28
8. Contusion of the abdomen 14
9. Shaking of the body 12
10. Invagination during pregnancy or in the puerperium 7
11. Catching cold given as the cause 6
12. Invagination following various acute and chronic diseases, or following a series of indifferent or etiologically doubtful factors 66

Total 593

Statistics have also been drawn up from the point of view of the age of the patients with invagination of the bowel, and all show that the disease is especially frequent during early childhood up to the fifth year. Of the 593 cases alone that Leichtenstern has tabulated, a large number —viz., 131—occurred during the first year of life; of this number, 80 cases occurred from the fourth to the sixth month; while from the second to the fifth year there were 49 cases of invagination. Pilz tabulated the case-histories of 162 patients with invagination of the bowel, and found that 91 of these occurred in children under one year old (3 under two months, 10 under three months, 55 from the fourth to the sixth month, and 23 from the seventh to the twelfth month); 71 of the cases occurred between the second and the fourteenth year.

Widerhofer collected 58 cases and found that 32 occurred in the first year (16 between the fourth and the sixth month), and that 11 occurred between the second and the tenth year; Weiss, 321 cases—177 in the first year, 85 in second to fourteenth year, 59 in later life.

Leichtenstern's statistics probably correspond in general with the individual experience of most physicians. It is interesting to analyze his tables in regard to the relative frequency of so-called *invaginatio spasmodica* and *paralytica*. A clinical analysis and review of the large amount of statistical material collected by Leichtenstern show the following results : In the first place the cases tabulated under the categories 1, 6, 10, 11, and 12 are quite valueless and prove nothing in this respect. Thus 371 cases are useless either because the etiologic factor is not given or because the factors which are mentioned might equally well produce either the spasmodic or the paralytic form of invagination.

The 111 cases in his second category are particularly important as bearing on the pathogenesis of intussusception, as they form the largest group of cases in which the details of the patient's illness are both perfectly clear and at the same time uniform, since in none of these cases is there any history of illness preceding the development of the intussusception. In all these cases we are, I think, justified in positively excluding paresis of the intestine, for I am quite at a loss to understand how local paresis of the intestine could occur suddenly in a perfectly healthy individual without some definite cause. Widerhofer, in discussing invagination of the bowel in early childhood, expresses himself as follows : " With very few exceptions the writers on this subject all describe the onset of the disease as an attack of colic occurring unexpectedly in a perfectly healthy child. This is usually followed by the early symptoms of stenosis of the bowel." If I understand this matter correctly, the true sequence of events is the following : While the bowel is performing normal peristaltic movements, an annular and strictly local constriction of the bowel happens to occur. This constriction may be greater than normal and so pronounced that the limit of physiologic invagination is exceeded, and the first degree of a pathologic intussusception develops, exactly in the same manner as it has been seen to do in our experiments. It is quite unnecessary to invoke any primary " causative " factor—all that is needed is a simple increase in the intensity of the normal movements of the bowel, which of itself is sufficient to produce this dangerous condition. On this basis, too, the great predominance of invagination of the bowel in early childhood can be satisfactorily explained, for it is usually admitted that at this age the bowel is more irritable and more motile than later in life. The proof of this assumption is that the so-called agonic form of invagination is most frequently found in the bodies of children.

Diarrhea and the ingestion of irritating articles of food probably produce increased peristaltic action of the bowel, but not paresis of the bowel. In this group of cases, therefore, it is probable that the invagination of the bowel is of the spasmodic, rather than of the paralytic, type.

The advocates of the paralytic form of invagination are wont to support their position by adducing those cases of intussusception in which the lesion develops after injury to the abdomen or general concussion. Personally, I do not believe that undeniable evidence has ever been furnished that traumatism of this character is capable of producing local paresis of the intestine. Very much will depend on the intensity and violence of the force brought to bear on the intestine and on the exact mode of action of the traumatic agency. We know that moderate mechanical injuries stimulate the peristaltic action of the bowel. Lichtenstein even describes a case in which an invagination of the bowel involving the left iliac region developed as the result of careless massage treatment that the patient was undergoing for habitual constipation. This group, therefore, proves nothing whatever in regard to the existence of a paralytic form of intussusception. Even if the existence of this form be admitted, it must be recognized as very rare. A comparison of the statistics of the two groups of cases will show that only a very small proportion of paralytic cases are recorded as against the overwhelmingly large number of cases of spasmodic origin.

Finally, another group of cases due to another cause—namely, the action of tumors of the intestine—must be briefly considered. This group of cases is particularly interesting. In carcinoma of the intestine the formation of an intussusception must be considered an exceptional occurrence; in benign tumors of the intestine, however (adenoma, fibroma, myoma, lipoma), in the superficial, particularly in the pedunculated, forms,—so-called polypi,—intussusception occurs with great frequency. When it is remembered how rare, absolutely speaking, these benign tumors of the intestine are, it is surprising in what a large percentage of these cases invagination of the bowel occurs. The polypus is usually found at the apex of the intussusceptum, and may vary greatly in size—sometimes it is no larger than a hazel-nut; in other cases it may be as large as a pear. In the overwhelming majority of cases they are found in the lower ileum, so that the invagination assumes the ileocecal or ileocolic type; occasionally, however, other topographic forms are found. In general, there is only one isolated polypus in the intestine; in some cases, however, a number of polypi are found, one of them, of course, alone causing the invagination. In one case reported by Treves three distinct polypi situated at a considerable distance from one another produced three distinct intussusceptions. Invagination of the bowel due to the presence of this causative factor must probably also be considered spastic in type. Some authors have advanced the view that a polypus, after it forms the apex of the invagination, so to speak, draws the portion of the intestine to which it is attached into the sheath—in other words, converts it into the intussusceptum. In a case of this kind it must be assumed that the invagination is primarily due to the traction exercised upon the intestine by the polypus attached to it. I do not consider this view tenable, especially as so many cases of invagination of the bowel are due to the action of very small tumors. I do believe, however, that the presence

of a small polypus in some portion of the bowel may set up intense peristaltic action of this portion and cause severe constriction of the intestine; whenever this occurs, invagination of the bowel may supervene according to the principles enunciated above. Boetticher and Fleiner each report a case in which a circumscribed myoma of the intestine first produced a lateral (incomplete), and later, secondarily, a central (total), invagination of the bowel.

It has often been noticed that intussusception is exceedingly rare in stenosis of the bowel and in carcinoma of the intestine. These two conditions belong to the same group for the reason that carcinomata of the intestine usually develop into circular growths and produce stenosis of the intestine. The absence of symptoms of invagination in stenosis of the bowel and in carcinoma, however, can easily be explained. Whenever the intestine becomes infiltrated with carcinomatous tissue, the affected area of the bowel is usually converted into a rigid cylinder. In addition, the intestine above the stricture becomes dilated and its walls thickened. Further, adhesions are apt to form, so that this portion of the bowel is particularly unsuited to become inserted as an intussusceptum into the portion of the bowel that is situated below it. The latter portion, moreover, owing to its rigidity, the presence of the carcinoma, and the absence of dilatation, is narrower than the portion above, and consequently particularly unfit to form the sheath of an invagination. It is only in exceptional cases and under most unusual circumstances that the coincidence of invagination of the bowel with carcinoma or other forms of stenosis of the intestine is found. Invagination may occur in carcinoma when the tumor is pedunculated, and consequently polypoid in character, or when the tumor originates in the ileocecal valve. Professor Hochenegg has reported a case of invagination of one of the flexures due to the development of the former condition,—that is, of polypoid carcinoma,—and Czerny has operated on a case of the latter character—viz., an invagination developing from a carcinoma of the ileocecal valve. Fleiner examined this latter case and reported it. Hochenegg also reported another very rare instance of invagination; in this patient the bowel was stenosed at the cecal valve (no carcinoma), and the section of the bowel situated above the stenosis was greatly dilated, its walls hypertrophied, and its lumen filled with a mass of enteroliths (medlar seeds covered with calcareous salts). Here an ileocecal invagination developed, so that the ileum was invaginated into the cecum.

In a few cases a Meckel's diverticulum, either patent or obliterated, has been the starting-point for the invagination, and less frequently the vermiform appendix (*cf.* literature of S. Weiss).

CLINICAL FEATURES.

The clinical picture presented by intussusception varies in a most remarkable manner in different cases. In order to understand these variations, it is absolutely essential to have a clear insight into the exact conditions that obtain in each individual case, and to understand the

anatomic changes that supervene and that produce, on the one hand, a narrowing or even an interruption in the permeability of the intestinal lumen, and, on the other hand, disturbances in the circulation of the mesentery and of the intestinal wall (strangulation). The different symptoms which develop in different cases will vary according to the presence of the one or the other condition, the combination of these two conditions, and the varying intensity of either one. It can readily be understood that all these possible modifications may produce a great variety of the clinical pictures. The syndrome, moreover, of invagination is typical and distinct, since the intestine produces occlusion of its lumen by its own bulk.

A short sketch of the ordinary symptom-complex presented will be given by way of introduction to a study of the clinical aspects of invagination of the bowel. This sketch is intended to depict, in a condensed outline, the clinical picture most frequently seen. After this the individual symptoms will be more elaborately described, and, finally, the possible differences in the course and termination of this disease will be dealt with.

Sketch of the Clinical Picture.—The onset of intussusception is nearly always sudden. The first symptoms appear unexpectedly and rapidly increase in severity, so that the disease reaches its maximum intensity within a short time. In general the first symptoms appear while the patient is in perfectly good health; in some instances disturbances of the intestinal function—as a rule, diarrhea or paroxysms of colicky pain—precede the onset of the disease. In other cases there is a history of trauma of the abdomen or of some other disease which is regarded as the primary cause—whether correctly or not is in the majority of cases an open question. The initial symptoms of invagination, as a rule, appear suddenly and most unexpectedly; they may either appear while the patient is perfectly quiet, while he is moving about, or even while he is asleep, and in breast-fed infants during suckling. It must be remembered, however, that the symptoms do not always become rapidly worse in cases with a sudden onset, and that a sudden and unexpected appearance of the symptoms does not necessarily determine an acute course; in a certain proportion of the latter cases the subsequent course of the disease is chronic and long-drawn-out; on the other hand, it occasionally, although not frequently, happens that the symptoms of invagination develop slowly and gradually. From the pathogenesis of invagination it can readily be understood why the onset and the formation of the invagination must always occur suddenly—that is, at once; it is not necessary, however, that an invagination should invariably produce distinct and recognizable clinical symptoms directly it is formed. It appears that those anatomic forms of invagination that involve the small intestine (the ileac and the ileocecal form) in the majority of cases develop acutely. The same applies to the great majority of cases of the ileocolic form. The colic form, and in particular the rectal form, on the other hand, very frequently develop slowly and gradually.

The first symptoms noticed in the majority of cases is a sudden violent pain of a colicky character; this pain occasionally seems to radiate from a definite point. Very soon vomiting supervenes. In small children this may be considered to be a constant occurrence. In adults, vomiting is not so frequently seen. Often an evacuation of feculent material occurs once or several times, followed in many instances by tenesmus. Very frequently diarrheic motions of varying character are passed, with or without tenesmus. This diarrhea does not always occur. The motions consist either of pure blood, of blood and mucus, or of mucus alone; or, finally, blood and mucus may be passed together with thin liquid bowel contents. Sometimes no blood or mucus is evacuated, and the dejecta consist entirely of diarrheic motions.

If the course of the case is severe, symptoms of collapse appear very soon—that is, within one to three days; this is seen with especial frequency in children. The pulse becomes small and rapid, the attacks of vomiting recur, even if they intermit for a time. In many cases, when vomiting persists for a long time, it becomes feculent. While fecal vomiting is present no fecal matter is passed by the anus, though a certain amount of bloody or mucous material is evacuated.

Advanced degrees of meteorism are only exceptionally found on examination of the abdomen. In many cases, however, although not always, a distinctly palpable *tumor* of a more or less characteristic constitution develops in the abdomen; if the course is very severe, particularly in children, death frequently occurs at this stage from collapse. In other cases the tumor changes its character and presents a number of modifications and phases which will be described below. A rise of temperature is rarely observed. It will be shown below how the intussusceptum may slough off and be passed by the anus, and how either recovery or death may occur after this event.

In isolated cases the most violent symptoms appear to become less prominent after the first stormy onset of the disease. In these instances the characteristic symptoms of chronic stenosis of the bowel may develop, a condition that in these cases is characterized by the presence of the peculiar swelling mentioned above. In cases in which the course of the disease is longer, the onset of the symptoms is also frequently less severe—that is, the pain is less violent and vomiting and sanguineous diarrhea may be completely absent. In the more chronic cases, however, particularly if the paroxysms of pain are severe and frequent, emaciation and general exhaustion may supervene; this is chiefly due to loss of sleep and impairment of the general nutrition, owing to the difficulty of taking food.

The following case illustrates the gradual development of ileac invagination:

Man, forty-three, mason, apparently always in good health, stools always regular, no history of intestinal disease. January 2, 1900, for first time had frequent short paroxysms of cramp-like pain about the umbilicus. Between attacks he felt perfectly well and was free from any pain. January 3, same as January 2. January 4 pains were more severe and frequent. Lasted even into the night. On the fifth, pains rarely absent, except for short intervals of a few minutes to

half an hour; unable to work; abdomen beginning to swell; **vomiting for the first time.** On the third, fourth, and fifth days he had normal motions; on the sixth, after injection, passed a scybalum; since then no flatus and no passage of feces. Laparotomy on the eighth revealed an invagination of the ileum 20 cm. in length, which was readily reducible and contained a tumor as large as a walnut; histologically a subserous myoma. Recovery.

Individual Symptoms.—Pain.—This symptom is never absent. It constitutes, almost without exception, the first indication of the onset of an acute intussusception, and it is certainly the symptom that is most distressing to the patient in cases that run a chronic course. As a rule, the onset of the pain is sudden and overwhelms the patient while in perfect health. In the majority of cases no disturbances of any kind precede the appearance of pain—it may attack the patients while asleep, even though they were perfectly well when they went to sleep. In those cases in which so-called predisposing or causative factors exist,— that is, in patients with some affection of the bowels (diarrhea, tumors, etc.),—certain painful sensations in the abdomen may be complained of at some previous time, but these painful sensations had nothing whatever to do with the intussusception itself. The intensity of the pain in invagination is occasionally terrible, and so great that adult patients may become collapsed and children may have convulsions. The patients are frequently seen to turn and twist in their agony; small children scream and moan; in other instances the pain is only moderate in severity. In the majority of cases the pain is most intense from the very beginning and overwhelms the patient; in other cases it is less severe at the onset, but rapidly increases in severity, so that the acme is reached within a short time; in other cases—which, however, are not so frequent—the greatest intensity is only reached gradually (see preceding example).

The character of the pain is nearly always colicky; occasionally, however, when the onset of the disease is acute, it may be continuous, particularly in the early stages; later it may intermit. Sometimes the pain is relieved by moderate pressure on the abdomen, so that the patients occupy the most peculiar positions, just as in other kinds of colicky attacks. The colicky nature of the pain is, moreover, manifested by its intermittent character; it may stop altogether, only to return with greater severity after a short time. The exact character of the pain will depend entirely on the subsequent course of the disease itself, and may vary with differences in the morbid process. If the course of the disease is acute, the pain may persist with great severity, only occasionally interrupted by short intermissions, until the death of the patient. Sometimes the pain disappears shortly before death; this may be due either to the paresis of the intestine, which then occurs [to gangrene—Ed.], or, particularly in small children, to loss of consciousness and stupor. In some instances again the pain does not intermit—there are no intervals free from pain, and consequently pain is a continuous symptom. Even in these instances, however, there are usually slight remissions in the severity of the pain, followed in general by an exacerbation which is colicky and paroxysmal in character. Occa-

sionally, in cases where the pain is continuous, it may become greatly increased shortly before the fatal issue. When the invagination of the bowel runs a chronic course, colicky, spasmodic pain may torture the patient for several months, may deprive him of sleep, and may reduce him to a state of great exhaustion. In these instances the pain may intermit for periods of one, two, or three days, only to return, however, with great severity and to torture the victim for from twelve to twenty-four hours at a stretch. Occasionally, the attacks recur in frequent paroxysms from eight to ten days, and then stop for a day or two. Under these conditions increased peristaltic movements of the bowels and stiffening and rigidity of certain loops of intestine are often seen, particularly during the paroxysms. In addition there may be loud gurgling and rumbling sounds in the bowels, and, in short, all the phenomena that are seen in the colicky attacks so characteristic of chronic stricture of the bowel. Sometimes gas or fecal matter is expelled by the rectum and the attack is somewhat relieved in consequence.

Adults frequently point out some strictly localized part of the abdomen as the seat of the very first attack of pain, and are usually very positive in this statement. The most frequent situation thus described is the right iliac region, corresponding to the primary seat of the ileac form of invagination of the bowel. Some patients go so far as to state that they can feel that the pain in all the following paroxysms radiates from this point. Children, as a rule, are less definite in their statements and usually describe the whole abdomen as painful; sometimes they seem to localize the pain around the umbilical region. In some instances there is localized tenderness on pressure in some distinct area of the abdomen; when such a painful pressure-point can be discovered, it frequently indicates the location of the invagination even in the absence of any tumor. Even in the cases, however, in which the starting-point of the pain is strictly circumscribed, the pain generally radiates into different portions of the abdomen and is consequently felt over a wide area.

Some authors state that the attacks of pain are more severe and of shorter duration in the ileac form of invagination of the bowel than in the colic form. Whether this is true remains to be determined by future observation.

The following statements may be made with some degree of certainty in regard to the origin and significance of the pain. The initial pain is apparently a true colicky pain, and is caused by the violent tetanic contractions of the intestine, which are similar to the contractions already described. The same applies to the paroxysms of pain which either accompany the progress of the intussusception or are the result of violent spasmodic contractions of the invaginated area, even when the development of further invagination is arrested and the process is stationary. These violent spasmodic contractions of the bowel may either occur in the swelling produced by the intussusception itself, or in that portion of the bowel situated immediately above the invaginated spot. The attacks of pain that occur in cases of invagination pursuing a chronic course,

and that are accompanied by visible stiffening and rigidity of certain loops of intestine, may be considered as the expression of tetanic contractions. They occur in those frequently dilated and hypertrophic portions of the intestine situated above the invagination. These contractions and the pain produced by them are analogous to the contractions and the pain seen in the loops of intestine situated above a stricture, which were described at some length in the section on Stenosis of the Bowel. The continuous pain that occurs later in the course of the disease is probably related in some way to strangulation of the mesentery and its consequences—namely, hyperemic swelling and peritonitis. Finally, very violent pain occurring late in the disease may be due to diffuse peritonitis.

In a certain proportion of the cases, too, the patients are tortured by **tenesmus**, which is occasionally as severe as in bad attacks of dysentery. It occurs more frequently in the acute than in the chronic form of intussusception, and is particularly frequent in the cases of invagination that occur in the first years of life; in fact, it is usually present in young children. From the fact that motions containing blood are occasionally passed in invagination of the bowel, it can be readily understood that the differential diagnosis between intussusception and dysentery may be difficult; and, as a matter of fact, all the clinical features of dysentery are sometimes so exactly imitated that an erroneous diagnosis is made. After spastic closure of the intestine has persisted for some time, this condition may be followed by paresis of the sphincter, so that the anus remains open and the intestinal contents are passed involuntarily. Many authors formerly believed that this symptom—namely, the patency of the anus—was a characteristic feature of invagination of the bowel; as a matter of fact, however, it has been shown that this is not the case, for the same condition is seen in dysentery and in new growths of the rectum. The period of the disease at which tenesmus appears and the intensity of the suffering from this cause are more or less dependent on the site of the intussusception. The nearer the invagination is to the rectum, the sooner does tenesmus appear, and the more violent will the pain be. Tenesmus develops very early when the invagination begins close to the anus; in other cases it develops as the invagination approaches the rectum. It is very difficult, consequently, to understand the origin of tenesmus in pure ileac intussusception. This symptom has, however, been described in isolated cases of this form of invagination. I believe that in most of these cases it must be assumed that some complication existed.

Vomiting is by no means a constant symptom in intussusception. In some cases it may be completely absent, or, at best, be a very insignificant and unimportant symptom. It occurs more frequently in the acute than in the chronic forms of intussusception, and is much more constant in small children than in adults.

The exact time in the course of the disease at which vomiting occurs may vary greatly. Sometimes vomiting is one of the first symptoms of intussusception, and occurs simultaneously with the initial paroxysm of

pain. In other cases, and especially when the bowel is beginning to become strangulated, vomiting follows very shortly after the onset of the pain. In instances of this kind vomiting must undoubtedly be regarded as a reflex symptom which is analogous to the vomiting that is seen in other forms of intestinal disease with an acute onset. The time at which vomiting occurs seems also, to a certain extent, to be dependent on the site of the intussusception, in so far that the lower down in the intestinal canal the invagination originates, the less likely is vomiting to be one of the initial symptoms; the higher up in the bowel the invagination begins, the more constant is vomiting at the onset. In other instances vomiting occurs irregularly and is not a very characteristic symptom, coming on for the first time after two or three days or even after a fortnight. In other cases it is a very frequent complication, and may be violent and a source of great distress to the patient; it may be combined with hiccup and nausea. In some cases fecal vomiting occurs toward the end of the disease. In cases where death does not occur until two or three weeks after the onset of the first symptoms, vomiting is not necessarily continuous, but may stop for a day or so at a time. When this occurs, false hopes are very apt to be raised. These fluctuations in the frequency and violence of vomiting in intussusception are very misleading. In these fatal cases running a protracted course two factors seem to cause vomiting : In the first place, peritoneal irritation ; in the second place, narrowing or occlusion of the lumen of the bowel. As a matter of fact, it can readily be shown that vomiting is obstinate and pronounced in proportion to the degree of of occlusion of the bowel. The more the passage of the bowel contents is impeded, the more severe is the vomiting. In cases complicated by diarrhea vomiting is less severe and less frequent. In cases of invagination of the bowel, finally, that run a chronic course, vomiting is frequently absent altogether ; at all events, it is relatively less frequent in chronic than in acute cases ; in other words, the onset of the disease may be very sudden, and may be accompanied, as has been shown, by vomiting as one of the initial symptoms ; then vomiting may stop altogether in the cases that run a chronic course and may never recur up to the recovery or the death of the patient. Sometimes in fatal cases vomiting reappears as a distressing and important symptom toward the end ; it is then merely the expression of total occlusion of the bowel or of a terminal diffuse peritonitis. In many of the cases that run a chronic course it will be found that the attacks of vomiting coincide with the onset of the paroxysms of pain which were described in one of the preceding paragraphs.

The composition of the vomit varies greatly. It usually contains gastric contents, mucus, and bile. Fecal vomiting is comparatively rare. In acute or subacute intussusception fecal vomiting appears in about one-fourth of the cases, and occurs still less frequently in chronic cases. Rafinesque's statistics on the frequency of vomiting in those forms of invagination which run a chronic course show that it occurred in only 3 out of 40 carefully observed cases. The higher up in the intestine the invagination, the sooner does fecal vomiting appear. When it does

appear, it is rare before the fourth day, even when the course is acute. The appearance of fecal vomiting so early in the disease must, however, be considered exceptional in the majority of cases; as a rule, it does not appear until the second week, and sometimes not until a very short time before death.

When intussusception runs an acute course, the **appetite** is completely lost, and in those cases that run a chronic course the appetite is at least diminished during the time of attacks of pain.

The **evacuation of the bowels and the character of the motions** are very important in this disease, and are very valuable guides to the diagnosis of intussusception. In order to make the description more comprehensive and clear, the appearance of the feces and the mechanism of evacuation of the bowel will be considered separately in the acute and chronic forms of intussusception.

In those cases of intussusception that run an acute course we may say that the anatomic conditions created constitute an obstacle to the propulsion onward of the intestinal contents proper. This may be considered characteristic for typical cases of acute intussusception; in other words, we may say that there is constipation, or, rather, some obstacle to the evacuation of the bowel. Notwithstanding this constipation or retention of bowel contents proper, we see, in typical cases of acute intussusception, that a certain amount of material is, nevertheless, evacuated; these motions are very frequent, but are by no means fecal in character, but consist of blood and mucus. The passage of the bowel contents from the upper to the lower portions of the bowel may be completely interrupted, so that the normal contents of the bowel as they come from above downward are arrested in some portion of the bowel. This applies to the solid and liquid as well as to the gaseous constituents of the bowel contents. Even under these conditions, however, the intussusceptum and the mucosa of the external cylinder, being hyperemic, swollen, and suggillated, furnish a pathologic secretion, so that a certain amount of material, consisting of blood and mucus, accumulates in the intestinal lumen, is propelled onward and downward, and is finally evacuated by the rectum. In this way the apparent paradox is explained that in acute intussusception diarrhea may be one of the symptoms even though there is occlusion of the lumen of the bowel. This diarrhea, we must remember, is only apparent, the bowel contents proper being really retained, and in reality there is a condition of constipation.

The first motion that is passed after the sudden onset of the initial pain and vomiting, or even a number of motions passed after these events, may still be fecal in character. They consist of the contents of the alimentary canal below the place of invagination at the time when the invagination occurred. As soon as all this material has been expelled, the stools assume a bloody, mucohemorrhagic character. No gas is expelled, and the character of the material deposited is in no way fecal. At this time violent peristaltic movements of the bowel are noticed. They are produced by the irritation that is exercised on the intestine by the swelling formed by the invagination itself. At the

same time, as has been shown in previous paragraphs, the mesentery of the intussuscepted portion of the bowel becomes incarcerated, and it is due to this incarceration of the mesentery that a considerable amount of morbid material, such as mucus and blood, is poured into the intestine and rapidly evacuated. In many of these instances, too, tenesmus complicates the disease-picture. It can readily be understood how this combination of bloody or mucous diarrhea with violent peristalsis and tenesmus may lead to an erroneous diagnosis of dysentery. At best, however, the two disease-pictures are only similar, not identical, for it is well known that in dysentery fecal dejecta are occasionally deposited between mucohemorrhagic motions, a phenomenon that is never seen in typical cases of intussusception.

The more acute the course of the disease, the more violent the strangulation of the peritoneum, the more pronounced will be the **hemorrhages from the bowel.** In a small proportion of the cases— not more than 20 per cent.—blood is always absent from the stools throughout the whole course of the disease. Hemorrhagic motions have been noticed in invagination situated in every portion of the bowel. Some authors state that hemorrhagic stools are most constantly seen in the ileocolic form of intussusception, and are least frequent in the pure rectal form; some go so far as to question the occurrence of bloody stools in the rectal form. The amount of blood expelled with the dejecta varies greatly; the motions may contain only traces of blood, or there may be profuse hemorrhages that in themselves contribute greatly to the fatal issue. If the hemorrhages are profuse, the motions, of course, consist of pure blood; if they are less profuse, the blood is usually diluted with mucus.

Complete interruption of the lumen of the bowel does not, by any means, occur in all cases of invagination of the bowel. In many instances some of the bowel contents can pass through the invaginated portion and be deposited with the stools. In instances of this kind there is a true diarrhea in the full sense of the word, for in addition to the diarrheic consistence of the motions, we see the bowel in a state of violent peristalsis as a result of the invagination. In most of these instances the motions contain some blood and mucus. The number of daily evacuations vary, for there may be ten or as many as twenty motions in twenty-four hours.

In the later course of the subacute form of intussusception the attacks of **diarrhea** may subside for a time, only, however, to return with greater frequency and greater violence as soon as the gangrenous intussusceptum begins to be eliminated. As soon as this occurs, the motions frequently emit a gangrenous odor. Careful macroscopic or microscopic examination of the stools in these cases will usually reveal the presence of shreds of tissue, or occasionally, as I have already said, of completely preserved pieces of the intestinal wall.

The character of the motions varies still more in chronic intussusception. Treves expresses himself dogmatically in regard to this point as follows: " The only thing that we can be certain about in regard

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to the evacuation of the bowels in invagination is that this factor is very uncertain."

Rafinesque has collected 46 cases of chronic intussusception, and has studied the stools in all these cases with the following results: In 7 instances the stools were normal; in 16 cases diarrhea predominated; in 12 cases constipation was a marked feature; and in 11 instances diarrhea and constipation alternated. Another question remains to be decided, viz., whether the exact seat of the invagination in the bowel in any way determines the character of the evacuation. The only method by which this question can be decided is to collect a large number of statistics or to study a large number of cases in which the intussusception was located in some one point of the intestine and in which the character of the stools was the same. Unfortunately, we do not posses data of either kind, so that it would be useless to enter into a discussion of this point. I need hardly emphasize the fact that in these cases of chronic intussusception the lumen of the intestine is not completely occluded. Cases of this kind run a course that is altogether similar and analogous to chronic stricture of the bowel, the only difference being that in the one instance the chronic narrowing is brought about by the intussusception; in the other, by stricture or constriction of the bowel. Rafinesque remarks expressly that in these instances the administration of laxatives is, as a rule, successful. In chronic intussusception blood is also occasionally found in the dejecta, but far less frequently than in the acute form—probably only in from 45 to 50 per cent.

An examination of the abdomen generally fails to reveal any constant or characteristic phenomena. I have repeatedly observed in these cases that the whole abdomen appears retracted immediately after the occurrence of the invagination. This appears to be the result of reflex contraction of the abdominal muscles. In other cases again no retraction of the abdomen is noticed immediately after the onset of the invagination, and the belly looks altogether normal. Dance has reported a symptom of this condition that is named after him—namely, a flattening of the ileocecal region. This symptom, however, is exceedingly rare. Rafinesque studied 53 cases of chronic invagination of the bowel statistically, and found that Dance's symptom was present only in 2 instances. Meteorism is another symptom that is quite inconstant and variable in chronic intussusception; consequently it may be considered as valueless in making a diagnosis of this condition. The variations in the amount of meteorism or its complete absence in certain cases can readily be understood, for the development and the degree of meteorism in any given case are dependent on the amount of interference with the passage of the bowel contents and the degree of constriction of the bowel lumen. These two factors vary in individual cases. The development and the degree of meteorism, moreover, are also to a certain extent dependent on the presence or absence of diarrhea, and in part on the exact location of the invagination, all factors that are uncertain and that vary in each case. If there is profuse

diarrhea, we may even find the abdomen flattened. This flattening of the abdomen may, under certain circumstances, be considered a fortunate event so far as the diagnosis of invagination of the bowel is concerned, for whenever the abdomen becomes flattened, a symptom can usually be discovered that absolutely clinches the diagnosis; this symptom may, in fact, be considered the most significant sign of intussusception, provided it is desirable to bestow the dignity of an absolutely diagnostic sign on any one manifestation of this disease; I refer to the appearance of a tumor.

Tumor.—A tumor of the abdomen that is due to intussusception differs in some points so radically from all other forms of swelling that may occur in the abdominal cavity that it usually enables us to recognize the disease with absolute certainty—provided, of course, that it can be palpated. Unfortunately, this is not always the case. Leichtenstern examined the reports of 433 cases of intussusception, and found that a tumor was mentioned in 222 cases; Rafinesque examined 53 reports of chronic intussusception and found that there was a tumor in 24. The absence of a tumor in many of these cases can be explained in different ways: sometimes the tumor cannot be felt for external reasons —the abdominal muscles, for instance, may be too tense to permit palpation of the abdominal contents, or they may be so thick, owing to the presence of a large adipose layer, that a serious obstacle is offered to palpation; in other cases the tumor may be situated behind the costal arch, may be hidden behind the liver, or, finally, may be covered by coils of distended intestine; in still other cases the process of intussusception is of such short duration or the strangulation and the incarceration of the mesentery with all its results so insignificant that no pronounced tumor formation occurs. In children tumors are, as a rule, more pronounced and more frequent than in adults. When the tumor is present and can be palpated, it presents the following characteristics:

It varies greatly in size, for it may be only as large as an egg or it may be as long as an adult forearm. Tumors of the smaller variety are more frequent. One point must not be overlooked in estimating the size of the invagination tumor, namely, that it may occasionally appear to be shorter than it really is. This impression is created principally in the ileocecal form of invagination, for here a large portion of the tumor may be hidden behind the right or the left hypochondrium. If the tumor is located in this position, and if it is very long, it may occasionally also impart the impression of a double swelling, particularly if the middle of the tumor is covered by the splenic flexure of the colon. Conversely, a chronic tumor from invagination of the bowel may appear larger than it really is, owing to the accumulation of fecal material above the constricted area.

The form of the swelling produced by an intussusception is usually cylindric or sausage-shaped; the tumors are generally elongated and somewhat curved. (This is the result of the traction exercised upon the invaginated portion of the bowel by the mesentery; see above.) The outlines of the tumor are usually indistinct, although in some in-

stances they are strictly defined, so that the tumor can be palpated in all directions and can almost be grasped with the hand.

No definite statements can be made in regard to the resistance offered by the tumor to the palpating hand. As a matter of fact, one of the most characteristic features of this form of abdominal swelling is that the consistence, and consequently the resistance offered, varies greatly at different times in the same patient. Generally speaking, we may say, however, that these tumors never become so hard as carcinomata of the bowel, and that even those swellings that offer the greatest resistance to the palpating hand are never stonily hard, but always yield a little and can always be slightly compressed. The following phenomena are sometimes observed : A tumor may be very hard, and yet become comparatively soft in a very few minutes ; the tumor may in this way vanish in a short time, and it is frequently surprising to palpate a hard, distinct tumor and to find, a few moments afterward, that no tumor-like resistance whatever can be felt in the same region. In order really to determine this point it is necessary to approach the palpation of the abdomen without preconceived ideas and without prejudice, for otherwise it is a very easy matter to imagine the presence of a tumor in the area where it was a few moments before. The disappearance of the tumor and the change in the consistence and resistance of certain portions of the bowel are due to tetanic contractions of the intestinal wall. It is perfectly correct, as some authors have said, that the absence of a swelling should never be positively asserted in cases of intussusception unless the patient is examined during a paroxysm of pain, for such a paroxysm is, in reality, the expression of a tetanic contraction of the bowel. In addition to these intermitting tumors of the intestine there are often others which are due to invagination of the bowel and that persist permanently ; tumors of this kind offer a moderate degree of resistance to the palpating fingers. They may be altogether painless in the intervals between the typical paroxysms of pain that we have described, and hurt only during the paroxysms themselves. They are usually both spontaneously painful and painful on pressure during the paroxysms. In other cases, when local peritonitis complicates the disease, they may be constantly tender on pressure.

The position of the tumor in the abdomen varies greatly. There are, however, certain points of predilection in which the tumor is most frequently found. The position of the tumor naturally depends on the exact anatomic form of invagination and the manner of its development. Leichtenstern has arranged a tabulated statement in regard to the position of the tumors produced by intussusception ; from these statistics we learn that the tumor is most frequently situated in the region of the sigmoid flexure ; then, in the order of frequency, in the following positions : protruding and prolapsed from the anus ; in the rectum ; in the region of the cecum, of the descending colon, of the transverse colon, of the ascending colon ; in the hypogastrium (the tumor is least frequently found in this position, and only in invagination of the ileum). I do not wish to convey the impression that the

tumor during life is always found in the portion of the abdomen that corresponds to the exact anatomic position of the portion of the intestine that is involved ; such a conception would be altogether erroneous. I have observed cases, for instance, in which an elongated tumor of the right iliac region extended upward and to the left toward the umbilicus and beyond.

A very striking symptom that is observed in some cases of invagination of the bowel is prolapse of the swelling through the anus. A tumor that appears in this way probably always represents an ileocecal or a colic form of intussusception. This accident may occur both in the acute and in the chronic form of this disease. In the chronic form it is often interesting to watch the gradual protrusion of the tumor ; the tumor may appear at the anal orifice and then recede, only to reappear ; this process is repeated for two or three days until, finally, the prolapse of the tumor is complete. In general, the prolapsed tumor is short (only about 5 cm.), but it may grow to 20 or 30 cm. The appearance of the tumor is dark and hyperemic, or it may show inflammatory changes, or may even be gangrenous. An orifice is often seen at the apex of the swelling ; this is the ileocecal ostium ; close by a second opening is occasionally presented to view, which is the entrance to the vermiform appendix. If the tumor is still situated in the rectum, but so far down that it can only just be reached with the finger, a peculiar impression is imparted to the palpating finger, like that of the os uteri as it is felt during a vaginal examination. Tumors of the bowel that are due to intussusception and that are found in the rectum, or that have prolapsed through the anus, have frequently led to errors in diagnosis ; they have repeatedly been mistaken for malignant or benign neoplasms or for simple prolapsus ani, and have sometimes been operated upon as piles.

The change that is occasionally observed in the resistance of tumors that are due to invagination of the bowel was mentioned in one of the preceding paragraphs, where it was also stated that, under certain circumstances, complete temporary disappearance of the tumor may occur. Attention may here be called to another peculiar feature of these tumors. The tumor produced by an intussusception may remain permanently in one position ; in fact, if the disease runs a chronic course, the tumor may become tightly fixed and anchored by adhesions, so that it constitutes an immobile swelling in one strictly circumscribed portion of the abdomen. In these instances a tumor from invagination acquires a property that is common to all tumors of the intestine with the exception of tumors of the rectum, for it acquires passive mobility to a greater or smaller degree ; in other words, while the tumor itself is anchored and fixed at one point of the abdomen, it can readily be moved to and fro by the hand of the examining physician. In addition, a tumor of this kind may actually migrate, so that, for instance, a tumor that was located at one time in the ileocecal region is subsequently discovered in the transverse colon, in the sigmoid flexure, in the rectum, or at the anus. This advance of the tumor is readily explained by the fact that a swelling of

this kind may greatly increase in size. This is occasionally seen if the disease runs a chronic course,—even several weeks after the onset of the intussusception,—but it is more apt to occur in cases that run an acute course. In the ileocecal form of invagination of the bowel, particularly in cases occurring in children, the tumor may advance so rapidly that it appears in the rectum on the second day after the onset of the invagination. Occasionally we also observe a retrograde movement. This peculiar development usually occurs spontaneously, and is in part due to loosening of the intussusception, an event that may either occur spontaneously or after the administration of enemata.

In conclusion, one other symptom must be mentioned that occurs in intussusception—namely, **stiffening and rigidity of certain loops of intestine** with increased peristaltic movements of the bowel. These phenomena are observed in the portions of the intestine that are situated above chronic stationary invaginations. Their genesis is the same as in chronic stenosis of the bowel. The same phenomena have also been described in a few exceptional cases that run an acute course. One author reports the occurrence of increased peristalsis and stiffening of loops of intestine in a case that lasted eleven days, and another one in a case that lasted only six days.

[Eve has pointed out[1] that there is a variety of intussusception in which the invagination begins at the caput cæci, or free end of the cecum, instead of at the ileocecal orifice. The free end of the cecum is the last part to be reduced in these cases, and in one instance he found it so hard and board-like from extravasation and edema that sutures had to be passed across it in order to maintain it in its natural position after reduction. The symptoms, of course, of this variety do not differ from those of the more usual forms of intussusception.—ED.]

COURSE AND TERMINATIONS.

The course of intussusception in the great majority of cases is acute, but in a small minority the condition constitutes a chronic disease. Rafinesque subdivides the acute cases into three groups—namely: (a) Very acute or fulminating cases, which terminate fatally within the first twenty-four hours; (b) acute cases which persist for a week; (c) subacute cases which run a course of four weeks. This more or less arbitrary method of grouping the cases is, nevertheless, of some practical value from a clinical point of view, for it includes under one heading at least the great majority of the cases that figure in the different groups. It is true that sharp and definite distinctions cannot be made, and, as a matter of fact, the different subforms of the disease merge into one another.

Cases that run a fulminating and always fatal course are the rarest. Leichtenstern, from his table, shows that this form occurred only 5 times in 269 cases: 4 of these 5 cases occurred in infants under one year; 1 in an adult. In the acute group the cases that terminate fatally

[1] *Brit. Med. Jour.*, 1901, vol. ii., p. 582.

predominate, particularly in children under one year. Such young subjects are, as a matter of fact, the most frequently affected, and 80 per cent. die, according to Leichtenstern's statistics. The subacute cases—that is, the cases that run a course of from two to four weeks—are most frequent in persons above ten years of age. This category offers, relatively speaking, the most favorable prognosis, and the majority of the cases of spontaneous cure that are reported all belong to this group.

[A case of spontaneous reduction of an ileoileac intussusception occurred at St. Bartholomew's Hospital on February 23, 1903 :

A boy, aged six months, was suddenly seized with abdominal pain on February 20th at midday. The pain continued, and he passed blood-stained mucus on February 21st. He was admitted into the hospital late at night on February 22d, and was brought to the operating theater at 1 A. M. on February 23d. The abdomen was then somewhat distended, and a tumor seemed to be present in the right hypochondriac region. The abdomen was opened in the middle line, above the umbilicus, and an examination of the small intestine at once revealed a piece of deeply congested small intestine, thickened throughout by exudation to the consistence of thin parchment. The inflamed portion of the intestine measured about 2 inches in length, and was marked near its middle by a more constricted portion. The inflammation was strictly limited above and below, and there was no peritonitis. The rest of the intestine seemed healthy, and there was no swelling in the ileocecal region, but this portion of the bowel could not be drawn up into the wound. The wound was sewn up, and the patient was sent back to the ward in less than a quarter of an hour from the time the operation was commenced.—ED.]

The last category, finally, of cases that run a chronic course, again offers a very unfavorable prognosis, and only a small percentage of these cases terminate favorably. One point, however, must be distinctly emphasized in studying these statistics—namely, that the tables were arranged before the introduction of antiseptic methods ; this refers particularly to the tables of Leichtenstern and Rafinesque. To judge from my recent personal experience, the prognosis of these cases is far more favorable nowadays, as operative interference is so largely perfected that surgical interference can be instituted with benefit [at the earliest possible moment.—ED.].

The termination of cases of intussusception is either in death or cure.

Death may occur in a great variety of ways as a result of intussusception. In those cases of invagination of the bowel that run the most rapid course death occurs after symptoms that resemble shock or after symptoms of collapse that develop very rapidly. This termination is seen particularly in cases of incarceration of the bowel. The symptoms of shock and the collapse are produced by the strangulation that occurs in this condition. Another factor that probably produces shock and collapse, particularly in the early years of childhood, is the sudden and violent pain that is experienced in incarceration of the bowel and the tremendous reflex effects that this pain has on the general nervous system. These factors are considered in the paragraph on the general symptoms of occlusion of the bowel.

In a second series of cases the fatal issue is chiefly brought about by the occlusion of the intestine itself. The symptoms of occlusion of the bowel are often complicated by symptoms that are due to the strangula-

tion of the intestine that usually occurs at the same time. In the section on the Anatomy of Invagination of the Bowel attention has already been drawn to the fact that the primary process leading to the development of occlusion of the intestine is not always the same in every particular in different cases. The different possible factors that can be operative in this direction will be discussed here briefly.

The angular knuckling of the intussusceptum that is produced by contraction of the mesentery may become so severe that the intestinal lumen is no longer permeable for bowel contents.

The same factor can, by its peculiarly mechanical action, dislocate the axis of the intussusceptum in such a way that it becomes eccentrically placed. Whenever this occurs, the lumen is converted from a round opening to a mere slit, and may, in addition, become directed toward the wall of the intussuscipiens in such a way that the passage of bowel contents again becomes impossible.

The whole intussusceptum or only certain portions of the intussusceptum near the head of the invagination may become so swollen and thickened that an almost complete occlusion of the bowel occurs. This swelling and thickening are, of course, the result of venous stasis and of peritonitic inflammation. The latter is undoubtedly the most frequent cause of occlusion of the bowel both in cases of acute and of subacute intussusception. Sometimes it will be found that the swelling of the tissues alone does not produce complete occlusion of the bowel, but that this effect is produced by a number of different complications which exist at the same time, as, for instance, the presence of a tumor near the apex of the invagination, blood-clots, some gangrenous shreds of tissue, or some solid ingestum present in the intestinal contents above the invagination.

All these causes of total occlusion of the bowel are chiefly operative in acute and subacute cases of invagination. In cases that run a more protracted course these causes rarely produce this effect. Only in exceptional cases does occlusion of the bowel occur in any other way— as, for instance, by secondary cicatricial stricture of the bowel following sloughing of the intussusceptum.

Another complication of intussusception that is much more serious and dangerous to life may also occur, namely, gangrene of the intussusceptum. If the intussusceptum sloughs off before a sufficient number of adhesions have formed near the neck of the invagination, perforation of the bowel occurs, followed by fatal peritonitis. Peritonitis may also develop without perforation directly of the gangrenous portion of the bowel and before sloughing occurs. The exact period at which peritonitis occurs depends largely on the rapidity with which the gangrenous process develops. If the gangrenous process develops rapidly, peritonitis, of course, occurs earlier than when the gangrenous process develops slowly. At all events, peritonitis, when it does occur, is always seen in the first weeks of the disease. Death from peritonitis usually occurs in the first week in children, usually in the second week in adults, less frequently in the third or the fourth week.

The form of peritonitis, however, which is due to sloughing usually develops in the course of the third week of the disease. It has been stated above that by far the greatest number of children under two years old who develop an intussusception die during the first week. When it is remembered, therefore, that peritonitis following sloughing usually occurs in the third week, it will be seen that in children death from peritonitis following sloughing of the intussusceptum after the first week must be a rare cause of death.

Gangrenous sloughing of the intussusceptum may lead to still other complications that endanger the life of the patient. Clinical observation shows that in individual cases the following sequence of events may develop : In the course of about the third week the gangrenous separation of the intussusceptum occurs, so that it sloughs off from the neck of the invagination. In this way it may happen that the whole intussusceptum remains more or less intact and constitutes a well-marked uniform and connected mass. Peritonitic fixation may have occurred at the neck of the invagination, but the adhesions may not be sufficiently firm to withstand great demands on their cohesive powers. As soon as the intussusceptum sloughs off, the lumen of the bowel becomes free and the bowel contents are propelled onward by the energetic peristaltic movements that immediately begin. The intussusceptum that is sloughed off advances in the lumen of the intestine, and in itself more or less obstructs the propulsion of the bowel contents. At the same time it constitutes an irritant to the bowel-wall, and stimulates active and violent peristaltic movements of the bowel-wall that are intended to aid in the propulsion onward of the intussusceptum. All these factors combined may lead to rupture of the adherent portions of the bowel, especially if these adhesions are not very firm. In this way peritonitis may be produced.

The gangrenous process may indirectly cause the death of the patient still later in the disease. Septic processes may originate from the point of sloughing, so that phlebitis is produced in the mesenteric veins, followed by thrombosis that extends far up into the vessels. Another sequel of these septic processes may be the development of sacculated peritonitic abscesses. In other cases an ulcerating surface remains that leads to colliquative diarrhea or finally to perforation of the bowel. Finally, the circular surface of the ulcer may heal, cicatrize, and in this way possibly lead to the development of stenosis of the intestine. One other possibility must be mentioned that may occasionally lead to the death of the patient, or at least precipitate the fatal issue in individual cases, namely, hemorrhage from the gangrenous portion of the bowel.

An invaginated portion of the bowel may become reduced and may heal. This has been known to occur. From the description of the ordinary course of the disease and its complications in the preceding paragraphs it will be seen, however, that a fatal issue is more frequent than a cure, even when artificial means are employed to arrest the progress of the disease. The prognosis, therefore, of invagination of

the bowel, generally speaking, is very bad. Nevertheless, a number of cases are on record, particularly in adults, that were cured, some of them without any interference from without; in other words, we may say that a spontaneous cure of this disease is actually possible.

A complete cure, however—a *restitutio in integrum*—is possible only under one condition—namely, in the absence of firm peritoneal adhesions, or at least when the peritoneal adhesions that have developed are insignificant and can readily be torn. There can be no doubt whatever that an invagination of the bowel may be completely corrected and the intussusceptum replaced. I do not refer to the cases of physiologic intussusception that are altogether analogous to the pathologic cases, but will only mention instances that are on record in which the diagnosis of invagination was positively made and the whole train of symptoms characteristic of intussusception was completely developed; in which, moreover, a tumor could be felt in the rectum; such cases, nevertheless, have been completely cured. In addition, it must be remembered that a few instances have been described in which distinct symptoms of intussusception preceded the death of the patient, though nothing was found at the postmortem examination but contraction of a circumscribed portion of the bowel. Treves assumes that spontaneous reposition of an intussusception can occur only in very recent cases, and probably only in invagination of the small intestine. Rilliet and Rafinesque have both reported a case of invagination of the bowel in which spontaneous reposition of the invagination occurred after the disease had persisted for some time. Treves, however, does not consider the evidence adduced by these authors in support of these statements to be altogether valid. In view of the fact, therefore, that this subject is still a matter of considerable controversy, the following remarkable case which I had occasion to observe myself may be briefly reported :

The patient was a man aged fifty who had always been healthy, except for a gunshot wound that he had sustained in the shoulder many years before. The patient always enjoyed a regular daily evacuation of the bowels until the onset of his attack. In April, 1892, while he was a member of a Military Commission and was attending to his functions in the office, he was suddenly attacked by a violent colicky pain which seemed to radiate from the ileocecal region. He was forced to return home at once and go to bed. Spasmodic colicky pain similar to the first attack recurred at intervals of from one to three days for nearly four months thereafter, each attack lasting from twelve to twenty-four hours. The paroxysms frequently became exceedingly violent and led to vomiting and nausea. Gradually the patient noticed the appearance of increased visible peristaltic movements in his abdomen and the appearance of rigid and stiff loops of intestine, particularly in the right lower half of the abdomen. The patient described this phenomenon very precisely and carefully. A daily evacuation of the bowels occurred during all this time. The time of defecation was quite regular and the stools were perfectly normal. A number of different plans of treatment were instituted, but no change was observed in the patient's condition; he gradually became more and more emaciated. Toward the end of June he was admitted to the Vienna Hospital and the diagnosis of an abdominal new growth was made. Surgical interference was proposed, but the patient refused to undergo an operation. A peculiar phenomenon was now noticed, namely, that as soon as he entered the hospital all the symptoms seemed to improve without any treatment. The improvement was so marked that the patient was able to leave the

hospital, free from all distress, four weeks after he was admitted. For a year and a quarter thereafter he enjoyed the best of health, so that he was even able to undertake arduous mountain tours. In November, 1893, all the old symptoms, however, suddenly returned; they persisted without change and were as constant as before, and finally the patient suffered so much from loss of sleep, pain, and vomiting, and emaciated so much owing to deficient nutrition and his inability to take food, that he entered my clinic on April 16, 1894—that is, five months after the recurrence of the disease. An examination of this patient revealed the classic picture of tumor of the intestine due to intussusception, notwithstanding the fact that the stools were normal and that the evacuation of the bowels occurred daily. The tumor possessed all the properties that are described above as characteristic of an intussusception. It was situated in the part of the abdomen which corresponds with the transverse colon. In addition to the tumor there were greatly increased peristaltic movements of the bowels and stiffening of certain loops of the small intestine. The patient complained bitterly of violent attacks of colic with vomiting. He was dismissed from the hospital at this time at his own request. A short time thereafter, however, the pain became so insupportable and he suffered so much that he consented to an operation. When the abdomen was opened, a very peculiar condition of affairs was found, namely, an ileocecal intussusception at the apex of which an intestinal polypus was seen. No trace of peritonitis could be discovered, the two surfaces of the serous coat were not adherent to each other, and the invagination could be reduced without any difficulty. The patient recovered from the operation, but no details of his after-career are attainable.

This case is very remarkable in two respects : in the first place, it is astonishing to find that the peritoneum was not affected in any way, notwithstanding the fact that the invagination had persisted for so long a time. In the second place, the spontaneous reduction of the intussusception in the first attack of illness (April to June, 1892) is remarkable, for there can be no doubt that in this first period the same conditions obtained as in the second period. This assumption at least is so probable that it is almost a certainty.

Another way in which a spontaneous cure may be brought about is the following: the intussusceptum may slough off as a result of a gangrenous process. This method of cure is not really a complete restoration to the normal, but it is essentially synonymous with this, inasmuch as it reëstablishes normal function, and in this way relieves the patient of all distress. Allusion has already been made to the great dangers which accompany gangrenous separation of the invaginated portion of the bowel; at the same time, however, it must be remembered that the same process—namely, gangrene—may, under favorable conditions, lead to a cure of the disease. The presence of peritoneal adhesions that are sufficiently firm is a *sine qua non* for so favorable an issue in gangrene.

An autopsy performed by F. Wechsberg shows what anatomic peculiarities occasionally result. The ileum, after the separation of the intussusceptum, did not anastomose with the cecum, but with the side of the colon at a point much further on, thus eliminating a large portion of the intestine. (For a further description of this unusual case the reader should refer to the original.)

There is danger, after recovery, of the formation of stricture from cicatrization, which has been described.

A few statistics on spontaneous separation of the intussusceptum

drawn from Leichtenstern's tables may be here brought under review. The position of the invagination seems to exercise a great influence on the spontaneous sloughing of the intussusceptum, for by far the greatest percentage (61 per cent.) of such cases were observed in the ileac form of intussusception, only 28 per cent. in the colic form, and not more than 20 per cent. in the ileocecal form. The age of the patient seems to exercise a still greater influence on the frequency of sloughing of the intussusceptum in invagination. Only 2 per cent. of all the cases of spontaneous sloughing occurred in the first year, notwithstanding the fact that invagination of the bowel is, absolutely speaking, very frequent at this period. Only 6 per cent. of the cases occurred between the second and the fifth year. This rather striking disproportion is not so difficult to explain as it might appear at first sight. The solution of this problem is very simple, for in very young subjects death from intussusception usually results so soon that no time is given for the spontaneous elimination of the intussusceptum.

These remarks on intussusception may be fittingly concluded by adding a few paragraphs on chronic intussusception. This is a subject that Rafinesque has studied with particular care. The case just reported is a very interesting example of this condition, and as the most important features of the symptomatology of this disease have already been described in the preceding paragraphs, only a few remarks are necessary here. The description furnished by Rafinesque is followed in its broad outlines.

All the different anatomic forms of intussusception may pursue a chronic course. This course is, absolutely speaking, least frequently seen in the ileac form of invagination, most frequently in the ileocecal form. The onset of this form of invagination varies; the process frequently begins acutely, and later on pursues a chronic course. The course and termination are dependent on a variety of factors; in cases, for instance, in which the anatomic changes are only moderate in degree and in which the incarceration of the mesentery is not severe, gangrenous processes are not so apt to develop, and the different component parts of the tumor gradually decrease in size, with or without the development of adhesive peritonitis. When this occurs, the disease runs a chronic and protracted course. In other cases again the disease develops slowly from the beginning. We are justified in assuming that this actually occurs, in so far, at least, as the intensity of the clinical symptoms enables us to draw any conclusions in regard to the severity of the primary state. Under these conditions we must assume that the abnormal anatomic changes which occur develop very slowly. The course of the disease may be prolonged for many months and even years. Pohl reports a case of invagination of the bowel that persisted for eleven years. The clinical symptoms are either so well pronounced that a diagnosis can be made without difficulty, or they are so obscure and indefinite that the diagnosis becomes mere guess-work. All the signs that I have described in the paragraphs on the individual symptoms of invagination of the bowel may be present, may be grouped in

various ways, or may be absent. At the same time the sequence of events, so far as the time of their occurrence is concerned, may also vary greatly. In the great majority of cases the group of symptoms presented by chronic invagination of the bowel corresponds more to the picture of chronic narrowing of the intestine of moderate degree than to any other condition. This similarity is further alluded to under the head of diagnosis, in which the characteristic features of chronic invagination of the bowel are discussed. The prognosis, as has already been stated, is usually bad unless surgical treatment is adopted.

DIAGNOSIS.

It may be quite impossible in many cases to make a diagnosis of this variety of occlusion, or better stenosis, of the bowel. Comparatively speaking, however, the diagnosis is easier than in any other variety of intestinal obstruction, and occasionally it is possible to make a very positive diagnosis.

The diagnosis of the acute form of invagination of the bowel—by far the most frequent variety—is based on the following symptoms : pain, surprising the patient suddenly while he is in perfect health ; vomiting ; occasionally tenesmus ; bloody and mucohemorrhagic stools or occasionally simple diarrheic stools ; then, symptoms pointing to complete occlusion of the bowel—*i. e.*, complete retention of feces and flatus, combined with relatively slight flatulent distention of the belly ; finally, the appearance in the abdomen of a characteristic tumor with all the peculiarities described elsewhere. The latter sign—viz., the appearance of a typical tumor—makes the diagnosis almost positive.

The diagnosis of the chronic form is much more difficult ; according to the instructive dissertation of Rafinesque, this chronic form may be confounded with a great variety of other diseases. It is easy to understand how errors of diagnosis may be committed, especially in the absence of an intussusception tumor, for the latter sign is to be considered the most pathognomonic one of chronic invagination of the bowel. When such an abdominal tumor, however, is palpable, a careful study of all the other features of the case and a thorough examination of the patient should, in the great majority of cases, lead to the correct diagnosis.

[The author very properly lays great stress upon the pathology and diagnosis of intussusception, but he hardly alludes to the remarkable change in the treatment of the condition which has taken place within the last few years. Intussusception occurs chiefly among the children of the poor, and the large majority of cases are met with in hospital practice. Hospital records, therefore, reflect faithfully the change of practice. Until 1882 cases of intussusception at St. Bartholomew's Hospital were admitted into the medical wards, and were only occasionally brought under the notice of the surgeons. A few cases recovered spontaneously at first, and after the use of enemata at a later period. Enemata, however, were soon found to be dangerous for children with

intussusception, as they occasionally caused rupture of the intestine, and this method was replaced by that of irrigation. Irrigation consists in introducing, through the anus, a nozzle connected with a piece of India-rubber tubing eighteen inches long, which is furnished with a glass funnel. Warm saline solution, milk, oil, or other bland fluid is poured through the funnel until the intussusception is reduced. The method was employed with an occasional primary laparotomy until 1897. The chief objection to it lies in the fact that it is impossible to tell how much reduction has occurred, and recurrence has, therefore, been frequent in spite of all precautions, and even when the irrigation has been performed by those with large experience. Irrigation, too, is of service only in the very simplest cases of intussusception and at the earliest period. Since 1897 all intussusceptions have come into the hands of the surgeon, who has not hesitated to open the abdomen and reduce the intussusception as soon as possible after its occurrence, for he judges rightly that the sooner the operation is performed, the more likely is the patient to recover. It is to be hoped, therefore, that the cases of chronic intussusception described above as lasting for weeks and months will become purely historic, and will never more be seen in contemporary practice.—ED.]

INTERNAL HERNIAFORM INCARCERATION OF THE INTESTINE (Incarcerationes Internae Herniaformes Intestini).

ANATOMY.

THE form of occlusion of the bowel dealt with in this section may be due to a variety of anatomic lesions. These different forms not only can, but must, be described from a common point of view, since the mechanism by which occlusion of the bowel is brought about in them all is essentially the same, and closely resembles, or may even be identical with, the mechanism which causes strangulation in the so-called external hernias. In the first place, therefore, the two varieties of intestinal hernia are pathogenetically related ; and, in addition, their clinical symptoms and course are identical. The same may be said of their prognosis and treatment. Their resemblance goes still further, for in both the same part of the bowel—namely, the small intestine—is involved, the colon only being affected in rare instances.

All the data bearing on the explanation of the finer mechanism of strangulation of external hernias apply *mutatis mutandis* to that of internal hernias. Pathogenetically speaking, the development of the general symptom-complex is the same in both. It is customary to regard one form—namely, internal hernia—as belonging to the physician's province, while the other is assigned to the surgeon. The reason for thus dogmatically arranging these two conditions under different categories is apparent.

The subject-matter of the following paragraphs may be arranged in different groups. From a purely anatomic point of view, a subdivision

can readily be made without attempting any forced classification ; in fact, the writers of the leading systematic treatises on this subject have formulated differences that are based on anatomic variations. They do not all adopt exactly the same classification that I do, but the differences are very slight. The description and classification of the different anatomic varieties of this disease given by Leichtenstern and Treves coincide essentially with those given here. I do not intend to review all the detailed statements scattered through the literature with regard to this disease, nor to write a historic monograph ; my chief object is to give a clear clinical description of internal strangulation of the intestine. No detailed description, therefore, will be given of all the anatomic differences which have from time to time been described in the various forms of internal hernia of the bowel. Anatomic descriptions are exceedingly interesting from a scientific point of view, but many of the details given by various authors are devoid of practical significance ; some of them, in fact, are medical rarities. All these details will be omitted from this description, more especially as no general principles or information of practical value can be gained by considering them. The account of the pathologic anatomy of internal strangulation of the bowel will, therefore, be confined to a brief summary of its extensive literature. (For the anatomic details I refer those of my readers who are particularly interested in this subject to the original monographs.) This limitation will enhance the clearness and the comprehensiveness of this description and make it of more practical value to medical men.

The different anatomic lesions that can produce internal hernia and strangulation of the bowel may be most conveniently and naturally subdivided in the following groups :

1. Strangulation by bands, etc., the result of past peritonitis.
2. Strangulation by Meckel's diverticulum.
3. Strangulation in slits and apertures.
4. Strangulation of internal hernias.

By far the largest proportion of cases are included in the first group ; the remaining cases are distributed through the other three groups in a descending order of frequency.

STRANGULATION BY ADHESIONS DUE TO PAST PERITONITIS.

On p. 470 of this work attention was called to the fundamental importance of chronic peritonitis in the pathogenesis of chronic stenosis of the bowel, and to the numerous and varying anatomic lesions which can produce this condition. Chronic peritonitis is an equally prolific source of internal herniaform incarceration of the bowel. The various possible methods by which chronic peritonitis can produce this lesion may be summarized as follows :

(*a*) Strangulation may be due to isolated peritoneal adhesions. As the genesis and nature of these adhesions will be described in detail in the section on Peritonitis, the following brief statement only is necessary

here : These adhesions are solitary bands or so-called false peritoneal ligaments. In many instances they are rounded and form cords. Their circumference varies ; some are no thicker than a thread, while others are as thick as a finger. Occasionally they are flattened and ribbon-shaped, and may be from 1 to 2 cm. broad or even broader, and vary greatly in length. Cord-like adhesions of 44 cm. in length have been reported—thus one extended from a loop of the small intestine in the epigastric region to the parietal peritoneum of the inguinal canal. There may be either a single band or cord of this character, or there may be a number of them at the same time. Any one of them may be the primary cause of strangulation of the bowel. A careful review of the literature shows that a few very rare cases are on record in which, wonderful to say, two peritoneal cords which were quite independent of each other produced strangulation of the intestine in two different places. [Lupton [1] reported a case in which the intestine was constricted in four places by separate bands ; only one was found at the laparotomy.—Ed.]

It is clear that in order to produce herniaform strangulation of the intestine a cord-like adhesion of this character must have two firm points of attachment, which may, of course, be in any part of the abdominal cavity. Treves has well characterized the almost boundless possibilities in this direction as follows : "Every connection between any two points of the abdomen that can possibly be connected will be found described in the literature on this form of intra-abdominal adhesions." In order to illustrate the variety of possible connections of this kind that may exist, I may mention adhesions between the femoral or inguinal ring of an external hernia, on the one hand, and the intestine, the mesentery, and the posterior parietal peritoneum, on the other ; between the uterus, the ovaries, and the bladder, on the one hand, and the parietal peritoneum of the pelvis, an external hernia, and the intestine, on the other ; between the tissues situated in the neighborhood of the cecum, on the one hand, and the parietal peritoneum or the intestine, on the other ; between different coils of the intestine ; between mesentery and mesentery ; between intestine and mesentery. Cases are on record in which the sigmoid flexure was connected by a peritoneal cord with the cecum or with a loop of small intestine situated in the right hypochondriac region. These few examples may convey a slight idea of the numerous variations which may be met with. It would be useless to enumerate all the different connections that have actually been described.

(*b*) The omentum also may be so radically changed in form and outline by chronic peritonitis that cords are formed which lead to strangulation of the bowel. A necessary condition for the production of such an internal incarceration is fixation of the omentum to some one point of the peritoneum. In cases of this character the omental cords are, on an average, coarser, thicker, and longer than ordinary peritoneal cords, and at the same time more rounded. Omental cords are also occasionally multiple—more frequently so than simple peritonitic strands. Cases

[1] Lupton, *Lancet*, 1897, vol. i., p. 1204.

are also on record in which two distinct omental cords produced incarceration of the bowel in two places.

The form of the omental cords in chronic peritonitis may vary greatly. The whole omentum may be rolled together so as to form one solid cord which is usually attached to some one point of the abdominal parietes, the surface of the intestine, or one of the pelvic organs. In these instances the base of the omentum, where it is attached to the transverse colon, is spread out like a fan. In other cases lateral strands form and become detached from the body of the omentum so as to form independent structures. In other instances the omentum is broken up into a series of separate strands.

As has been pointed out, the free margin of the omentum may become fixed to various points of the abdominal parietes; this also holds good with the cord formed by the omentum in cases of chronic peritonitis. By far the most frequent point of attachment, however, is the peritoneum near a hernial ring, particularly the femoral ring. Experience shows that the omentum most frequently becomes adherent on the left side, apparently because the omentum is mainly situated in the left side of the abdomen.

(c) Chronic peritonitis can produce strangulation of the intestine in a third way, namely, as follows: Peritonitis may lead to the fixation of some structure that is ordinarily freely movable, and may in this way produce conditions favorable to the development of internal strangulation of the intestine. This applies principally to the vermiform appendix. It may very well happen that the free extremity of the appendix becomes anchored in some place by peritoneal adhesions; whenever this occurs, a cord is produced that is tightly anchored to two solid points of insertion. The most frequent point of attachment for the vermiform appendix is the mesentery of the lowest portion of the ileum, the ileum, or the cecum itself, or, finally, the peritoneum in the right iliac fossa. Precisely similar conditions are occasionally seen when a loose and freely movable Meckel's diverticulum becomes tightly anchored in some position by chronic peritonitis. In the case of this structure the possibility of its retaining its attachment to the inner surface of the umbilicus must be borne in mind, for this will form a band with two definite points of attachment. This peculiar abnormality will be dealt with in a special section.

A few instances are on record in which the Fallopian tube became adherent to neighboring portions of the peritoneum, and in this way led to strangulation of a loop of the small intestine. In very rare cases a loop of intestine has been known to become strangulated underneath a cord formed by some portion of the mesentery which had become fixed and anchored. Cases are also on record in which one of the longer appendices epiploicæ became fixed as the result of some localized peritonitis. Sometimes an appendix epiploica which has been pushed forward by the diverticulum of the colon may become attached in the same way. Abnormal attachments have been found between

epiploic appendices and the omentum, the intestine, the abdominal parietes, one of the neighboring appendices epiploicæ, etc.

[Perry [1] described 2 cases in which the small intestine was strangulated by a ring formed by adhesions between the tips of two adjacent appendices epiploicæ.—ED.]

In this way a bridge is formed under which the intestine may pass, and finally become strangulated. A case is also on record in which strangulation of the ileum was produced by the pedicle of a large ovarian cyst.

[J. Hutchinson, Jr.,[2] reported a band at least six inches long attached to the great omentum above and to the right broad ligament below, which was due to elongation of a hydatid of Morgagni which had become adherent to the great omentum.—ED.]

Strangulation of the bowels by the different kinds of bands and cords just described may occur in a variety of ways. The different possibilities may best be described under the heading of two principal types : in the first place, strangulation under a narrow arch formed by the band ; in the second place, incarceration by loops and knots that form in the band itself.

In order that the loop of intestine should become incarcerated *underneath* a false ligament or cord in the manner outlined above, two physical conditions are requisite—namely, in the first place, the band must be short and tense ; in the second place, it must be attached to an unyielding base. As a matter of fact, the bands of adhesive tissue in cases of this kind are rarely longer than 5 cm., and the arch is seldom very wide, so that only from one to three fingers can be inserted. The base of the arch is either formed by hard or by soft and yielding tissues, such as the wall of the pelvis, the right iliac fossa, the uterus, and occasionally by a firm mass of adhesions or the mesentery. If a loop of intestine once slips underneath a bridge of this kind, the same consequences develop as in strangulation of the bowel in an external hernial opening, the processes that subsequently develop being identical.

In order, on the other hand, to allow of strangulation of the bowel by a loop of adhesions which surrounds and continually constricts the intestine, the ligamentous band which forms this knot must be relaxed, loose, and at the same time of considerable length. As omental cords are, on an average, longer than other peritoneal cords, they are more often the cause of this form of strangulation than pure peritoneal pseudoligaments. The bands of omental ligaments are usually long, loose, and attached only at one extremity, the other being free. This free end may form a simple or a spiral loop. The band may surround a loop of intestine or, conversely, a loop of intestine may slip into the loop of pseudoligament. Leichtenstern has called particular attention to the fact that the loop of intestine which becomes strangulated by slipping underneath a pseudoligament of this kind usually presents anatomic peculiarities which existed before it entered the loop and

[1] E. C. Perry, *Trans. Path. Soc.*, vol. xl., p. 93.
[2] J. Hutchinson, Jr., *ibid.*, vol. xlvii., p. 100.

became strangulated. He says that "the two ends of such a loop are often close together (as the result of chronic mesenteric peritonitis), and thus differs in this respect from the loops in its immediate vicinity. It might almost be said that a loop whose ends are thus approximated is *pedunculated*, and, so to speak, protrudes from its mesentery. The condition seen is similar to that seen in the sigmoid flexure in volvulus of this part of the bowel." Strangulation of the bowel may also occur as the result of the formation of a true knot in the pseudoligament, and is almost as common as the simple strangulation of a portion of the bowel by a loop of a false peritoneal ligament just described. Leichtenstern has given a very comprehensive and clear description of those processes that lead to strangulation of the bowel by knotting of a peritoneal pseudoligament.

These two chief types of strangulation of the bowel by peritoneal cords do not occur with equal frequency, strangulation underneath a band of adhesions being much more frequent than incarceration by the formation of knots and loops in peritoneal pseudoligaments. The proportion, according to Treves, is as 6 to 1. According to Leichtenstern's more extended statistics, there were 62 cases in the first category and 27 in the second. Strangulation by loops and knots in the false peritoneal ligaments occurs only inside the abdominal cavity, whereas incarceration under an arch or a bridge formed by a false peritoneal ligament can occur both in the abdominal cavity and in the pelvis ; as a matter of fact, the latter form, as has been pointed out, is more frequent in the pelvis (see above). The reason why strangulation by loops and knots is more frequent in the abdominal cavity is that the strands of pseudoligamentous tissue, in order to form loops and knots, must be of considerable length, and this is possible only in the abdominal cavity. Strangulation of the intestine underneath a bridge formed by adhesions usually involves small pieces of intestine, whereas in strangulation by loops and knots of a false peritoneal ligament whole coils of the intestine are often affected.

Two other possible ways in which strangulation of the intestine by bands of adhesions may occur must be considered, which are quite different from the two described above. Though comparatively very rare, they may both lead to acute occlusion of the bowel, and, therefore, must be referred to here. One of these two forms is what may be called strangulation of the bowel over a band.

Treves describes this process as follows : " If several loops of a thin rubber tube filled with water are thrown over a tense cord, the lumen of the tube is completely obliterated at those points that are lying on the cord." It can well be imagined how an analogous process may occur in the living organism whenever a long loop of intestine that is filled with bowel contents comes to lie over a tense cord of adhesions. In a case of this kind the weight of the dependent portions of the loop is the primary factor in producing compression. It appears that this process has actually taken place in a few recorded cases. (Compare also Schnitzler's *Mesenteric Compression of the Intestine.*)

Finally, a few very rare cases of occlusion of the bowel by the action of ligaments and cords are on record in which the bowel became acutely kinked by traction. This may happen when the organ to which one end of the cord is attached undergoes a sudden change in position or in shape. Examples of kinking and of obstruction of the bowel brought about in this manner have been observed after tapping an ovarian cyst and after expulsion of the fetus from the uterus.

STRANGULATION BY MECKEL'S DIVERTICULUM OR BY THE VERMIFORM APPENDIX.

Meckel's diverticulum plays quite an important rôle in the pathology of herniaform internal incarceration of the bowel (compare in particular the communications of Leichtenstern, Bolt, E. Neumann). Meckel's diverticulum may produce herniaform incarceration of the bowel in two ways : it may either act like a false ligament attached in two places, or like a cord which is attached only at one end and is floating free in the abdominal cavity. The first possibility has been mentioned in the previous paragraph—*i. e.*, in the remarks on strangulation of the bowel by bridge- or arch-like cords or false peritoneal ligaments.

Meckel's diverticulum, in the great majority of cases, has one extremity free. It is, however, very often adherent either to the umbilicus or to the abdominal parietes below that point. When it is adherent in this way, it may constitute either a solid cord or, less frequently, a hollow tube with a patent lumen. In cases in which Meckel's diverticulum merely forms a cord from the ileum to the abdominal wall, strangulation of the intestine is probably never produced. If, on the other hand, the free end of the diverticulum is secondarily anchored by peritoneal adhesions, strangulation of the bowel may readily be produced. This secondary fixation of the free end of Meckel's diverticulum may occur in a number of different positions in the abdomen, the most frequent point of predilection being the mesentery, either above the point where the diverticulum starts from the ileum or between this point and the cecum. In other cases the free end of the diverticulum becomes attached to the omentum, to some other point of the small intestine, or to some point in the abdominal parietes. It may also become attached to the cecum, to some pelvic organ, or to the abdominal parietes in the immediate vicinity of the inguinal or femoral canals.

It is clear that in this way short or long, tense or loose, cords are formed. These cords formed by the diverticulum may produce hernial strangulation of the bowel in the same way as the false peritoneal ligaments described in the previous section ; in other words, they may either form bridge-like, tense cords under which loops of intestine become strangulated, or they may form loops and knots which surround a loop of intestine and so cause strangulation. Finally, they may produce obliteration of the intestinal lumen by exercising so much traction on a portion of the bowel that kinking occurs.

In addition, there is another very peculiar kind of occlusion of the bowel which may be produced by a free Meckel's diverticulum, and

in very rare instances by the free vermiform appendix. To allow of strangulation of the bowel by the free diverticulum or the free vermiform appendix the following conditions must be fulfiled : The diverticulum must be of considerable length and perfectly free—that is, it must be attached to the ileum only at its point of origin. In addition it must have a drumstick enlargement at its free end. This enlargement or swelling of the free end is usually produced by ampullary dilatation. If these conditions are fulfilled, the free end of the diverticulum may form a ring into which the loop of intestine becomes inserted ; more frequently still it may form a knot around one branch of a loop of intestine, the enlargement at the end of the diverticulum rendering the loosening of this knot impossible.

[A. E. Halstead,[1] in a review of 69 cases of intestinal obstruction due to Meckel's diverticulum, found that in 60 cases where the sex was recorded 44 were males and 16 females. In 48 cases the diverticulum was attached to the peritoneum and in 15 it was free, while in 6 cases its condition was not recorded. In the 48 cases it was attached to the mesentery in 23, to the umbilicus in 15, to mesocolon, mesorectum, omentum, and to the tissues around the appendix in 1 each, to the small intestine in 3, and in 3 the point of attachment was not determined. In 66 cases death occurred in 45 and recovery in 21, the mortality being 68.1 per cent. Of 54 cases operated upon, 32, or 59.1 per cent., died. Meckel's diverticulum is the cause of the obstruction in about 6 per cent. of the cases of intestinal obstruction, and probably occupies the next place to intussusception as the cause of intestinal obstruction.

Halstead suggests that inflammation, from fecal accumulation, of Meckel's diverticulum may set up ulceration and cicatrization of the adjacent intestine, and that all cases of stenosis at the point of attachment of the diverticulum are not necessarily congenital.—ED.]

M. Schmidt has compiled from the literature nine possible ways in which entero-occlusion may arise in connection with Meckel's diverticulum. But as these individual forms have no direct practical interest and can never be diagnosed during life, their detailed description here is unnecessary.

STRANGULATION OF THE BOWEL THROUGH SLITS AND APERTURES.

Cases belonging to this group are not so often met with as those of the second group, and are much less frequent than those of the first group. Many instances of this form of strangulation have been reported, but they must be considered as anatomic curiosities and as extremely rare. There are a number of different methods in which strangulation can be brought about, but it is seldom that two cases are exactly like. Slits and apertures in the mesentery and omentum are the causes most frequently found.

Slits in the omentum may be congenital, but they are more frequently due to abdominal traumatism of one kind or another.

Slits in the mesentery may also be either congenital or traumatic in

[1] A. E. Halstead, *Annals of Surgery*, 1902, pt. cxii., p. 471.

origin. Their most frequent position is in the mesentery of the lowest portion of the ileum, and usually very close to the intestine.

[Treves describes a special situation for a congenital abnormal aperture in the mesentery near the lower end of the ileum which contains no fat, glands, or blood-vessels; it is bounded by an anastomosis between the ileocolic branch of the superior mesenteric artery and the last of its intestinal branches. Atrophy of the serous layers in this situation may easily occur and lead to a "mesenteric hole."—ED.]

They vary in size and shape. Sometimes they form mere slits; in other cases they are larger and round. They may be very small, or they may be of considerable size. Slits are rare in the mesentery of the upper portions of the ileum, the jejunum, or the colon. A case is on record in which strangulation of the bowel occurred in a fissure situated in the mesentery of the vermiform appendix.

In several cases there have been slits in false peritoneal ligaments; in other instances fissures have been formed by the close approximation of the free margin of a false peritoneal ligament to a normal peritoneal fold, such as to the broad ligament of the uterus. In both of these instances loops of intestine slipped into these fissures and became strangulated. [Treves operated upon a case in which a loop of small intestine had passed through a slit in the peritoneal fold supporting the spleen (sustentaculum lienis).—ED.]

More frequently the intestine becomes incarcerated in slits, apertures, or rings formed by peritoneal adhesions between various abdominal organs. Different loops of intestine, for instance, may become adherent to one another, or a loop of intestine may become adherent to the uterus, to the abdominal wall, or to the wall of the pelvis—particularly to the abdominal parietes in the immediate vicinity of hernial openings. Or, again, the appendices epiploicæ may become adherent to one another in many different ways, and in this manner form fissures, holes, or rings, in which loops of bowel become incarcerated.

In addition a long series of curiosities of this type are on record; for instance, strangulation of a loop of intestine in a tear of the uterus and of the bladder, strangulation of a loop of intestine in a slit situated in the suspensory ligament of the liver and in the broad ligament of the uterus. In a few cases a loop of intestine was strangulated underneath the peritoneum after having become inserted into a slit of the parietal peritoneum that had been produced by trauma.

INCARCERATION OF INTERNAL HERNIAS.

Strangulation of internal hernias is the least frequent form of internal strangulation of the bowel, according to Leichtenstern's definition of internal hernia. Leichtenstern applies the term internal hernia to those which "are either situated altogether within the abdominal or thoracic cavity or are situated subperitoneally or retroperitoneally parallel to the abdominal wall and protruding into the abdominal cavity, without at any time, even if they continue to grow, passing in an outward direction."

From a practical point of view the most important cases of this kind are those cases of so-called " external " hernias which become strangulated in such a manner that no swelling can be felt externally. These cases are of paramount importance because they run the same course as an internal strangulation, and in the absence of a swelling the exact nature of occlusion of the bowel cannot be determined ; in other words, there is nothing to show that there is any external hernia. This sequence of events is occasionally seen in very small femoral hernias ; less frequently in inguinal hernia in very obese subjects ; and, finally, in hernias of the linea alba. Still more important are those cases in which a hernia develops in an accessory and interstitial fossa situated between the muscles and the fascia of the abdominal wall. A hernia may develop in this position and become strangulated either independently or when associated with an ordinary freely movable inguinal or femoral hernia. No doubt many medical men have had the following experience : In a patient with the symptoms of strangulation of the bowel the medical attendant in the course of his systematic routine rightly examines the external hernial apertures and finds an inguinal hernia ; he operates ; finds the hernia reducible, returns it, but the symptoms of strangulation still persist, and at a second operation or at the autopsy the presence of an interstitial pocket or hernial sac is discovered which strangulated the bowel.

Some other forms of hernia present the features of so-called " internal " incarceration of the bowel even more frequently than the inguinal and femoral hernias. These forms sometimes present all the features of a genuine external hernial swelling. As these lesions properly belong to surgery, and as careful descriptions of these different forms of hernia will be found in works on the operative treatment of hernia, they will merely be mentioned by name here : they are the obturator, sciatic, perineal, lumbar, rectal, vaginal, abdominal, and intercostal forms of hernia.

Genuine internal hernia, in the sense defined above, can never be diagnosed. An exception to this rule is the diaphragmatic form. When a genuine internal hernia becomes strangulated, all that can be done is to recognize the symptom-complex of strangulation of the bowel in general. It is impossible to diagnose strangulation of an internal hernia, and absolutely impossible to make out what form of internal hernia is present. In view of all these facts and the great rarity of strangulation of internal hernia, no detailed description of this lesion will be given. Jonnesco has given a very exhaustive description, both of the clinical details of this condition and the pathologic anatomy of strangulated internal hernias. Their account here will be limited to a brief enumeration of the different forms of incarceration of internal hernias so far described.

Broesicke has recently published a very careful and minute description of these different lesions ; the reader should refer to his monograph for details. The following facts are quoted from his work :

Hernia of the Foramen of Winslow.—This form of hernia was found

8 times : 6 times involving the small intestine and twice the large intestine. In 4 of these cases the bowel became strangulated. [According to Treves,[1] all the cases have been in adults between the ages of twenty-five and forty-four years and all but one males.—Ed.]

Hernia of the intersigmoid recess (herniæ intersigmoideæ) has been described twice. [Treves quotes 4 cases.—Ed.]

Hernia of paracecal peritoneal pockets—viz., the ileo-appendicular form of hernia (rare), the retrocecal form (more frequent) : Jonnesco found 11 cases of the latter in the literature, 1 of which died from incarceration. Broesicke has analyzed these 11 cases and has found that 4 of them do not properly belong to this group, at least not from an anatomic point of view.

[For a useful summary of the anatomy of the pericecal fossa the reader should consult R. J. A. Berry's[2] monograph.—Ed.]

Hernia of the Recessus Duodenojejunalis and Duodenalis (So-called Treitz's Hernia).—This is by far the most frequent form of internal hernia, for nearly 50 cases of this variety have so far been described. In several of these cases incarceration of the bowel occurred.

Hernia in the Recessus Parjejunalis (Hernia Parjejunalis s. Mesentericoparietalis).—Of this form, very few cases have been noted. A few of the cases were complicated by incarceration.

The only form of internal hernia which requires a short description here is :

Hernia Diaphragmatica.—This is the form of internal hernia most frequently met with, and is important from another point of view, for it is the only form of internal hernia which can, under exceptionally favorable conditions, be diagnosed. As a matter of fact, the diagnosis of diaphragmatic hernia has been made in a number of isolated cases. As I have never had an opportunity of studying this form of internal strangulation during the life of a patient, the following excellent description of this lesion is taken from Leichtenstern.

Grosser has recently contributed a careful anatomic description covering 433 cases up to 1899, including the earlier compilations of Lacher and Thoma. According to Struppler, the cases in 1901 numbered almost 500.

Diaphragmatic hernia may be congenital and the result of arrest of development during fetal life. More frequently it is acquired and may be the direct result of trauma of different kinds leading to rupture of the diaphragm. The traumatic factors may be penetrating wounds, contusions of the whole body or of the abdomen, and in very rare instances violent paroxysms of vomiting.

A distinction is drawn between true and false diaphragmatic hernias. In the former variety the abdominal viscera which project through the opening in the diaphragm into the thoracic cavity are covered by a hernial sac consisting either of peritoneum or of pleura or of both. In the latter variety there is a perfectly free communication between the

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[1] Treves, *Intestinal Obstruction,* new ed. of 1899.
[2] R. J. A. Berry, *The Cœcal Folds and Fossæ,* Edinburgh, 1897.

abdominal and the thoracic cavities through which the abdominal organs pass. This condition is really an ectopy of the abdominal organs. The so-called false form of diaphragmatic hernia is much more frequent than the true form. [In 254 cases of diaphragmatic hernia, only 24 were true (Boas).[1]—ED.]

Both forms of diaphragmatic hernia are chiefly found on the left side. This is probably due to the protective action of the liver on the right side. Certain portions of the diaphragm seem to be more easily penetrated than others, especially the esophageal foramen and the diaphragm in its immediate vicinity. In addition an area immediately behind the sternum often becomes perforated, and allows the abdominal viscera, usually the colon or the small intestine, to prolapse into the anterior mediastinum, the left, or quite frequently the right, pleural cavity. Again, hernial protrusion not uncommonly occurs between the lumbar and the costal parts of the diaphragm ; finally, diaphragmatic hernia may occur where the sympathetic nerve passes through the diaphragm. The latter form is comparatively rare, and has been observed only in a few cases. The stomach is the abdominal organ most frequently involved in diaphragmatic hernia. When this happens, the greater curvature constantly becomes turned upward, the lesser, downward. When the fundus of the stomach passes into the thorax while the pylorus remains in its normal position, axial rotation of the stomach with symptoms of incarceration may occur. The colon, especially the transverse colon, becomes herniated almost as frequently as the stomach ; the omentum and the small intestine also pass into the thorax, but not so often as the stomach and the colon. The small intestine and the omentum are both involved with approximately equal frequency ; the spleen is involved less often, and only occasionally the liver (portions of the left lobe) and the pancreas ; in very exceptional cases the kidneys may be involved.

[In a case reported by C. Ogle,[2] a false diaphragmatic hernia contained the cardiac end of the stomach and a pedunculated piece of the left lobe of the liver measuring 5 by 4 inches and containing so much extravasated blood that it looked like a hard spleen.—ED.]

In the great majority of cases two or more of these organs are involved at the same time. If only one is involved, it is usually the stomach, less frequently the transverse colon, and still less frequently the small intestine or the omentum.

Occasionally diaphragmatic hernias, both the true and the false form, are accidentally found in the course of an autopsy. In cases of this kind it may be said that they produce no striking and conspicuous symptoms during the life of the subject ; occasionally, however, they produce a clinical picture that is sufficiently characteristic to enable the diagnosis of internal diaphragmatic hernia to be made *intra vitam*, provided, of course, that the patient is very carefully examined and all the details of the case taken into consideration. The diagnosis must be

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[1] Boas, *Diseases of Intestines*, Amer. translation, p. 374.
[2] C. Ogle, *Trans. Path. Soc.*, vol. xlviii., p. 114.

based on the fact that certain abdominal organs containing air (the stomach, the colon) enter the pleural cavity and thus give rise to the physical signs of pneumothorax. In addition, the following signs are presented : The side of the thorax involved (usually the left side) is less motile, so that the respiratory movements are smaller than on the other side ; at the same time there is bulging of the walls of the thorax on the affected side. The heart becomes displaced, pectoral fremitus is slight, normal breath-sounds are absent, the percussion-note is deep, loud, and in some cases tympanitic, in others not. Auscultation and percussion with the plessimeter bring out metallic sounds. In general pneumothorax all these phenomena persist for a certain time and remain more or less constant ; in addition, the symptoms of an effusion of fluid with Hippocratic succussion sounds are often present, as well as the well-known change of sound that occurs when the position of the body is changed. In diaphragmatic hernia the following characteristic features are found : the signs obtained by percussion and auscultation are very inconstant and variable, as they depend entirely on the amount of air or of semisolid material contained in the abdominal organs present in the pleural cavity. It can readily be understood that when they are greatly distended with air or fluid contents, the percussion and auscultation sounds over the chest will be different from the signs presented when these viscera are comparatively empty, and that the sounds will be modified according to the proportion of gaseous, liquid, and solid material present at the time percussion or auscultation is performed. It need hardly be mentioned that in order to determine this peculiar inconstancy and variability of the percutory and auscultatory signs, the patients must be frequently examined. The physical examination, moreover, should be carried out with exceptional care in these cases, and should be repeated at frequent intervals for a prolonged period of time. Sometimes the diagnosis may be cleared by filling the displaced stomach with water or by pumping air into the dislocated colon, for which a number of cases reported in recent years serve as illustration (Hirsch, Widenmann, Struppler). If signs of incarceration of the stomach or of the intestine appear in doubtful cases, the diagnosis of diaphragmatic hernia may be considered fairly well established. The Röntgen rays have lately assumed great importance in the diagnosis. By their proper application we succeed in bringing into view displaced organs, particularly the one most frequently displaced—*i. e.*, the stomach, which we can see in the thoracic cavity. Struppler has called particular attention to gastrodiaphany in the diagnosis of the position of the stomach, even though it is not so valuable an aid as the *x*-rays.

Occasionally diaphragmatic hernias which originate acutely as the result of trauma become chronic. Diaphragmatic hernia of this kind has been known to become chronic and persist for a long time. The symptoms in such cases vary greatly : as a rule, there are considerable distress, pain in the thorax or abdomen, and attacks of dyspnea which often depend on the degree of distention of the stomach and on the amount of physical exertion undertaken. In other cases there is diffi-

culty in swallowing, or a great variety of other functional disorders of the digestive canal develop. When death finally occurs in these chronic cases as a result of the diaphragmatic hernia, it is usually directly brought about by strangulation of the stomach or the intestine in the aperture in the diaphragm, or by acute rotation of the intestine around its mesenteric axis, or occasionally by acute suffocation. The latter termination may be brought about when from any cause the displaced abdominal organs become excessively distended (overloading with gaseous or solid contents) and compress the heart or the lungs. The same processes also lead to a fatal issue in cases of traumatic diaphragmatic hernia that occur suddenly.

Whatever the cause and nature of strangulation, the ileum is almost always the part of the intestine involved. It is very seldom that the colon or the lower portion of the jejunum becomes strangulated, while the rectum or the duodenum is never involved in the process. Strangulation chiefly occurs in the lower part of the ileum. There are many reasons why the ileum is so frequently involved : in the first place, it is very freely movable ; in the second place, all the anatomic lesions which dispose to herniaform incarceration of the bowel favor its occurrence in this portion of the intestine. The peritoneal cords and bands described above most frequently originate from inflammatory processes around the pelvic organs, the appendix vermiformis, and the various hernial openings with which the ileum is in immediate contact. In addition, Meckel's diverticulum starts from the ileum, while abnormal openings and fissures are very frequently found in its mesentery ; all these factors and a number of other causes account for the enormous predominance of internal incarceration of this portion of the intestine.

There is considerable variation in the extent of the ileum which becomes incarcerated ; in some instances only very short segments are involved in the process ; in other cases more than a meter is affected. Generally speaking, the extent of intestine involved is short when strangulation is due to fissures or holes and longer when due to loops or knots formed by false peritoneal ligaments. Variations, however, are seen in both directions. The length of the strangulated intestine is not the only factor determining the gravity of the clinical symptoms.

As soon as a definite portion of the intestine becomes incarcerated in one way or the other—that is, either by becoming constricted underneath a cord of adhesions, by becoming surrounded by a loop of a false peritoneal ligament, or by becoming incarcerated in a fissure or in an intra-abdominal hernia—a series of anatomic changes immediately develop, both in the affected section of the bowel and in its mesentery. The changes found postmortem in these cases vary considerably. Three factors require special consideration as regards their influence on the anatomic lesions—namely, first, the degree of incarceration—that is, the amount of pressure that is exerted by the constricting ring or cord ; second, the length of the segment of intestine that is involved ; third,

the duration of the incarceration from its onset to the fatal issue. The least advanced anatomic changes are naturally found in those cases which run a very acute course and end fatally within from twelve to thirty-six hours from the onset of the symptoms. The most advanced anatomic changes, on the other hand, are naturally observed in those cases in which a long piece of intestine, together with its mesentery, becomes incarcerated, and in those in which the closure of the incarcerating ring or the constriction exerted by the incarcerating band is very strong.

In a certain proportion of the cases the anatomic changes resemble more or less those in strangulated external (femoral or inguinal) hernias. In another series of cases the appearances resemble those of volvulus.

That piece of the bowel in immediate contact with the constricting band or ring and consequently directly exposed to pressure from the incarcerating agency is more or less narrowed mechanically. As a result of this pressure necrosis of the intestinal wall may occur, especially when the pressure is long continued. The serous coat of the intestine usually resists this necrotic process longer than the other structures of its wall. Finally, gangrene and perforation may supervene. The strangulated segment of the bowel undergoes all the changes which naturally result from interference with the circulation of the blood through the mesentery. In moderate degrees of strangulation there is venous stasis with rhexis and diapedesis; in more severe degrees of strangulation the supply of arterial blood may be completely prevented, and, consequently, gangrene results. In order to avoid constant repetitions the reader is referred for the details of the anatomic picture presented in these cases to the description of strangulation given in the general part of this work—namely, in the sections on the pathologic anatomy of occlusion and of axial rotation of the bowel. I need hardly emphasize that peritonitis may develop in all these cases, either in the immediate vicinity of the strangulated portion of the bowel or in more distant portions of the abdominal cavity, and, in fact, may be quite general. Peritonitis, however, is by no means a constant occurrence in intestinal strangulation. In some instances peritonitis appears very early in the course of the disease; thus it has been found in patients who died within twenty-four hours of the onset of strangulation; in other cases again peritonitis is absent when the disease runs a course of one or even two weeks.

Another remarkable observation is worthy of mention in this connection—viz., that in a few isolated cases of acute strangulation of the bowel, running a rapidly fatal course, the postmortem appearance resembled that of cholera. This does not apply merely to the external aspect of the body, but also to that of some of the internal organs. In some cases of incarceration the same dryness of the serous membranes and of the muscular tissues, the same contraction of the urinary bladder, etc., so characteristic of cholera, were noticed. This peculiar appearance is especially seen in cases with profuse vomiting.

PATHOGENESIS AND ETIOLOGY.

The pathologic anatomy alone of strangulation of the bowel shows that the mechanism of the process is by no means the same in all cases and in all its different varieties. There can be no doubt that strangulation of the bowel may be brought about by a cord formed from peritoneal adhesions, by Meckel's diverticulum; that it may occur underneath an arch of adhesions or in a fissure; and that these different forms of strangulation may occur so suddenly and so energetically that the whole symptom-complex of complete obstruction is immediately produced. In other cases a portion of the intestine is so placed that it is in danger of being constricted and narrowed, but pronounced symptoms of strangulation do not necessarily develop at once. This delay is often seen when large pieces of the intestine become constricted, though symptoms of strangulation usually appear later. The development of the symptoms may be slow or rapid; at all events, no acute development occurs in these cases where the onset of symptoms is gradual and progressive; in other words, the structural condition of the intestine may be one of great danger and yet there may be no functional disturbance, and the time at which strangulation occurs is, therefore, not synchronous with the onset of the grave symptoms characterizing acute strangulation. The question arises, What are the factors which cause strangulation of the bowel in the last class of cases?

Surgeons have always been busily occupied with investigations into this question. They have been particularly interested in the causes which determine the strangulation of external hernia, and a great deal of information has been obtained on this point. All the anatomic and experimental evidence bearing on the mechanism of strangulation of external hernia applies with slight modifications to the same condition in internal hernias. It is unnecessary, however, to reproduce the results of all the work that has been done on this point in detail, and for this the reader should refer to the excellent descriptions given by von Hueter, Schmidt, and Bardeleben. The following condensed remarks on this subject will be sufficient here:

The older surgeons believed that one of the possible causes of strangulation of external hernia was spasmodic narrowing of the hernial aperture. This explanation, as well as a number of others that have been offered from time to time, are now regarded as insufficient and unsatisfactory by the majority of modern surgeons. This is also true as regards internal hernias; thus it is well known, for instance, that a slit in the mesentery or a peritoneal fibrous band never contracts. A detailed consideration of these theories is unnecessary.

Those cases in which a long peritoneal cord or a Meckel's diverticulum forms a loop or a knot around a loop of intestine are very easy to interpret and to explain. The same applies to those rare cases in which loops of intestine become constricted over a false peritoneal ligament. The main questions to be answered are the following: What prevents a loop of intestine from becoming released from the constric-

tion by its own peristaltic movements? What prevents it from slipping out of a slit or away from under an arch of adhesions or out of an intra-abdominal sac? If the peristaltic action of the bowel can force the intestine into such a narrow place, why should it not be able to carry it out again?

One of the *physiologic* causes for the permanent arrest of a loop of intestine in a narrow place, and one of the factors that occasionally renders a spontaneous and independent release impossible, is the entrance of intestinal contents into the strangulated loop. In some instances it was found that a loop of intestine which at first was only loosely fixed subsequently underwent axial rotation and then became tightly fixed. One of the most prolific factors, however, in preventing spontaneous cure of the strangulation, are the circulatory disturbances in the intestine and mesentery which come on very soon after the bowel and mesentery are strangulated. The direct consequences of circulatory disturbances in the intestine and the mesentery are serous transudation and extravasation of blood, which soon fills the lumen. A number of experiments have been performed by many different investigators to explain the *physical* factors which cause strangulation under these conditions. Among some of the theories that have been enunciated by different authors may be mentioned the "valve theory" of Roser, the "distention theory" of Kocher, the "hydrostatic pressure theory" of Lossen, the "dynamic theory" of Busch, and a number of other theories. (For the details the reader should refer to the text-books on surgery mentioned above.)

A few general points of view which have an important bearing on the etiology of strangulation of the bowel remain to be considered. Certain differences are observed in regard to the frequency with which strangulation of the bowel occurs in the two sexes, internal herniaform incarceration of the bowel being somewhat more frequent in men than in women. According to Leichtenstern's statistics, however, strangulation of the bowel by false peritoneal ligaments occurs with approximately the same frequency in both sexes. This "statistical equilibrium" is established in a very peculiar manner—viz., in women pelvic peritonitis is very frequent and predominates enormously as compared to its incidence in men. In male subjects, on the other hand, perityphlitis and peritonitis around the hernial openings are so much more frequent than in women that the difference is equalized, while, as already pointed out, strangulation of the bowel by false peritoneal ligaments occurs with approximately equal frequency in the two sexes. The absolute predominance of internal strangulation of the bowel in general in male subjects is due to the fact that peritoneal adhesions in connection with Meckel's diverticulum, the vermiform appendix, and the omentum occur much more often in men than in women, so that all the forms of hernia and strangulation dependent on lesions of these parts are more frequent in men. The reason why the omentum more commonly shows peritoneal adhesions in male than in female subjects is that, being comparatively

short, it is consequently more readily involved in peritonitis in the neighborhood of the vermiform appendix and the hernial rings (forms of peritonitis that are more frequent in men) than in pelvic peritonitis (which is more frequent in women).

Age also seems to exert a certain influence on the incidence of internal strangulation of the bowel. This, again, is due to similar variations in the frequency of certain forms of chronic peritonitis in the young and the old. The forms of peritonitis which are of paramount etiologic importance in strangulation of the bowel occur most commonly between the twentieth and the fortieth year. All the forms of internal herniaform strangulation of the intestine now under consideration also occur with great frequency during this period of life. Pelvic peritonitis, which is so intimately connected with the sexual life of women, occurs most often at this time of life. In male subjects, on the other hand, inflammation of the vermiform appendix and chronic peritonitis originating in connection with hernia are most frequent during the same period. It need hardly be insisted on that all the forms of strangulation described above may occasionally occur before and after the period of life mentioned—viz., before the twentieth and after the fortieth year. As a matter of fact, strangulation of the bowel due to Meckel's diverticulum is met with chiefly between the tenth and the thirtieth year.

The following conclusions as regards the etiology of strangulation of the bowel, clinically speaking, may be deduced from the general description of the pathologic anatomy of this lesion given above. In many cases and in many forms of internal herniaform strangulation of the bowel no etiologic factor whatever can be found. This applies to all congenital abnormalities, to the cases in which Meckel's diverticulum is abnormally long, to congenital slits and fissures, to cases of intra-abdominal hernia and of slow and insidious forms of local peritonitis. Clinically speaking, the following factors may be utilized in determining the primary etiology of the lesion—the discovery of chronic peritonitis (in particular of perityphlitis and of pelvic peritonitis), old external hernias, and abdominal traumatism.

CLINICAL FEATURES.

The clinical picture of a case of acute internal strangulation of the bowel in its main features is identical with that described in a previous section in the account of the symptoms of occlusion of the bowel. As a matter of fact, the description of the symptoms of occlusion of the bowel is really based on our knowledge of the symptoms of acute internal herniaform strangulation of the intestine. (For the general symptomatology of this disease, therefore, the reader should refer to the description given in the section on Occlusion of the Bowel.) Here, however, special attention will be called to a few symptoms that are peculiar and characteristic of internal strangulation of the bowel. I will preface my account of some of these peculiar symptoms by a short sketch of the clinical picture.

Clinical Picture.—The onset of the disease is usually sudden—that is, more or less characteristic symptoms appear while the patient is enjoying the best of health and is perfectly quiet. No external cause can, as a rule, be discovered, and no morbid symptoms precede the onset. This mode of onset is the rule, and applies to the great majority of cases. In exceptional cases, however, certain agencies and certain conditions can be discovered which, without doubt, can and must be regarded as the immediate causative factors of strangulation of the bowel. The most frequent of these causative factors is some sudden and violent bodily movement—a fall, a jump, or a blow on the abdomen—or excessive straining at stool, and particularly lifting some heavy weight. In a few cases the disease begins with an attack of diarrhea or after the administration of a purgative. Sometimes the first symptoms were noticed very soon after a large meal ; a few cases are on record in which the reappearance of an old external hernia ushered in the symptoms of strangulation. A number of other conditions of long standing, such as perityphlitis, pelvic peritonitis, and hernia, can hardly be regarded as direct causes of strangulation of the bowel : they merely favor the development of those pathologic conditions that render it possible.

As a rule, the patient develops sudden and usually extremely violent abdominal pain, followed very soon after, or at least within a few hours, by an attack of vomiting. The symptoms of collapse develop rapidly ; the face turns pale, and the expression becomes anxious. The pulse becomes rapid and small, the temperature normal or subnormal. The pain persists from the beginning and is continuous, though exaggerated from time to time by short paroxysms of colic. After a time, the vomit becomes bile-tinged. The patient is greatly distressed by attacks of hiccuping and severe nausea. The urine becomes scanty, and the passage of feces and of flatus is completely arrested. Examination of the abdomen occasionally shows a moderate degree of meteorism ; this may, however, be entirely absent in cases that run a very rapid course. In addition, there may be slight tenderness on pressure in certain circumscribed areas of the abdomen ; no other typical signs are discovered. The condition of collapse rapidly increases in severity, and after two to four days, occasionally within the first twenty-four hours, death occurs. The patients usually remain perfectly conscious until the end.

In rare cases the pain appears quite suddenly, but it is not so overwhelming in its intensity from the very beginning. In these cases, however, it also usually rapidly increases in severity, so that it reaches its height within a few hours, or less frequently at the expiration of from one to two days. Vomiting usually follows very soon after the onset of the pain ; the whole symptom-complex described above may in these cases appear less rapidly, but be fully developed in one or two days. In those cases in which death does not occur so rapidly, the initial collapse may not be so great. Pain, complete constipation, and vomiting, however, persist uninterruptedly. The latter usually soon asumes a feculent character. The patient becomes exhausted by the continuous

pain, is quite unable to take any food, and is tortured by persistent vomiting, so that death usually occurs within a few days; occasionally the fatal issue is postponed for two weeks. In some instances the clinical picture is still further complicated by the appearance of symptoms of general peritonitis—namely, diffuse pain all over the abdomen and general meteorism.

INDIVIDUAL SYMPTOMS.

The pain is, as a rule, the first clinical manifestation of the occurrence of strangulation of the bowel and is always present. The pain is not necessarily so severe as to disguise all the other symptoms, but it is probably never entirely absent. As a rule, the onset of the pain is sudden and occurs while the patient is in perfect health. When it appears, it is usually very severe from the beginning, and is occasionally colicky in character, so that the patients twist and turn in their agony. In exceptional cases the pain is not so severe at first, but rapidly increases in severity so that it reaches its height within a comparatively short time. If the general course of the disease is very acute, the pain may be most severe throughout the course of the disease, so that it is as agonizing later as it was at the onset of the first symptoms. In the majority of cases, however, that run a somewhat protracted course, the severity of the pain is somewhat mitigated in the later stages of the disease; if collapse, gangrene of the intestine, and stupor supervene, the pain may disappear completely before death. Only a very small number of cases are on record in which patients complained merely of insignificant pain at the commencement, and were free from pain later in the course of the disease.

In complete occlusion of the bowel by strangulation the pain is continuous; at the same time, it is true, it may remit occasionally, and then again increases in severity, but it never intermits completely.

The patients frequently point to the umbilical region as the seat of the pain; but sometimes it is felt elsewhere in the abdomen. On careful examination it will usually be found that the painful point in many instances actually corresponds to the seat of the strangulation; some authors believe that the seat of the strangulation can be localized by the pain, but Treves denies this very positively and states that the seat of the pain is in no way an index of the seat of the strangulation. From my personal experience I fully indorse Treves' objection to this statement. I have frequently seen patients who complained of the greatest pain in some portion of the abdomen far removed from the seat of the incarceration. It is exceedingly important to recognize this fact, for ignorance or neglect of this point may frequently be fraught with serious consequences, particularly when these cases are treated by surgical interference. I have seen a case in which serious results followed an operation based on erroneous premises as regards the correspondence of the seat of the pain and the seat of the incarceration. (Compare section on Localization of Stenosis and Occlusion of the Bowel.)

It is not an easy matter to explain the pain in strangulation of the bowel, although this may, at first sight, seem a very simple matter. It is possible that occasionally the pain originates directly from the strangulated portion of the bowel. In the majority of cases, however, we are forced to assume that the pain is largely radiating in character and reflex in origin.

This initial pain must be differentiated from another form of pain that is frequent in this disease, but is secondary. If the disease runs a very acute course, the latter form of pain does not always develop. When the disease, on the other hand, runs a slow and protracted course, this secondary pain always appears in addition to the initial pain already described. This secondary pain is always due to peritonitis developing later in the course of the disease. Sometimes the pain is circumscribed, and is then due to swelling of the tissues and to localized peritonitis in the area of the strangulated portion of the bowel. This pain is exaggerated by pressure exerted from without, whereas the initial pain, just as genuine colicky pain, is, if anything, relieved by external pressure. The situation of this secondary circumscribed pain, which is increased on pressure, is frequently valuable in determining the seat of the incarceration. As soon as the peritonitis becomes general, the whole abdomen becomes painful to pressure, and at the same time the spontaneous pain radiates throughout the abdomen.

Treves makes a very acute observation as regards another kind of pain. Occasionally the abdomen becomes generally tender on pressure, even though no peritonitis is present. This tenderness on pressure is only very slight and appears only after the initial pain has persisted for several days. Treves compares this pain to the tenderness on pressure felt after any muscular spasm,—for instance, after a cramp in the calf,—and attributes the abdominal tenderness on pressure to increased sensitiveness of the intestine resulting from the violent contractions of the intestinal musculature.

Vomiting is never absent in internal herniaform strangulation of the bowel. It is one of the main symptoms of the disease, and is second in importance only to the pain. Some isolated cases are on record in which vomiting was the first clinical symptom observed, and in which it appeared before the onset of the pain. In the overwhelming majority of cases, however, it always comes after the pain—either following the onset of the pain immediately or within a very few hours. Only in exceptional cases does vomiting begin later than twenty-four hours after the onset of the initial pain. After vomiting has once begun, it usually persists until the death of the patient. As a rule, it is accompanied by attacks of hiccup, nausea, and belching. Vomiting may persist up to the actual time of death, and may torture the patient during the agonal stage, or, like the pain, it may stop a few hours before death occurs.

At first the stomach-contents are vomited; later bile-tinged, thin, brownish, pea-soup-colored, yellow material, like the yolk of an egg, is vomited. The vomit usually has a disagreeable odor, and finally

becomes decidedly feculent in character. Fecal vomiting may be considered the rule in this form of occlusion of the bowel. Gay noticed it 26 times among 37 cases. In Treves' statistics of 50 cases it occurred 28 times; was absent in 17; and in 5 a careful description of the symptoms of the case was lacking. This author also states that among the cases characterized by the absence of feculent vomiting a certain number ran a subacute course, while others ran an exceedingly acute course, so that death occurred before the time at which fecal vomiting usually appears in this disease. The average time for the appearance of fecal vomiting is the fifth day; cases are on record, however, in which it occurred on the second day of the disease, and others in which it did not appear until the ninth.

The third leading symptom is due to the complete obstruction of the lumen of the bowel. This symptom is absolute constipation. As soon as strangulation of the bowel occurs, constipation appears at once. Neither feces nor flatus are expelled. There is no positive explanation for the peculiar fact that the bowel-contents present in the colon at the time when strangulation of the ileum, for instance, occurs, is not expelled spontaneously. It is very probable that in these cases the intestinal musculature is paralyzed by reflex inhibition. We know, at all events, that all peristaltic movements below the point of incarceration stop completely. Treves carefully analyzed 50 cases of internal herniaform strangulation of the bowel, and found only 6 in which some spontaneous evacuation of bowel motions occurred after the onset of strangulation. In all these cases, however, certain peculiarities and complications were noticed which could account for this spontaneous defecation. It need hardly be mentioned, of course, that by means of rectal enemata the evacuation of the fecal material present in the colon at the time when strangulation occurred can always be brought about. It is recognized now that in internal incarceration of the bowel the motions never contain blood, whereas in volvulus, and particularly in intussusception of the bowel, blood is nearly always present. Treves found only two cases described in which blood was passed in the stools, and seems to be somewhat skeptical as regards these two cases, for he submits them to severe criticism. For this reason the following case, for which I am indebted to my colleague, Gersuny, in whose private practice it occurred, may be of interest, as showing that blood may appear in the motions in cases of strangulation of the bowel. The case is interesting and remarkable in another respect, as it shows that local meteorism may, under certain conditions, be absent in strangulation of the bowel:

The patient was a woman of fifty years who had repeatedly suffered from attacks of pain in the cecal region that, however, had not been followed by any tangible results.

On January 22, 1896, she suddenly developed violent colicky pain in the night, chiefly localized in the ileocecal region. The onset of the pain was followed by vomiting; after this neither feces nor flatus were expelled. An enema of two and a half liters was given, but nothing was washed out of the bowel but a few particles of fecal material.

On January 25 her condition was as follows: The patient is a rather delicate, thin woman; the pulse 92, the temperature 99.2° F. (37.4° C.); the abdomen is somewhat distended; there are occasional paroxysmal attacks of pain. During these paroxysms three tense ridges situated parallel to one another appear in the middle of the abdomen. Palpation of the cecal region reveals the presence of a transverse cord about as thick as the little finger. This cord can be moved somewhat in an upward direction. In an area below this cord there is increased resistance on palpation. Two enemata of three liters each were given, and when returned, were distinctly blood-stained (light red), and contained a few particles of fecal matter.

On opening the abdomen, a large amount (at least a liter) of blood-stained serum poured out of the peritoneal cavity. In the right iliac fossa there were coils of the lowest part of the ileum, measuring 80 cm. in length, which were quite empty and contracted (about as thick as the little finger), and felt hard, as though the walls were thickened. The surface of the bowel was dark brownish red in color, and appeared injected. At the upper boundary of this portion of the intestine a deep constriction was seen; above this constricted area the ileum was slightly inflated. The constriction of the bowel was due to the presence of a short, ribbon-shaped cord about three-quarters of a centimeter broad, which passed from the mesenteriolum (that is, the mesentery of the vermiform appendix) to the peritoneum of the iliac fossa. Between this band, the mesocecum, and the lower portion of the wall of the cecum there was a small round opening through which about 80 cm. of the lowest portion of the ileum had slipped. In addition, a piece of the medial wall of the cecum, together with the insertion of the ileum in the cecum, had slipped into this orifice, so that the strangulated loop of ileum was only constricted above, and as it was patent below, its contents could readily pass into the cecum. The cord was severed, and the constriction relieved. The injection of the intestinal wall immediately passed away, and the patient was very much better for two days; she, however, developed symptoms of sepsis and died four days after the operation.

Very little can be added to the description of the general symptoms of strangulation of the bowel beyond what has been said in the sections on the General Symptoms of Acute Occlusion of the Bowel. It is, therefore, unnecessary to give a fresh account of the collapse and the general prostration, the temperature, the pulse, the excretory abnormalities of urine, etc., seen in internal strangulation of the bowel.

Sometimes an examination of the abdomen fails to reveal any very striking abnormality whatever, particularly when the course of the disease is extremely acute. When vomiting is very severe, there may be no abdominal distention at all. In other cases meteorism may supervene, but it is rarely marked, and never, at all events, so extreme as in volvulus of the sigmoid flexure or in acute general peritonitis. In cases of strangulation complicated with peritonitis, meteorism, of course, becomes extreme. The violent paroxysms of vomiting and the belching in uncomplicated cases prevent the development of advanced degrees of meteorism; percussion also fails to reveal any characteristic signs. There may be a circumscribed area of dulness which, in a few cases, is possibly produced by the strangulated loops of intestine. This sign, however, may be due to such a number of factors that its value in the diagnosis of strangulation of the bowel is very subordinate. The same applies to the significance of a tumor, for a swelling may be produced by strangulated loops of intestine either when the intestine is empty or when it is filled with blood, but such a swelling is by no means pathognomonic. In the few cases in which such a tumor has

been felt, it was about as large as an adult fist, with indistinct bound-
aries, and was tender on pressure. The diagnostic value of a tumor,
however, must necessarily be very slight, for it is found only in a few
exceptional cases of strangulation of the bowel, and then only *sub
finem vitæ*.

COURSE AND PROGNOSIS.

Nearly all forms of internal strangulation of the bowel, if allowed
to run their course without interference, prove fatal. In a certain
proportion of cases surgical intervention may save life. Only in a
very small proportion of cases is so-called internal treatment of
any benefit. A spontaneous cure of this disease is exceedingly rare.
Some cases run a fulminating course and lead to death in a very
short time ; other cases run a more moderate and protracted course.
The duration of the disease varies from a few hours to about two
weeks. LeMoyne, for instance, reports the cases of two young soldiers
who, while they were in perfect health, suddenly developed strangu-
lation of the bowel and died—the one after ten hours, the other after
eighteen hours. In the one case the ileum had become strangulated in
a fissure of the mesentery ; in the other, the ileum was strangulated
under a band formed from a Meckel's diverticulum. In general it
may be said that extremely severe internal symptoms usually indi-
cate that the case is going to run a rapid course, whereas those cases
in which the onset of the initial symptoms is gradual and the severity
of the symptoms slowly increases run a protracted course. In excep-
tional cases the reverse holds good—namely, cases with a violent onset
run a protracted course, and cases terminate rapidly in which the initial
symptoms come on slowly.

The severity of the symptoms and of the whole course of the disease
is essentially determined by two factors—principally by the character
of the strangulation itself and then by the amount of intestine and
mesentery involved. When a portion of the bowel becomes surrounded
by a nooze or knot formed by false peritoneal ligaments or by a Meckel's
diverticulum, both these factors are present and the strangulation is
very tight and the extent of intestine and mesentery involved is usually
considerable. For this reason the course of these forms of the dis-
ease is on an average more severe and shorter than of those forms in
which the bowel becomes strangulated underneath cords and bands
formed by false peritoneal ligaments or in fissures and hernial apertures.

Death may be brought about in different ways. In the cases that
run a fulminating, very rapid course, death is due to shock, to the
general exhaustion brought on by the pain and the profuse vomiting, and
less frequently to peritonitis. The latter complication, as we know, is
not specially frequent in the form of strangulation of the bowel now
under discussion. Finally, death may result from perforation of the
bowel, but this is very rare, since death usually occurs before gangrene
leading to perforation has had time to develop.

It is quite probable that a spontaneous cure of incarceration of the

bowel occasionally occurs, but it is, of course, very difficult to prove this. A few cases are on record in which the patient gave a history of having survived two previous attacks presenting the same symptom-complex of internal strangulation of the bowel as was seen in the last and fatal attack, which was shown postmortem to be due to internal strangulation of the bowel. It is possible that in cases of this kind the former attacks were really due to internal incarceration of the bowel. This, of course, is at best only an assumption that is more or less probable, but not irrefutable, as no direct proof of this assertion can be adduced. In order to explain the possibility of a spontaneous cure of internal herniaform strangulation of the bowel, it must be assumed that possibly the loop of intestine was not completely strangulated, or that, in becoming incarcerated, it performed axial rotation, and that, later, it rotated back into its normal position and that the incarceration was released in this way. In the section on the Mechanism of Incarceration this process has been described and attention was called to the fact that conditions of this kind have actually been observed. Another possible method of spontaneous cure of internal herniaform strangulation of the bowel is suggested by an observation occasionally made in operations and postmortem examinations on these cases. In a few instances the constricting band of adhesive tissue has been found to be very thin and gangrenous, so that it was on the point of giving way. It must be borne in mind that the constricting band itself is greatly compressed by the pressure of the strangulated intestine, and that, consequently, its nutrition must suffer. Of course, it is impossible to prove that in any one case a spontaneous release of an internal incarceration of the bowel really followed giving way of a band which had become gangrenous. The most plausible and at the same time the most probable view is that the bowel, provided the strangulation is not too tight, may become spontaneously released when the abnormal degree of intra-abdominal pressure which usually exists is reduced (by lavage of the stomach, etc.) or when peristaltic movements of the bowel are carried out that favor the release of the strangulated loop. But whatever the exact mechanism of liberation of a strangulated loop of intestine may be, we know from clinical experience that such spontaneous cures of internal herniaform strangulation of the bowel do actually occur in a certain proportion of cases where the treatment has not been of a distinctly surgical character.

DIAGNOSIS.

The diagnosis of internal strangulation of the bowel is one of the most difficult problems in ordinary medical practice. There is no single symptom which is confined to this condition or can even be considered pathognomonic when it occurs in combination with other symptoms. The diagnosis of strangulation of the bowel, as a matter of fact, can never be positively made. In a few instances it may be possible to make a diagnosis by exclusion, after having first determined that the bowel is occluded and that the occlusion occurred acutely. All the

other factors capable of bringing about acute occlusion of the bowel must be excluded, or it must, at least, be shown that they are probably not present in the individual case. When all these possibilities have been excluded, the existence of internal strangulation may be suspected. In the differential diagnosis the following are the conditions which must chiefly be considered : strangulation of an external hernia, obstruction of the intestine by foreign bodies, intussusception, general peritonitis, partial paresis of the bowel (so-called dynamic ileus), the sudden exacerbation of chronic stricture leading to complete occlusion of the bowel, and, above all, volvulus of the intestine. In the description of these different conditions the possibilities and means of diagnosis of these various lesions in any given case were considered, and the essential points of diagnostic importance dealt with. In order to avoid repetition here I will briefly summarize the points that may lead us to suspect the presence of internal strangulation. They are the following : Sudden onset of the disease, either while the patient is in perfect health or preceded by one of the predisposing conditions enumerated above. The onset of the disease is accompanied by violent pain, soon followed by vomiting, hiccup, nausea, complete constipation, and, comparatively early in the disease, symptoms of collapse. Unless death occurs at this stage, vomiting becomes feculent after a few days. At the same time examination of the abdomen in the great majority of cases fails to provide any absolute guide to the diagnosis. A striking feature of the disease is that, notwithstanding the existence of such severe abdominal symptoms, meteorism is usually quite moderate. Only in cases complicated by peritonitis is there more marked meteorism. In the presence of this complication the diagnosis becomes quite impossible.

Another point that may possibly be of value in the diagnosis is a history of past diseases which produce the anatomic conditions which most often favor the development of internal strangulation of the bowel —namely, perityphlitis, pelvic peritonitis, and old hernias. These diseases all lead to the formation of false peritoneal ligaments, which, in their turn, are the most prolific cause of strangulation of the bowel. Positive evidence of the occurrence of such diseases before the onset of the acute symptoms described above would not necessarily point directly to the diagnosis of strangulation of the bowel ; while, on the other hand, the absence of diseases of this kind would not in the least militate against the diagnosis of internal strangulation.

VOLVULUS; AXIAL ROTATION OF THE INTESTINE; KNOTTING OF THE INTESTINE (Volvulus Intestini).

THE various pathologic conditions to be described in this section belong to one group, as they have many points in common, both as regards their pathogenesis and clinical features. Formerly a number of other forms of occlusion of the intestine, particularly some forms of internal strangulation and even of intussusception, were included under this name, but more recently only those pathologic conditions which

will be considered in this section have been called volvulus. This classi-
fication is adopted, as it is simpler and more in accord with modern
medical opinion.

Two forms of intestinal occlusion are included under the name of
volvulus, namely : volvulus of the intestine proper and a condition
that may be described as knotting of the bowel ; in the latter form it
must be remembered that knotting occurs between two loops of intestine
and not between a loop of intestine and a band or cord formed by a
false ligament or adhesions. When a loop of intestine becomes strangu-
lated by a peritoneal band, the knotting occurring in the band and not
in the bowel, the condition belongs to a different category and is not
dealt with in this section. Strictly speaking, of course, the first form
—viz., axial rotation—is at the same time knotting of the bowel, and
the second form—viz., knotting—an axial rotation, but it is customary
to differentiate the two conditions as described above, and it is advisable
to adhere to this method of subdivision.

ANATOMY AND PATHOGENESIS.

1. Rokitansky was the first to give a clear description of the anat-
omy and pathogenesis of volvulus. He formulated a scheme of the
various possible conditions that can be developed when this accident
occurs, and as his scheme is a model of clearness and comprehensive-
ness, and as all that he said then applies with its original force now, his
description will be followed here. He distinguishes the following pos-
sible forms of volvulus :

(*a*) A piece of intestine may perform a complete or partial rotation
around its longitudinal axis ; when this occurs, the walls of the intes-
tine become so closely approximated that the lumen of the bowel is
obliterated.

(*b*) The whole or part of the mesentery performs either half a rota-
tion, a whole rotation, or several rotations, together with the piece of
intestine attached to it ; in cases of this kind rotation of the bowel
occurs around the mesenteric axis.

(*c*) A piece of intestine together with its mesentery forms an axis
around which another loop of intestine with its mesentery undergoes
rotation. [(*a*) and (*b*) constitute " simple," and (*c*) compound, volvulus
(Moynihan).[1]—Ed.]

Although volvulus may occur in many different parts of the intes-
tine, it is most frequent in the sigmoid flexure ; here rotation takes place
around the mesenteric axis ; as a matter of fact, volvulus occurs so fre-
quently in the sigmoid flexure as compared with other portions of the
intestine that this form really constitutes the only variety of volvulus
that is commonly met with. The pathogenesis of this form of volvulus
may be regarded as a prototype of all the other possible forms, and the
account of volvulus will, therefore, first deal with volvulus of the sig-
moid flexure around its mesenteric axis.

[1] Moynihan, *Medical Chronicle*, February, 1903.

The following anatomic conditions are necessary for the development of volvulus : the sigmoid flexure must be very large, and its mesocolon correspondingly long and very narrow ; when the mesentery of this portion of the bowel is narrow, the two ends of the sigmoid flexure are approximated to each other and the piece of mesocolon situated between these ends is converted into a pedicle around which rotation of the sigmoid, when all these conditions are fulfilled, can readily occur.

This peculiar formation of the sigmoid flexure and of its mesocolon may be congenital ; in the majority of cases, however, it appears to be acquired, and may be due to chronic inflammation of unknown origin. In the great majority of instances, however, habitual constipation is the direct cause of the narrowing of the mesocolon, as is shown by the fact that there is a history of obstinate chronic constipation in a large number of the cases of volvulus of the sigmoid flexure. As soon as large masses of fecal matter accumulate in the sigmoid flexure and remain there for some time, the excessive weight of this portion of the bowel produces local displacement and a disturbance in the normal relation of the parts ; the sigmoid is dragged out of place, usually outward ; when this occurs, the mesentery too is distorted and usually becomes elongated ; at the same time the two ends of the sigmoid flexure become approximated. The conditions thus created favor volvulus. It is possible that the irritation exerted on the mesentery by the traction of the distended and overloaded sigmoid flexure induces chronic peritonitis in this portion of the mesocolon, so that secondary contraction of the mesenteric pedicle occurs. When this happens, the two ends of the sigmoid flexure are, of course, drawn tightly together, so that the development of volvulus is rendered still easier.

This is the anatomic basis for the development of volvulus of the sigmoid flexure. The rotation itself is usually the result of some extraneous cause, such as bodily exertion, jumping, etc. Another possible way in which volvulus may occur in cases in which the anatomic conditions just described are present is the following : The sigmoid flexure may become overloaded and distended with fecal matter, so that one branch of the flexure, being heavy and distended beyond its normal size, drops down over the other ; this accident may be due to purely mechanical causes, or may be brought about by the peristaltic action of the sigmoid flexure. The two branches become rotated around their mesenteric axis in such a way that the colonic end of the flexure and the rectal end come in contact with each other ; the former usually lies in front of the latter ; in exceptional cases the reverse occurs—viz., the colon is in front and the rectum behind. The two branches are usually rotated through 180 degrees ; less frequently, through 360 degrees ; as the axes of the two branches run parallel to the axis of the mesentery (at least in their first part), some rotation of these branches of the sigmoid around their own axis also occurs. Occasionally the bowel performs two or three rotations around its mesenteric axis. Von Samson believes that rotation of the bowel to 180 degrees, as a rule, does not produce symptoms of stenosis or strangulation in the sigmoid flexure,

but that the bowel must perform rotation of at least 270 to 360 degrees before this can happen. He believes, however, that rotation through 180 degrees can produce volvulus of the sigmoid in cases where there is scar tissue in the mesocolon belonging to the rotated portions of the bowel.

Leichtenstern has published an interesting case, showing that volvulus of the sigmoid flexure may actually exist without producing any of the functional disorders commonly found in this condition. The case was one of pseudoleukemia occurring in a boy of eleven years, who had never suffered from constipation ; at the autopsy chronic volvulus of the sigmoid flexure was found ; the bowel was rotated in such a manner around its axis that the two ends of the flexure were in intimate contact. Leichtenstern inflated the colonic limb of the rotated flexure with air, and was thus able to cause rotation of the affected loop, so that it resumed its normal position ; as soon as the air was allowed to escape so that the bowel collapsed, the sigmoid flexure immediately assumed its abnormal rotated condition. The author concluded from this experiment that during the life of the patient masses of fecal matter entering the rotated loop from above probably caused dilatation of the colonic end of the flexure in the same manner as the inflated air, and that in this way the flexure was rotated into its normal position, so that the lumen becomes patent and the passage of the fecal material possible.

Borgen records another case of the same kind, but more severe in degree ; in the course of nine years there were numerous typical attacks of acute intestinal occlusion ; in the last, there was sudden collapse. On section there was found volvulus of the sigmoid flexure, with abnormal movability and abnormal length of the sigmoid and its mesentery.

Observations of this character which are essentially analogous to physiologic intussusception of the bowel, seem to prove that it is quite possible for a piece of bowel in a state of volvulus to return to its normal position. In other words, it must be assumed that some additional factors must be present in cases of irreducible volvulus to prevent this spontaneous physiologic cure. It is only in the presence of these additional factors that permanent occlusion of the lumen of the bowel is met with, and we must assume that this condition is chiefly brought about by the pressure exerted on the bowel by loops of intestine rotated in such a manner as to lie in close proximity to one another. The most important factor causing this permanent rotation is undoubtedly the weight of the loops of intestine which are involved in the process ; as soon as they become distended and filled with fecal material, they become very heavy and bulky, so that they drop down in the abdomen and cannot be raised by the peristaltic muscular efforts of the bowel-wall. When this occurs, a series of sequelæ develop which will be described in detail below and consist essentially in stasis of blood—*i. e.*, congestion of the intestinal walls followed by transudation of serous fluid and the accumulation of gas in the affected area of the bowel ; these anatomic conditions effectually prevent a restitution to normal because they

render the rotation of the bowel into its normal position very difficult or impossible. Another factor that may prevent the rotation back into a normal position is the accumulation of large quantities of gas in the colon; such an accumulation of gas reduces the intra-abdominal space and in this way mechanically prevents the release of the rotation; in other words, the sigmoid flexure may become fixed in its abnormal position merely from the fact that there is not enough room for it to rotate back in. Melchiori has performed a number of experiments that seem to show that the resistance offered by the abdominal walls may act in the same way—viz., mechanically. A dynamic factor must finally be considered—namely, the distention and atonic weakness of the muscular walls of the sigmoid flexure that are the natural result of the prolonged constipation present in the majority of cases of volvulus. Sometimes the return of the bowel is prevented by adhesions and by false ligaments. While a few cases are on record in which a number of loops of the ileum pressed on the rotated portion of the bowel in such a manner as effectually to prevent its rotation back into a normal position, the same effect has also been produced by compression of the stem of the volvulus by that portion of the mesentery that was attached to such a collection of loops of ileum in the vicinity of the rotated portion of the intestine.

Mesocolic axial rotation of the sigmoid flexure is more frequent in men than in women, and seems, moreover, to be a disease of advanced years, for it is much more common in old than in young subjects, and is only rarely seen under forty years of age. This can readily be understood in the light of its etiology, inasmuch as the chief predisposing and causative factor of volvulus of the sigmoid flexure is chronic constipation, which is essentially typical of old subjects and is comparatively rare, in its fully developed form, in younger persons.

Portions of the colon other than the sigmoid flexure are rarely involved in volvulus or rotate around their mesenteric axis. Certain anatomic peculiarities and anomalies are an essential condition for the development of the lesion, notably certain congenital anomalies of position and deformities of the bowel; the nature of these abnormalities is approximately the same in all the cases—viz., the affected loops of colon have an abnormally long mesocolon. In all the cases of volvulus occurring in portions of the bowel other than the sigmoid flexure the secondary processes that lead to the development of the typical picture are practically the same as those described in a preceding paragraph under the heading of volvulus of the sigmoid flexure.

Zoege von Manteuffel has recently made an exhaustive study of cecal volvulus. He collated the available cases (20) and added a few new ones. He distinguishes two forms—one characterized by twisting about the axis of the intestine, and the other by twisting about the mesenteric axis, with strangulation. The general pathogenetic conditions are the same as in other cases of volvulus.

Rotation of the small intestine around its mesenteric axis occurs more frequently than rotation of the colon, but far less often than vol-

vulus of the sigmoid flexure. This statement must be modified to include only those forms of axial rotation of the small intestine that are of spontaneous origin; in the course of strangulation of the small intestine by bands and cords formed of adhesions volvulus is by no means infrequent; attention has already been called to this fact in the paragraphs on these lesions of the small intestine. In the cases of spontaneous rotation of the small intestine, either a small portion of the small intestine alone may be involved or the volvulus may comprise large numbers of loops—whole convolutions of bowel in fact; sometimes the whole jejuno-ileum may be involved in the process. Leichtenstern, who has made a special study of the latter form, is of the opinion that volvulus of the whole small intestine—*i. e.*, from the first portion of the jejunum to the end of the ileum—is found in those subjects in whom the ileum, the cecum, and the ascending colon have a common mesentery; he argues that such a congenital anomaly is a necessary predisposing factor in the production of volvulus involving such large portions of the small intestine.

Generally speaking, the conditions that favor the development of axial rotation of the small intestine are the same as those that determine volvulus of the large intestine—*i. e.*, of the sigmoid flexure. In both instances the part of mesentery that causes the rotation must be abnormally elongated between its origin and its intestinal attachment, and must also be narrower than normal at its root and so form a pedicle. In both instances the abnormal condition of the mesentery is either congenital or acquired later in life as the result of peritonitis involving the mesentery. This peculiar form of mesenteritis chronica is specially frequent in the mesentery of loops of small intestine which have been strangulated in an external hernia or have at least been in an external hernial sac, or, finally, in the immediate vicinity of an external hernia.

Still other relations occur: In a case of Schnitzler's, described by L. Wechsberg, in which the radix mesenterii, and with it the jejunum and ileum, was twisted some 360 degrees, the entire intestine was really very long, but its base was not appreciably narrowed.

[Gall-stones may give rise to volvulus of the small intestine; this may be brought about either by the violence of the colic or by contortions induced by the passage of a large concretion through the small intestine. Mayo Robson[1] has reported 2 cases. From a case reported by Briddon,[2] Treves[3] admits the possibility that a lipoma on the mesentery might have the same effect. Volvulus of the small intestine may depend on a congenital factor, such as an absence of the mesentery for the lower few inches of ileum. In a case reported by the editor,[4] the lower five inches of the ileum had no mesentery and was bound down to the posterior wall of the abdomen by the parietal peritoneum; there was no trace of any old inflammatory change in this

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[1] Mayo Robson, *Trans. Royal Medico-Chir. Soc.*, 1895, vol. lxxviii., p. 117.
[2] Briddon, *Ann. of Univ. Med. Sci.*, 1894, vol. iii., quoted by Treves.
[3] Treves, *Intestinal Obstruction*, ed. 1899.
[4] Rolleston, *Trans. Path. Soc.*, vol. xli., p. 129.

region. Where the abnormally fixed ileum joined the mesentery-possessing ileum, volvulus occurred and proved fatal. The ileum alone is involved in approximately 60 per cent. of the cases of volvulus of the small intestine (Moynihan).—ED.]

Permanent occlusion of the bowel in mesenteric axial rotation of the small intestine may be brought about in a number of different ways : The portion of intestine that is closed at either end by the axial rotation of the affected loop may be very heavy—that is, overloaded—from the outset or fecal material may accumulate in it subsequently, and in this way produce overweighting of the loop ; at the same time there may be excessive flatulent distention to start with, or this, in its turn, may subsequently accumulate, causing distention of the bowel-wall ; these two factors usually produce paresis of the muscular wall of the piece of intestine involved in the process, so that rotation backward into a normal position soon becomes impossible. In other instances the pedicle of the rotated loop of intestine—that is, the portion where the two ends of the twisted loop are in contact— is compressed by other loops of intestine superimposed upon it, or by some false ligament that forms in its immediate vicinity.

The forms of volvulus described so far are rotations of the bowel around its mesenteric axis ; in all these cases only the first and last portions of the affected loop—*i. e.,* the two ends which are in close proximity to the mesenteric pedicle—are in a state of axial rotation ; the conditions in this respect are the same as in volvulus of the sigmoid flexure. In very rare instances, however, the intestine may be rotated only around its vertical axis ; this occurs only in the cecum and the colon.

In all cases of this character without exception congenital anomalies, certain errors of development which date back to fetal life, are present ; these anomalies are specially seen in the colon and the cecum, and consist in malpositions of these parts of the bowel and in abnormalities in the length of the mesentery attached to them ; in all cases these anomalies cause abnormal motility of the colon and the cecum. Leichtenstern calls attention to the fact that in the majority of the cases that have been included under the heading of rotation of the bowel around its own vertical axis the lesion consisted in lateral kinking of the bowel following some form of displacement (the latter, it can readily be understood, is the result of the congenital anatomic anomalies described). In very few cases was Leichtenstern able to determine the existence of true rotation of the intestine around its own axis. Kinking of the bowel in all these cases occurred either in a direction diagonal to the vertical axis of the bowel or around the transverse axis of the intestine. Kinking alone does not necessarily determine occlusion of the bowel ; in many instances there are only symptoms of enterostenosis for long periods of time ; these symptoms may be succeeded either gradually or suddenly by the symptoms of intestinal occlusion, as soon, namely, as compression of the bowel, usually from without, occurs ; a mass composed of loops of small intestine may, for instance, become

superimposed upon the kinked portion of the gut, and may cause complete occlusion of the bowel either by direct pressure or by the traction or pressure exerted by the mesentery attached to these convolutions.

2. Volvulus Due to Two or More Loops of Intestine being Twisted or Knotted Together.—When volvulus of two or more loops of intestine occurs, the affected portions of the bowel often perform the most complicated and astonishing movements; the portions of intestine most frequently involved in the process are the sigmoid flexure and the ileum; less frequently, two loops of the small intestine, and very rarely a loop or loops of the small intestine and the ascending cecum and colon. To enable any one of these different forms of volvulus to develop, the same predisposing anatomic factors as those concerned in volvulus of the sigmoid flexure must be present. In other words, there must be a relatively narrow mesenteric pedicle and at the same time the mesentery must be abnormally long, either congenitally or acquired as the result of changes occurring later in life. The processes which dispose to the development of this form of volvulus of the intestine vary greatly; the different loops of intestine may become knotted and crossed in many different ways. Kuettner and Leichtenstern have given a very clear description of these processes and their mode of development. This subject is chiefly of interest to the anatomist, and is only of subordinate importance and interest to the clinician; for this reason a description of the possible varieties of volvulus can be omitted, without really detracting from the completeness of the account of this disease; in fact, I believe that the subject will appear simpler and clearer if no detailed description of all the possible varieties of volvulus described by pathologic anatomists is attempted. In almost every individual case some differences will be found; hence an exhaustive dissertation on this part of the subject would be superfluous. It may be pointed out that in the great majority of cases of volvulus a certain amount of axial rotation of the bowel will also be found; one or the other loop of intestine will be found rotated either around its mesenteric axis or, at either extremity, around the axis of the loop of intestine itself. Kuettner goes so far as to say that volvulus of the ileum and of the sigmoid flexure is always preceded by axial rotation of these portions of the intestine.

[According to Treves, volvulus due to twisting of two suitable coils of the small intestine is very rare. In a case recorded by G. R. Turner,[1] a boy fell twelve feet and became collapsed, vomited, and had severe abdominal pain. The abdomen was opened, and volvulus due to one coil of the small intestine being twisted around the other was found and successfully released.—ED.]

The following is a summary of the causal factors which determine the development of axial rotation and of volvulus of the bowel:

1. The disease, as a rule, occurs in the later years of life in subjects with thin abdominal walls and a small amount of fat in the mesentery

[1] G. R. Turner, *Trans. Med. Soc.*, 1893, vol. xvi., p. 16.

and omentum, the absence of fat in the abdominal walls, omentum, and mesentery producing a relatively large intra-abdominal space. It is true that volvulus of the bowel and axial rotation occasionally occur in young subjects with well-developed fatty layers in the abdominal wall, the omentum, and the mesentery, but leanness must, nevertheless, be considered one of the most important predisposing factors for the development of this lesion. [Cripps and Pitt have described cases of volvulus of the ileum in newly born children.—ED.] Kuettner is very emphatic in his statements to this effect; of course, he considers the anatomic anomalies as described above to be the really essential factors in this condition.

2. Habitual constipation: this condition is specially important in volvulus of the sigmoid flexure, the production of which has been described in a preceding paragraph. Several cases are on record in which the development of acute volvulus of the sigmoid flexure was preceded by an attack of acute diarrhea. Such attacks of diarrhea, however, as well as certain other factors, such as trauma of the abdomen, jumping, lifting heavy loads, etc., must be considered as purely accidental causes which play an active part only in isolated cases of the disease. Israel has reported a case of axial rotation of the bowel in which rotation of the sigmoid flexure occurred immediately after a common enema had been administered to a patient suffering from constipation. Israel believes that the injection of water into the rectum caused an increase of pressure in the lower portion of the bowel, and that this sudden increase produced axial rotation of the sigmoid flexure; in order that this sequence of events could occur, the two ends of the flexure must have been in close proximity, probably as the result of a contracting mesenteritis.

3. Abnormal length of the intestine or of parts of the mesentery favors the production of axial rotation and of volvulus; in fact, axial rotation and volvulus of the bowel cannot possibly occur unless such an anomaly exists. The normal relation between the length of the mesenteric attachment to the back of abdomen and the length of the intestine may be changed in a variety of ways. In the case of the small intestine the extent of the root of the mesentery is determined by the amount of space intervening between the duodenojejunal flexure and the end of the ileum. This amount of space is constant and always remains the same; it follows, therefore, that when this space remains normal *per se*, excessive length of the small intestine may produce relative shortening of the mesentery. This peculiar fact may also be utilized to explain the relative frequency of axial rotation of the bowel in the people of Russia, for W. Gruber and Kuettner have found that Russians have longer intestines than German, English, or French subjects. The former author determined by actual measurement that the average length of the intestine in a subject of Russian nationality is 56 feet; this is probably due to the fact that the poorer classes in Russia are forced, by economic conditions, to live almost exclusively on a vegetarian diet; certain religious rules and customs, moreover, prescribe many days of fasting in Russia.

Aside from this national form of disproportion, individual cases occasionally occur in which this condition is congenital. Such anomalies may be seen both in the small intestine and in the sigmoid flexure. The mesocolon, for instance, of the sigmoid may measure from 10 to 15 cm. at the apex, and be very narrow at its base; again, in other cases, the mesocolon may be completely absent, so that the two limbs of the sigmoid flexure are in direct contact and lie close together, bowel-wall touching bowel-wall.

Other important predisposing factors in the development of rotation of the bowel and of volvulus are certain forms of contracture and shrinking of the mesentery, which may be the result of chronic mesenteric peritonitis; this disease usually produces contraction at the root of the mesentery, and in this way creates the conditions described at some length in a preceding paragraph.

The **anatomic changes** that follow the occurrence of axial rotation of the bowel and of volvulus will now be dealt with. Here, as in the preceding paragraphs, the simplest form of the disease—viz., volvulus of the sigmoid flexure—will be described first.

One of the most characteristic conditions in this lesion is local meteorism; the loop of intestine forming the S of the flexure is often enormously distended and greatly changed, while, at the same time, all the other loops of the intestine appear perfectly normal. The sigmoid loop may be excessively inflated, so that it is pushed forward and lies in front of the rest of the intestines, covering them completely; when the abdomen is opened, this inflated loop of intestine alone may consequently present. The distended portion of the bowel may extend as far upward as the right hypochondriac region, touch the stomach and liver, and may force the diaphragm upward. The walls of such a distended and displaced loop are, as a rule, thickened, sometimes to a considerable degree, usually rigid, and of a dark, blood-red color. The serous covering of the bowel is frequently torn and fissured, the fissures occasionally extending down into the muscular layer of the bowel-wall; the mucosa in these cases, however, generally remains intact, as it is more distensile than the other layers constituting the wall of the intestine (Esau). Hemorrhages may be seen in all the layers of the intestinal wall. The lumen of the loop of intestine involved in the process may be filled with gas, and may, in addition, contain blood (in some cases as much as 2 kg.) or other mucous and fluid material of a fecal character; particles of solid fecal matter are also often found in addition to a considerable quantity of fluid feces. The portion of the mesentery attached to the twisted loop, which is usually also rotated out of place, is in general quite hyperemic and infiltrated with blood.

The twisted portions of the bowel themselves are, in contradistinction to the above, very thin, attenuated, solid, and quite pale. If the disease persists for a long time, gangrenous destruction of tissue may occur at the line of demarcation; or, again, peritonitis may originate from this spot.

The flexure itself is enormously distended, whereas the other por-

tions of the intestine—viz., the large and the small intestine in their entirety—are empty, collapsed, and pale, and are, moreover, always situated behind the swollen and distended flexure. This contrast is particularly apparent in the rotated limbs of the flexure itself, for here the transition from flat and empty loops of intestine to distended loops is very abrupt. In some cases the colon is also slightly distended; this, as a rule, is the direct result of meteorism from congestion and stasis. In rare instances the small intestine may be slightly distended and meteoristic; this, however, is generally not the result of intestinal occlusion, but occurs in cases in which there is sufficient time after the development of the rotation of the bowel for the development of secondary peritonitis.

Peritonitis in its different forms and stages is of relatively frequent occurrence in axial rotation of the bowel; sometimes it is strictly local; at other times, it is more general; in some cases the peritonitic exudate is very scanty and fibrinous; in others, it is extremely abundant and serous. A common occurrence in these cases, even when peritonitis proper does not develop, is the exudation of a hemorrhagic fluid into the peritoneal cavity; this is derived from the blood-vessels of the rotated loop, which are in a state of venous hyperemia and consequently favor the exudation of a sanguineous fluid from the vessel-walls. Perforation of the bowel is a rare accident as a rule.

Mutatis mutandis the same anatomic lesions and the same contrast between the rotated and the normal portions of the bowel are seen when other parts of the intestine undergo axial rotation, as in the volvulus of the sigmoid flexure which has just been described. In cases in which kinking and knotting of the bowel occur, very complicated conditions may result; in a previous paragraph we described an interesting case of this kind reported by Kuettner, and have shown that the most marked contrast between the different portions of the intestine and the most complicated forms of knotting may be seen in many cases of this disease. (For the details the reader should refer to the description of occlusion of the bowel in general in a preceding section.)

CLINICAL FEATURES.

The symptoms and the course of the disease vary somewhat according to the different forms of volvulus. As mesenteric axial rotation of the sigmoid flexure is by far the most frequent form of intestinal rotation, and as the clinical picture presented by this lesion is the most uniform of all, I will begin my description of the clinical features of volvulus of the intestine by a description of the symptomatology of this lesion.

Sketch of the Clinical Picture.—The onset of the disease is almost always sudden. It usually occurs in individuals past middle life or of advanced years; sometimes the onset of the disease is not sudden, but more gradual, so that the symptoms of the lesion appear slowly. In many cases there is a history of constipation; in other

cases again the onset of the symptoms is preceded by an attack of acute diarrhea or the patient had taken violent physical exercise immediately before he was seized. In the great majority of cases, however, the first sign of the disease—the first warning symptom—appears quite unexpectedly while the patient is in perfect health.

The patient is suddenly seized by a violent pain in the abdomen, followed in many cases by violent and profuse vomiting; in fulminating cases and in those that run a more protracted course the latter symptoms may be absent. In some instances the patients suffer from tenesmus. In exceptional cases an evacuation of the bowel contents occurs after the onset of the abdominal pain, either with or without tenesmus; in isolated cases these dejecta may contain a little blood. In the great majority of cases, however, complete constipation supervenes from the very beginning, and neither feces nor gas escape from the anus after the onset of the disease. Meteorism usually develops very rapidly after this; in torsion or rotation of the sigmoid flexure this meteorism possesses certain characteristic features which are occasionally of use in arriving at a diagnosis; it is, as a rule, strictly localized at first, so that the affected loop forms a tense, elastic swelling which can often be felt without difficulty. Later in the course of the disease meteorism becomes more general and soon loses its local character. The abdomen is only rarely tender on pressure, and then only to a slight degree; pain on pressure never becomes very severe unless local or diffuse peritonitis develops in the course of the disease.

Sooner or later, however, collapse comes on; usually it develops very rapidly and within a few hours of the onset of the disease; there is nothing specially distinctive about the symptoms of collapse in rotation of the bowel; unless operative or other external aid is given without delay, the patients succumb in a very short time in all cases where the symptoms of collapse appear early and develop rapidly. If the symptoms of collapse appear less rapidly and not so soon after the onset of the disease, the phenomena described above continue, while at the same time meteorism reaches enormous degrees; it is a remarkable fact that fecal vomiting is a very rare occurrence in these cases.

INDIVIDUAL SYMPTOMS.

Pain is never absent, and is always the first sign of volvulus. As a rule, it is quite severe, but, as Treves remarks correctly, it does not in general become so severe and so excessive in intensity as the pain of acute internal strangulation of the bowel. Treves is inclined to attach considerable importance to this point. Such a distinction is, of course, purely relative. The pain in this disease is, as a rule, continuous; sometimes it remits temporarily and assumes a more colicky character; complete intermission of the pain probably never occurs. The constant character of the pain is most likely due to the axial rotation of the bowel *per se,* and the exacerbations of the pain to the increased contractile efforts of the intestinal wall above the obstruction. The

pain is occasionally localized in the left lower portion of the abdomen, but may be felt in many different parts of the abdomen; sometimes it seems to be strictly localized in the umbilical region or in some other portion of the belly. In addition to spontaneous pain, the patients frequently complain of pain on pressure in a portion of the abdomen that corresponds to the seat of the volvulus; this pain on pressure is usually moderate at first, but increases in severity as peritonitis begins to develop around the lesion; when peritonitis becomes general, the pain on pressure also becomes general.

Tenesmus is a frequent symptom, although it does not occur as frequently as in acute intussusception; nevertheless, it may be considered an important symptom in the diagnosis of volvulus of the sigmoid flexure, particularly in making the differential diagnosis between this condition and acute internal strangulation of the bowel; any points of difference between these two diseases are important, as they so closely resemble each other in most respects.

Vomiting is also a very common symptom, although it is by no means constantly seen in volvulus of the sigmoid flexure. In some of the reported cases vomiting was completely absent; in other cases it occurred only at rare intervals during the course of the disease, and the amount brought up was very scanty; as a rule, however, vomiting, when it does occur, is profuse. It may also be stated that in the great majority of cases it is an early symptom, and is continuous throughout the course of the disease. In nearly all the cases it is accompanied by nausea, eructations, and hiccup. In some cases, moreover, it may become feculent in character; it is a remarkable fact, however, that fecal vomiting is comparatively rare in this condition, at least in comparison with acute internal strangulation of the bowel; this again is important to remember, for, as pointed out above, these two diseases are very similar in their manifestations, and are frequently very difficult to differentiate. Treves, for instance, reports fecal vomiting only 3 times in 20 cases of axial rotation of the bowel—that is, in only 15 per cent. The reason for this difference between volvulus of the sigmoid flexure and acute strangulation of the bowel is the different position of the obstruction : in the former instance, the bowel is occluded low down, toward the rectum, whereas in the latter the obstruction is, as a rule, situated high up—somewhere in the ileum.

Constipation is complete both for feces and flatus, and, as a rule, obtains from the very onset of the disease. The contents of the rectum may, of course, be evacuated by artificial means, and in some instances be spontaneously passed after the onset of the disease; occasionally the bowel contents evacuated at this time may be thin and of a liquid consistence. In rare instances the dejecta may contain a small quantity of blood ; the passage of bloody stools, however, is much more common in cases of acute intussusception of the bowel than in this condition. In acute intussusception of the bowel, moreover, the amount of blood is much greater and the passages of bloody material much more frequent than in volvulus of the intestine.

One of the most important symptoms of the disease is local mete-
orism ; this sign alone may enable a positive diagnosis of acute vol-
vulus of the sigmoid flexure to be made.   In the account of the general
features of occlusion of the intestine attention was called to the factors
that favor the development of meteorism, and its pathogenesis was con-
sidered at some length.   Meteorism, as a rule, develops rapidly in
volvulus of the intestine, and may attain immense proportions within
from forty-eight to seventy-two hours after the onset of the first symp-
toms ; in cases of this kind the abdomen soon becomes greatly dis-
tended, and may be balloon-shaped within a short time.   When
meteorism attains excessive degrees, its diagnostic significance is, of
course, greatly reduced ; nevertheless, it is of some value in the diag-
nosis even under these circumstances.   The only other condition in
which meteorism develops so rapidly and reaches such advanced
degrees is acute general peritonitis ; this disease, moreover, presents
a general clinical picture that in many respects resembles that of vol-
vulus ; in peritonitis, however, the abdomen is usually exquisitely
tender to the touch, whereas in the rapid form of meteorism which
occasionally develops in volvulus of the sigmoid flexure the abdomen
is only slightly tender on pressure, or in many instances not at all ;
with proper care in estimating the significance of symptoms acute and
rapidly progressing meteorism may be of considerable value in the
diagnosis of volvulus of the sigmoid flexure.

Meteorism, when occurring early in the disease and strictly localized,
is of paramount diagnostic importance.   When local meteorism develops
in acute volvulus of the sigmoid flexure, the left lower portion of the
abdomen protrudes ; a tense and elastic swelling, feeling very much like
an inflated rubber ball, can usually be felt there ; in the area of swelling
visible peristaltic movements are never seen ; on percussion, tympanitic
or metallic sounds are elicited.   In exceptional cases a dull percussion-
note may be heard over a tumor of this kind ; this is especially likely
to occur in cases in which the walls of the intestine are very edematous
and consequently thickened, or when the lumen of the bowel is filled
with large quantities of bowel contents ; the latter, as a rule, consist of
a mixture of blood and feces or of blood or feces alone.   These tumors
are nothing more nor less than the branches of the sigmoid flexure which
are completely distended by local meteorism, edematous, or filled with
blood and fecal material ; the two loops of the flexure are so close
together that they constitute a swelling that cannot be separated into its
two component parts by physical means.

As the development of gas proceeds, the loop of intestine rises more
and more and extends toward the right upper portions of the abdomen ;
ultimately it fills the whole abdomen, which becomes distended and
protrudes.   Under these circumstances the percussion-note over the
whole abdomen is tympanitic.   When the whole abdomen is distended
it becomes impossible, of course, to determine whether or not there may
be local meteorism from stasis in the colon or general meteorism of the
whole peritoneal cavity as a result of general peritonitis.   Bayer noticed

in a case of volvulus of the sigmoid flexure an S-shaped protrusion of the abdomen, most prominent at the upper left and lower right quadrants. I might mention here that in volvulus of the small intestine several tense elastic tumors that I have described may occasionally be felt and seen in different portions of the abdomen.

These general symptoms and collapse are, as a rule, very pronounced in the majority of cases of volvulus—particularly so in volvulus of the sigmoid flexure; here the same conditions obtain in this regard as in acute internal strangulation of the bowel. (For the details of the different clinical symptoms I must again refer the reader to the sections in which the general aspects of occlusion of the intestine were considered.)

In axial rotation occurring in other portions of the intestine—and in the small intestine—the same general symptoms arise as in volvulus of the sigmoid flexure; certain differences are, of course, observed that depend essentially on the anatomic peculiarities in regard to position and connections belonging to different parts of the bowel. The pain may be felt in many different parts of the abdomen, and is occasionally referred exclusively to the back, where it seems to remain permanently throughout the whole course of the disease. In nearly all the forms of axial rotation of the bowel involving portions of the intestine other than the sigmoid flexure blood is usually constantly absent from the stools, while tenesmus is also absent; there are, however, exceptions to this rule: Naunyn, for instance, reports a case of simple and uncomplicated torsion of the small intestine in which large quantities of pure blood were passed; the patient, on one day, passed as much as 1000 c.c.; on another, 400 c.c.; and on yet another occasion, 400 c.c. more. In these cases absolute constipation both for flatus and for fecal material does not necessarily always occur at the very onset of the disease; in fact, large quantities of fecal matter may be passed either spontaneously or after the administration of copious enemata for several days after the onset of the first symptoms of torsion of the small intestine high up. Finally, the position of the local meteorism is different in these cases: it does not always start from the left lower portion of the abdomen, as in volvulus of the sigmoid flexure, but may start from almost any part of the abdomen; its position will naturally vary according to the position of the twisted and distended loop of intestine. It might be expected that in many of the cases of axial rotation of the bowel involving parts of the intestine other than the sigmoid flexure the course of the disease would be more violent and more stormy than in the latter lesion; this, however, is by no means the case: a point on which special emphasis should be laid. The following is a brief account of a case under my care which illustrates these different points:

The patient was a man of thirty-one years whose bowels had always acted regularly and had never been confined. On the afternoon of May 4th he was suddenly seized with violent pain localized in the neighborhood of the umbilicus which lasted about ten minutes; very soon afterward he began to vomit and passed a pultaceous motion. Up to the fourteenth he did not vomit again, but suffered daily attacks of colicky pain that recurred frequently and were very violent. These colicky pains were always localized around the umbilicus. On the twelfth,

thirteenth, fourteenth, fifteenth, and sixteenth he passed, either spontaneously or after an enema, two or three thin watery stools; on the sixteenth, several lumps of solid fecal matter were found in the liquid dejecta that were spontaneously evacuated by the patient. On the fourteenth and the fifteenth, moreover, he suffered from attacks of vomiting. The attacks of colicky pain persisted until the patient entered the clinic on the seventeenth. The patient had the aspect of suffering and appeared ill, but was by no means in a state of collapse; the pulse was good throughout; the upper portion of the abdomen was tympanitic and distended, and there was also a moderate degree of meteorism in the flanks; the lower portion of the abdomen also protruded slightly (the other details of the physical examination of this patient will be found in the paragraphs on the diagnosis of the localization of this lesion on p. 604). The urine contained large quantities of indican. On the eighteenth, in the afternoon, the patient suddenly collapsed and died on the night of May 18–19th. At the autopsy the following conditions were found: The upper portion of the jejunum, beginning about six or seven centimeters below the duodenum, was converted into a convolution of greatly distended loops, showing a high degree of venous hyperemia and numerous hemorrhagic spots; the mesentery of the jejunum was twisted in different places. Throughout the whole large intestine and cecum pultaceous masses of fecal matter were found. There was also purulent peritonitis.

The clinical picture presented in knotting of the intestine is essentially the same as in axial rotation of the bowel; the symptoms, however, develop more rapidly and are also more severe than in the latter disease. Knotting of the intestine must be considered one of the most rapidly fatal diseases.

COURSE OF THE DISEASE.

The course of any case of complete occlusion of the bowel following axial rotation or knotting of the bowel is always acute; in fact, it may be said that the course of this particular form of occlusion is more rapid than that of any other form, with the possible exception of occlusion of the bowel following internal strangulation of the intestine. The severity of the symptoms and the rapidity of the course will largely depend on the length of the portion of bowel and mesentery that are strangulated: the longer this portion, the more violent the symptoms and the more severe the course of the disease. It can readily be understood, moreover, that knotting of the bowel must rapidly lead to the death of the patient, usually within the course of a day or two; it makes no difference whether the knotting of the bowel occurs between two loops of the small intestine or whether it occurs between a loop of the small intestine and the sigmoid flexure of the colon. In one patient death resulted within twelve hours after the onset of the first symptoms of the disease; in another patient (Kuettner), within fourteen hours after the first signs of the condition became apparent; the latter case, it is true, occurred in an old man suffering from senile decrepitude; death in this patient occurred with symptoms of the most profound collapse, while at the same time the mental faculties were perfectly free; the only symptoms that developed in this case were sudden pain and moderate distention of the abdomen; there was no vomiting at any time during the course of the disease. In this case the condition found postmortem was very remarkable and fully accounted for the rapidly fatal issue; the sigmoid flexure and no less than 21

feet of the 28 feet of the small intestine were in a condition of axial rotation and knotting, and were completely twisted.

In cases of simple and uncomplicated volvulus of the sigmoid flexure of the colon the course of the disease is generally somewhat more protracted, and on an average lasts for a week; the shortest duration of the disease is about three days; the longest, about three weeks. Death may result in several different ways in this lesion. In some instances the patient becomes collapsed; in others, he becomes thoroughly exhausted, and dies in this way; in still other cases general peritonitis develops; the latter complication is, however, rarely due to perforation of the intestine, since death usually occurs before gangrene and perforation of the bowel can supervene. Occasionally death appears quite suddenly without any premonitory symptoms; in cases of this character the condition of the patient may not have changed for the worse for several hours, when suddenly death occurs; it is probable that in such instances the fatal issue is brought about by paralysis of the heart due to the compression exercised on the organ by the tympanitic distention of the abdomen, which encroaches on the available intrathoracic space by upward displacement of the diaphragm.

Death is the inevitable result in every case of clinically pronounced complete axial rotation of the bowel with knotting—*i. e.*, rotation to 360 degrees—unless operative interference is undertaken in time to save the patient. As we have seen above, death may, of course, occur in many different ways in this disease.

I wish to call particular attention to the fact that in this disease, as in acute strangulation of the bowel, the internal administration of laxatives seems to accelerate the fatal issue.

Chronic axial rotation of the bowel: Spontaneous cure of this condition—that is, spontaneous rotation back into a normal position—is quite impossible after knotting has once occurred; this can readily be understood from a consideration of the anatomy of the parts and the pathologic anatomy of the lesion. The same applies to simple axial rotation of the intestine, provided the bowel is rotated so far that the intestine is completely rotated and twisted and distinct symptoms of occlusion of the intestinal lumen have made their appearance. On the other hand, there undoubtedly are cases of axial rotation on record, particularly of the congenital variety, which essentially constitute semi-rotations of the bowel around its own axis, and may persist for a long time, especially when the lesion is in the sigmoid flexure of the colon; these patients occasionally recover after having developed pronounced symptoms of intestinal obstruction and interference with the passage of fecal matter through the affected area, or even all the symptoms of acute occlusion of the intestine. In these cases the appearance of symptoms of acute occlusion of the bowel does not justify the prediction that the patient is doomed and that the disease will run a rapidly fatal course, as in the other forms of axial rotation mentioned above. The case reported on page 570, and quoted from Leichtenstern, is a very good example of this kind and fitly illustrates the meaning

I wish to convey. There are, moreover, a number of cases on record of rotation of the sigmoid flexure and of the small intestine around the mesenteric axis in which the final fatal attack was preceded by a number of alarming seizures pointing to occlusion, but which did not prove fatal; several of these cases gave a history of attacks of sudden pain, vomiting, constipation, and complete retention of feces for a time occurring several months before the last attack; as a rule, the retention of feces in these first attacks yielded spontaneously or as soon as the patients remained in one definite position—occasionally after the administration of opiates, after lavage of the stomach, or after the administration of enemata. In all these cases we are unquestionably justified in assuming that semirotation of the bowel around its axis occurred several times, corresponding to each attack of pain and retention of feces, and that in each instance the bowel rotated back into its normal position, so that its lumen again became patent and the passage of the motions was rendered possible; ultimately, however, the bowel was unable to rotate back into place and the patient succumbed to the attack. It is, of course, impossible positively to prove that these events actually occurred in all the cases that are recorded, but it is more than probable that in many instances semirotation of the intestine with restitution can occur in the way suggested; while, therefore, the explanation I have offered for the occurrence of these peculiar attacks preceding the fatal issue in cases of axial rotation of the bowel is purely hypothetic, the probability of the hypothesis is very strong.

[Thorburn[1] reports 2 cases in which sharply localized gangrene of the small intestine was found on opening the abdomen; he regarded them as cases in which a volvulus had become spontaneously reduced after injuring the intestine so as to produce gangrene.—Ed.]

DIAGNOSIS.

The following symptoms, which are common to all forms of acute occlusion of the intestinal lumen, are also characteristic of volvulus of the bowel: sudden pain in the abdomen, vomiting, retention of feces, and all those symptoms that denote collapse and constitute the syndrome characteristic for this state. All these symptoms, of course, merely denote occlusion of the bowel and nothing more, and in many instances it is quite impossible to carry the diagnosis any further than this point. In some cases, however, it is possible to at least suspect the existence of axial rotation and to determine that this lesion is the primary cause of the intestinal obstruction and the symptoms that are due to this condition. Occasionally, moreover, it is possible to make a diagnosis of volvulus of the sigmoid flexure, especially when the following train of symptoms develops in addition to the ordinary signs of occlusion of the intestinal lumen: meteorism appearing rapidly and remaining localized, especially in the left iliac region; in addition, tenesmus and the admixture of blood in bowel motions passed after the symptoms of occlusion have developed.

[1] Thorburn, *Medical Chronicle*, April–Sept., 1898, vol. ix. (new series), p. 164.

PARALYSIS AND MOTOR INSUFFICIENCY OF THE IN-TESTINE (Paralysis et Insufficientia Muscularis Intestini).

PARALYSIS of the intestine may be fitly discussed after narrowing and occlusion of the bowel, as, although it does not actually narrow or occlude the lumen of the bowel, it produces similar symptoms, since the passage of feces is interfered with and all its attendant results follow. The condition brought about by paralysis of the bowels is usually called paralytic or dynamic ileus, this term being used in contradistinction to the term mechanical ileus. A special section is devoted to this disease in order to define more clearly its clinical position and to make the description of the condition more comprehensive and intelligible.

Paralysis of the intestine means that the motor powers of the intestinal muscular walls are completely arrested. Insufficiency of the intestine means that these motor powers are merely insufficient or reduced; the paralysis or insufficiency may involve the whole length of the bowel, or may affect only a certain portion of the intestine—*i. e.,* either a small or an extensive area. These terms, moreover, are intended to describe functional disorders, which may be due to a great variety of primary etiologic factors. They may, of course, also occur in association with one or the other of the anatomic lesions already described, but this is not necessarily the case. The term "paralysis of the intestine" has long been employed, and the symptoms due to this disease have been described by many of the older writers; formerly, as has been shown in a preceding section, the collection of symptoms called paralytic ileus was believed to be due to paralysis of the bowel. Henrot was the first to enlarge the conception conveyed by this term and to introduce the term "pseudo-occlusion" or "pseudo-incarceration." He has recently made a classification of the etiologic conditions of paralysis of the intestine from various standpoints. Rosenbach deserves the main credit for having more clearly emphasized the significance of insufficiency of the intestine.

While no one doubts that paralysis of the intestine may occur in consequence of peritonitis or overdistention, yet the occurrence of a purely functional form, apart from nervous causes, is not generally conceded, though some would regard the paralysis as toxic or bacterial. We are absolutely certain, however, that a purely functional nervous paralysis does occur. Its infrequency is no argument against its occurrence in the abstract. Cases, such as one described by Werth and one of my own, admit of no other explanation. The cases of paralysis of the intestine may be grouped pathogenetically as follows:

1. **Functional Nervous Paralysis of the Intestine.**—Occasionally the clinical picture of paralysis of the intestine is presented without any direct involvement of the intestine proper. The primary factors which cause the development of the symptoms of paralysis of the intestine only indirectly affect the bowel—namely, to the extent of producing

paresis. Treves has reported a characteristic example of this group of cases, which may be quoted here by way of illustration : The patient was a child with all the symptoms of strangulation of the intestine—viz., great prostration, incessant vomiting, constipation, and a considerable degree of meteorism. The diagnosis of a strangulated hernia was made on the grounds that a hard and very tender tumor was felt in the inguinal region which could not be reduced ; this tumor was afterward found to be an inflamed, undescended testicle. The application of ice to the swelling soon caused all symptoms of inflammation to disappear, with improvement of the child's condition, and ultimately complete recovery. In other cases conditions, such as inflamed hydrocele, contusions of the testicle, operations on piles, inflammations and suppurations in the inguinal region, in a hernial sac, or even in the skin of the abdominal wall, have been known to produce the symptoms of occlusion of the intestine. I once observed a case in which the symptoms of paralysis of the intestine were due to a remote cause of this kind. The patient was a woman with serious valvular disease of the heart and ascites ; the accumulation of fluid in the abdomen was so great that paracentesis abdominis was performed in the usual manner, and a large amount of transudate, amounting to 6400 c.c., was evacuated ; soon afterward the symptoms of paresis of the intestine developed.

Cases have been recorded in which abdominal injury produced symptoms of occlusion of the intestine ; in the majority of these cases the injury was inflicted by a blunt instrument without producing an open wound ; in very severe cases of this character, with all the symptoms of occlusion, in which the patient died, autopsy has often failed to show any anatomic evidence of occlusion of the intestine ; it is difficult to decide in some of these cases whether or not the intestinal wall was really injured directly, or whether, possibly, these cases belong to this group—*i. e.*, whether the paresis of the bowel was merely an indirect effect of a remote cause which did not affect the bowel directly.

There is another group of cases where the primary etiology of the paralysis of the intestine is probably the same as above,—viz., indirect, —but in which the primary lesion is found in the intestine itself and not in some other tissue or organ, and must, at least, be considered the possible primary cause of the paralysis of the rest of the intestine. Paresis and paralysis of the intestine occurring after laparotomy are well known, although not frequent. Kocher believes that wherever intestinal paresis sets in, there is inflammation of the serosa, be it ever so slight, and that it is the peritonitis, on the one hand, and the circulatory disturbances attendant upon laparotomy, on the other, that actually cause the so-called dynamic ileus. In over 1000 laparotomies Werth saw 8 cases of ileus, 7 of which showed intestinal adhesions as concurrent hindrances to movability, in addition to the intestinal paralysis. In one case a " pure ileus paralyticus " was found on autopsy, all mechanical causes being absent. I have had one case myself analogous to the case of Werth. These cases are of the following type : A patient

undergoes an operation for the removal of a growth which is causing constriction of the lumen of the bowel; a portion of the intestine is resected, the patient makes a good recovery from the operation, and the patency of the bowel is established. The evacuation of the bowel, however, is insufficient or no motions at all are passed, and marked tympanites and all the other symptoms of intestinal obstruction rapidly develop. The patient dies, and at the autopsy the lumen of the bowel is patent throughout, and no anatomic obstruction to the passage of its contents exists in any part of the bowel, while the peritoneum may also be entirely normal. Perfectly analogous conditions are occasionally found after the relief of strangulation of the bowel—as, for instance, the release of an external or an internal hernia—or after the release of a portion of the bowel which has been twisted or is in a condition of volvulus; in these cases, even when the actual obstruction to the passage of bowel contents has been removed, there may still be absolute constipation and all the characteristic symptoms of intestinal obstruction; the same result occasionally follows laparotomies for the relief of intra-abdominal lesions other than those of the intestine—for example, of the female genital organs. This may also follow the reduction by taxis of a strangulated external hernia, even when this is quite successful and the lumen of the bowel is, anatomically speaking, patent. It is true that in the majority of these cases, as the statistics of modern surgery show, the possibility of a second mechanical obstruction to the passage of bowel contents elsewhere must always be thought of in the first instance. Examinations in the living subject, as a matter of fact, often show the presence of such real obstructive agencies after an apparently successful operation for strangulated hernia. Another group of cases may fitly be included in this group—namely, Littré's hernia, in which there are often symptoms of complete occlusion of the bowel, although the lumen of the intestine is by no means completely occluded. It must also be assumed that there is paralysis of the intestines in those cases of acute internal herniaform strangulation of the small intestine in which no feces are passed by the bowel. Here the obstruction of the lumen of the bowel may be very high up in the small intestine, but the contents of the large intestine are, nevertheless, completely retained; this phenomenon, it seems, can be explained only on the assumption of paralysis of the muscular coats of the colon.

Finally, it is to be observed that the clinical picture of intestinal occlusion occurs during severe attacks of renal or biliary calculous colic. We cannot at present decide whether it is the result of enterospasm or of enteroparalysis.

2. In a further series of cases marked anatomic lesions of the intestine produce motor insufficiency of the muscular walls of the bowel. In advanced cases the motor function of the intestine is completely arrested, and paralysis results. One of the most important anatomic lesions of the intestine causing an arrest of the motor function is acute general peritonitis; often, however, particularly in acute perforative peritonitis, paralysis of the intestine by reflex inhibition of its motor

function, as described above, may supervene; it is necessary to remember this and to distinguish, if possible, between the two. In chronic forms of peritonitis mechanical stenosis of the bowel is much more frequent than paralysis of the muscular walls of the intestine.

Paralysis of the intestine in embolism of the mesenteric artery, described elsewhere, belongs in this group. It includes also the intestinal insufficiency in stenosis and habitual constipation which sometimes becomes complete paralysis. Finally, this group includes paralysis from overdistention, as in excessive accumulation of gas.

In one of the preceding sections it was mentioned that no case had occurred in my own practice in which symptoms of paralysis of the intestine appeared in the course of intestinal ulceration—for instance, typhoid fever, tuberculosis, dysentery—except possibly in cases complicated by stenosis of the bowel as a result of the ulceration. I maintain, at all events, that paralysis of the intestine in simple ulceration without stenosis is exceedingly rare.

3. As a further distinct group, toxic enteroparalysis may be mentioned. In this form the suspension of motor activity is determined by the action of bacterial toxins on the intestine.

These various pathologic conditions, which may all produce paralysis of the intestine, are radically different in character; they all, however, have this in common, that they are capable of producing the clinical picture of intestinal obstruction. In all these cases, moreover, postmortem examination shows that the lumen of the bowel was patent throughout or was at least partially patent during a great part of the time that the symptoms of occlusion of the bowel were present—as, for instance, in the cases described in group 2; anatomic examination or exploratory operation of the abdomen in all these instances fails to reveal any anatomic explanation for the occurrence of complete functional impermeability of the intestine. On these grounds clinicians are forced to assume that in these cases the muscular wall of the bowel is paralyzed. It may be worth while now to investigate the genesis of this form of paralysis of the intestine as it presents itself to the mind of the clinical investigator. It is probable, of course, that the primary cause of the paralysis of the bowel and the exact mode of its development vary greatly in different cases, since, as we have seen above, this symptom may appear in such a number of different morbid conditions.

The first and the second group of cases present the greatest physiologic interest. According to the views now held by the majority of clinicians, this form of paralysis of the bowel is due to reflex stimulation of the inhibitory nerves of the intestine; as soon as this inhibition becomes sufficiently strong, the movements of the intestinal muscular coats are arrested; the chief inhibitory nerve of the intestine is the splanchnic, and it must be imagined that when its action becomes excessive, the peristaltic movements of the bowel are completely arrested. The centripetal stimulus starts either from the nerves in the vicinity of the intestine and peritoneum, or, still more frequently, from the nerves of the intestine and peritoneum themselves. It must be remembered

that the irritant which stimulates the splanchnic nerves reflexly also causes a slowing of the heart action and the phenomena of acute collapse ; this has been shown by Goltz in his celebrated " Klopf-Versuch " (" tapping experiment "). In this way a group of symptoms is produced which clinically resemble those of acute mechanical occlusion of the bowel due to strangulation. The clinical appearances in the two conditions, in fact, may be absolutely identical in every particular. We are hardly justified in calling this condition of the bowel a paralysis in the strict sense of the word, for it is really merely an arrest of peristalsis ; the functional effect of this arrest, however, is the same as that of actual paralysis.

Reichel has advanced another view, which is in direct antagonism to the theory just mentioned. He believes that paralyses of the bowel after laparotomies and herniotomies are not due to reflex inhibition of the muscular coat of the bowel, but to local infection of the bowel-wall, and supports his view by a number of experiments which are intended to show that local paralysis of the bowels can be brought about in this way. Even though no anatomic signs of peritonitis are found, it does not follow that there is no bacteriologically demonstrable peritoneal infection or true intoxication. In the same way Mannaberg states that in both pneumonia and purulent pleurisy he has seen the symptoms of intestinal occlusion arise, even to be so marked as to require operation, but no trace whatever of obstruction was found. The process which causes paralysis of the bowel-wall in peritonitis is not very easy to understand ; he, therefore, believes, and no doubt rightly, that the cases were due to toxic paralysis from absorption of toxic substances. Cases of this kind form the group previously described as toxic. Whether the bacterial poisons act upon the nervous apparatus of the intestine or upon its musculature is still entirely unknown.

Even though, as a rule, a direct toxic enteroparalysis appears very plausible, it can be accepted as an adequate explanation only in cases where its pathogenetic possibility exists—*i. e.*, in the presence of an acute infective disease. When there is no such condition, and the peritoneum is in no way locally affected, as in Treves' and similar cases, it is, no doubt, right to adhere to a neuropathic origin. My own opinion is that in this disease several different factors are responsible for the paralysis of the intestinal wall. Originally it was believed that the weakness or paralysis of the muscular coat of the bowel which is so frequent in peritonitis was due to direct anatomic involvement of the bowel-wall in the peritonitis. Stokes, who first advanced this view, believed that serous infiltration or collateral edema of the bowel-wall was sufficient to produce functional insufficiency of the muscular coat of the parts ; in other words, he held that the paralysis of the bowels is directly due to a participation of the bowel-wall in the inflammatory process affecting the peritoneum. I do not intend to combat the validity of this view nor to deny that it is fully justified in certain cases ; at the same time, I must confess that in many instances it must be considerably modified and curtailed before it can be regarded as a

correct explanation of the phenomena in question. This subject will be considered in detail in the section on Peritonitis. An important point to remember, and one which is interesting and significant in this connection, is that in the most acute forms of peritonitis—viz., perforative peritonitis and peritonitis following perityphlitis—in which the syndrome of intestinal occlusion and paralysis most readily appears, reflex irritation of the bowels may cause the complete picture of occlusion and paralysis of the intestine. Moreover, we must, undoubtedly, consider Reichel's theory of toxic bacterial action, as well as reflex irritation. One other possible cause for paralysis of the bowel may be mentioned—viz., the absorption of gas may be so much interfered with in a case of peritonitis that large quantities of flatus accumulate in the intestine and lead to stretching and distention of its wall; if the tension becomes excessive or too prolonged, the muscular coat of the intestine becomes insufficient and paralysis of the intestine develops, followed by all the manifestations of occlusion of the bowel. The last-named factor—viz., overdistention of the bowel-wall—also plays an important rôle in the development of meteorism from stasis which is so frequently seen in cases of real occlusion of the bowel immediately above the site of the occlusion. I have been able to show experimentally that in this condition the intestinal wall above the occlusion is so enormously distended by the accumulation of gas that even the strongest electric current applied directly to the distended portion of the intestine is unable to make the muscular coat contract. If the accumulated gas is got rid of by any means sufficiently early in the course of the disease, the contractility of the bowel-wall may be reëstablished; if, on the other hand, the gas remains in the intestine for too long a time, the bowel-wall ultimately loses its power to respond to stimuli; in this way overdistention of the bowel-wall may gradually pass into paralysis.

Paralysis of the bowel-wall also occurs in another very important group of diseases—viz., habitual constipation and chronic stenosis of the bowel. The mode of origin of this serious complication in these diseases is not the same as in the preceding class of cases. Pathogenetically two groups of cases can be distinguished. In some cases of long-lasting fecal accumulation and in all cases of stenosis of the bowel which come on slowly hypertrophy of the muscular coat of the bowel develops immediately above the obstruction. This process must be regarded as compensatory in character and as primarily intended to overcome the resistance offered to the passage of the bowel contents by the narrowing of the bowel-lumen. Eventually, however, the muscular wall of the intestine becomes, so to speak, fatigued, and the compensation fails; the process is exactly analogous to the fatigue and failing compensation in other groups of muscles which undergo hypertrophy in response to an excessive amount of work. Finally, the powers of the muscular coat of the intestine become insufficient to overcome the obstruction offered to the passage of the feces, and intestinal insufficiency results. When this occurs, the intestinal contents are no longer driven on; stasis of

fecal material occurs, and all the symptoms of complete occlusion of the bowels develop. In cases in which the muscular wall of the intestine is able to rest between the periods of excessive work, and provided that no overdistention of the intestinal wall occurs from the gaseous and semisolid contents of the bowel in the mean time, or in cases in which the obstruction is artificially removed, the dangerous condition may be relieved and the patient may recover. In many cases, however, a vicious circle is created before the muscle has an opportunity of resting. In these instances the symptoms of total occlusion develop in the manner described above. When the muscle can rest, however, the intestinal insufficiency must be regarded merely as a transitory condition, which does not progress so far as to lead to complete paralysis of the bowel. In course of time, however, histologic degeneration of the hypertrophied muscular coat occurs in all these cases. This process is exactly analogous to similar degenerations in the heart muscle in cases of valvular disease where compensatory hypertrophy of the ventricles of the heart has occurred. In cases of this kind insufficiency of the intestinal muscular coat gradually passes into complete paralysis, frequently without the presence of any microscopic changes in the muscular coat. The result of this paralysis of the bowel-wall is intestinal obstruction, which can be relieved only by surgical interference. The same conditions may develop, in the second place, in cases of permanent fecal accumulation, even when the muscular coat of the bowel is normal but not hypertrophied. The pathogenesis of this form of paralysis is the same as that of paralysis following failure of compensatory muscular hypertrophy. Finally, in cases where atrophy of the intestinal muscular coat occurs, insufficiency and paralysis of the bowel may develop still more readily.

Leichtenstern has called attention to certain factors present in many cases of acute internal strangulation and axial rotation of the bowel which may produce insufficiency and paralysis of the bowel-wall. His statements are so much to the point that they are reproduced here verbatim : " A dynamic factor plays an important rôle in the loss of motor power. Insufficiency of the intestine may be produced in two ways : in the first place, the lumen of the bowel may be partially occluded by some mechanical agency and paralysis of the affected area of the intestine may develop as a result of disturbances in the circulation of this injured loop ; this must eventually be followed by complete stasis of the bowel contents and absolute constipation. In the second place, insufficiency of the intestinal muscular coat may be the primary process and secondarily lead to strangulation." This is seen in volvulus of the sigmoid flexure.

From the description just given it will be seen that either short portions of the intestine may become paralyzed or insufficient, or that the paralysis or insufficiency may involve extensive areas of the intestine. The paralyzed portion of the bowel is usually inflated and distended with gas ; this is due to loss of muscular tone of the bowel. Leichtenstern, however, emphasizes the fact that occasionally the paralyzed

bowel may be found contracted at the autopsy. He believes that these cases clearly show the actual condition of the muscular coat of the intestine, and that in certain cases the bowel-wall is actually in a state of contraction, although the symptoms of intestinal paralysis were present during life. I have no hesitation in indorsing this view, for, as we have seen above, paralysis of the intestine does not necessarily mean that the bowel-wall is paralyzed in the sense that the muscular tissue of the intestinal wall has actually lost all power of contraction. In many cases, as I have shown, there is merely inhibition of the muscular contraction, which may quite well be due to reflex irritation of inhibitory nerves. It is quite possible, therefore, for the symptom-complex of paralysis of the bowel to develop without actual paralysis of the intestinal muscles; indeed, the bowel-wall may even be in a condition of contracture, as indicated above.

In paralysis of the bowel-wall the mechanism of intestinal obstruction is the following: The driving power of the intestinal wall becomes insufficient, and, as a result, the contents of the affected segment of intestine remain stationary and are not propelled onward. Stasis of bowel contents and accumulation of fecal matter naturally occur, the material successively filling different portions of the intestine in a direction upward toward the stomach. When this occurs, all the processes and conditions seen in cases of mechanical obstruction of the intestine develop. In many cases, moreover, the loops of intestine become so overloaded with fecal material that they become displaced, drop down in the abdomen, and may even become kinked. It will be seen, therefore, that the same primary and secondary effects occur in cases of paralysis of certain portions of the bowel-wall as in cases of mechanical occlusion of the intestine.

This view of the mechanism of the obstruction in paralysis of the intestine is generally recognized as correct. Leichtenstern, however, has put forward another theory by which he attempts to explain certain cases of impermeability of the intestinal lumen, particularly those occasionally seen in peritonitis. He speaks of this process as gas occlusion,—" Gassperre,"—and argues that " an intense degree of general gaseous meteorism may exercise the same effect as a stricture of the uppermost portions of the small intestine, particularly when the intestine becomes paralyzed and is fixed in one position by meteorism." This view must be interpreted as follows: Those loops of intestine that are inflated and distended with gas, particularly if they have a long mesentery, always rise upward in the abdomen and occupy the highest possible position. When the patient is lying on his back, these inflated loops naturally rise upward and press against the anterior abdominal wall. The distended loops, on the other hand, which have a short mesentery, particularly the duodenum and the uppermost portion of the jejunum, do not move far away from the spinal column, or, in fact, do not move away from it at all. It can, therefore, be understood that if the patient is lying in the dorsal position, the pylorus will be situated lower down than the distended loops of intestine, which have

risen upward against the anterior abdominal wall. The natural result of this displacement is that the liquid contents of the duodenum and of the upper part of the jejunum, in obedience to the law of gravity, very readily run backward into the stomach and do not pass on into the distended portion of the bowel. In this way gaseous meteorism produces results which are exactly the same as those of stenosis of the small intestine.

The clinical picture presented by a case of paralysis of the intestine is exactly like that of mechanical occlusion of the bowel. The etiology, of course, of any given case may differ, so that there are different forms, some with, some without, symptoms of strangulation, while some develop acutely and others slowly. In many instances there are vomiting, absolute constipation, meteorism, then fecal vomiting, and in acute cases very severe prostration. It will be noted, therefore, that the general clinical features of paresis of the intestine are exactly like those of internal strangulation of the bowel and of volvulus. The condition of insufficiency or paralysis of the bowel which frequently supervenes in fecal accumulation may exactly imitate carcinomatous or cicatricial stricture of the intestine which has gone on to complete occlusion of the intestinal lumen.

The diagnosis of this condition may be exceedingly difficult, particularly in the acute cases. When paralysis of the bowel is brought about by peritonitis, the diagnosis may be exceptionally obscure. [Speaking generally, leukocytosis is in favor of peritonitis.—Ed.] Attention will be called to the salient points in the differential diagnosis in the section on the Diagnosis of Stenosis and Occlusion of the Bowel, to which the reader should refer for the details. The diagnosis of paresis must be based on the results of the physical examination described in that section.

The course and the prognosis vary greatly and essentially depend on the cause of the condition. In cases due to inflammation of the testicles the most severe and acute symptoms may subside and recovery follow if the inflammation be reduced and the severe nervous irritation which is directly responsible for the functional paresis of the bowel be allayed. In those cases of paralysis of the bowel following surgical operations on the intestine the prognosis is very grave. In cases in which paresis of the bowel is due to peritonitis the prognosis is, as a rule, lethal. Specially interesting conditions are occasionally observed in the form of relative insufficiency of the bowel-wall, which is frequently seen in the intestine immediately above a mechanical stricture. In cases of this character typical attacks develop which present all the features of complete occlusion of the bowel, and are, in fact, due to complete occlusion. But these symptoms often pass away completely either spontaneously or as the result of treatment. If the primary cause of the stenosis is of a benign character, attacks of this kind may be frequently repeated. Even under these conditions, however, the prognosis is unfavorable, since permanent paralysis of the muscular coat of the intestine eventually supervenes and terminates the scene. This unfor-

tunate result can, of course, in some instances be prevented if operative interference is undertaken at an early stage of the disease and the
primary anatomic cause of the trouble is removed.

[" Ballooning " of the rectum is due, according to Treves, to some
phase of paralysis, and should, therefore, be mentioned here.   The
lower part of the rectum is greatly dilated, but is not blown out with
gas, as the name ballooning implies ; it gives the impression as if the
rectum were a rigid cavity, like the inside of a cocoanut, lined by
mucous membrane.   It is often, but not necessarily, associated with stricture or new growth in the descending colon or sigmoid flexure.—Ed.]

DIAGNOSTIC REMARKS ON STENOSIS AND OCCLUSION
OF THE INTESTINE.

THIS subject is one of great importance to every medical practitioner,
and interests the surgeon as much as the physician.   The diagnosis of
stenosis and occlusion of the bowel is extremely important for several
reasons.   In the first place, the clinical symptoms and the lesions themselves are very severe and dangerous ; in the second place, stenosis and
occlusion of the bowel frequently call for rapid and dogmatic decisions
on the part of the medical man, as it is usually imperative that something
should be done at once.   In these diseases, in particular, all uncertainty
and all vacillation must be avoided, for uncertainty is probably more
dangerous here than in any other disease ; lastly, the diagnosis is frequently exceedingly difficult, and therefore requires detailed consideration in a special section.   The diagnosis of these lesions and of their
results is more difficult than in any other symptom-complex in the
whole of medicine, and it must be fully recognized that the diagnostic
difficulties are often numerous and in many instances almost insurmountable.   It is, therefore, desirable to consider the diagnosis of stenosis and
occlusion of the bowel in a special section.

In each individual case three diagnostic problems must be considered and, if possible, solved—namely :

(*a*) Is the clinical picture presented due to stenosis or occlusion of
the intestine ?

(*b*) If so, in what portion of the intestinal canal is the stenosis or
occlusion situated ?

(*c*) What is the anatomic character of the lesion producing the
stenosis or occlusion of the intestine ?

Every medical man who has had some experience in this class of
diseases will undoubtedly agree that occasionally, when the conditions
happen to be favorable, these three questions can be satisfactorily answered, but that in the great majority of cases it is quite impossible to
do so ; that, in fact, in many instances the first fundamental question as
to the existence of stenosis or occlusion must remain unanswered.   Even
the most experienced surgeon and the most skilful physician must
acknowledge that every fresh case of this disease frequently presents
most unsuspected and surprising features.

We will now proceed to consider these three questions, devoting a special section to each one.

THE DIAGNOSIS OF STENOSIS AND OCCLUSION OF THE INTESTINE.

The Diagnosis of Stenosis of the Intestine.—For a description of the individual symptoms of stenosis of the bowel the reader must refer to the description of this lesion given in preceding sections ; this remark also applies to the individual symptoms of occlusion of the bowel. Only a general summary of the more important symptoms will be given here.

Occasionally the diagnosis of stricture of the intestine can be made with absolute certainty ; in other cases the diagnosis is simply impossible. A certain number of cases occupy an intermediate position between the two in which the diagnosis can be made with more or less probability. The diagnosis of stricture of the bowel may be considered positive and absolutely certain under two conditions : (1) When digital examination of the bowel definitely proves the existence of a stenosis of the rectum in cases where the functional disturbances, described in the preceding paragraphs, of stenosis of the bowel are fully developed ; (2) when inspection of the surface of the abdomen shows the peculiar tetanic stiffening of certain loops of intestine and those increased peristaltic movements of the bowel during the paroxysms of colic which were described at length in a previous section and shown to be pathognomonic of stenosis of the bowel. Conversely, the diagnosis of stenosis of the intestine must be considered impossible when an impalpable stenosis allows the intestinal contents to pass through the stricture ; this may be due either to the fact that the contents of the bowel in the affected portion—for instance, in the small intestine—are chiefly fluid, or to the fact that the stricture is not sufficiently advanced to interfere with the passage of semisolid and solid fecal matter. [It may also occur in rare instances of spontaneous anastomosis in cases of intestinal carcinoma. Pollosson [1] has recorded two cases, and the editor has had a case of this kind under his care.—ED.]

The existence of constipation alone naturally does not justify a diagnosis of stenosis of the intestine, as it may be due to a great variety of causes. Nevertheless, the existence of an apparently simple and uncomplicated constipation may occasionally arouse the suspicion of stenosis of the bowel, and may sometimes direct attention to this diagnosis. This suspicion is sometimes corroborated by a careful study of the different data of the history of the case. In other instances a study of the history may immediately dispel the suspicion of stenosis ; for instance, when a patient has had habitual sluggishness of the bowel for a number of years, the question of stenosis of the bowel need hardly be considered ; but if, on the other hand, obstinate constipation appears without recognizable or assignable cause in an individual who previously had perfectly regular actions, and in whom no other primary cause of obsti-

[1] Pollosson, *Lyon Médical*, April 16, 1899, p. 557.

nate constipation (for instance, an acute attack of intestinal catarrh, sudden changes in the mode of life, psychic factors, neurasthenia, and many other causes) is forthcoming, the possible existence of stenosis of the bowel must always be considered. This suspicion will be strengthened if the patient has recently suffered from some disease which may produce stricture of the intestine, such as dysentery, typhoid fever, intestinal tuberculosis, or some form of intestinal ulceration, general or local peritonitis, such as perityphlitis, inflammation of the female sexual organs, pericholecystitis felleæ, etc., conditions which may all lead to constriction and kinking of the bowel ; or if there is a history of external hernia having been reduced by taxis, or, finally, if there is a history of some foreign body having been swallowed ; again, if in addition to obstinate constipation the patient becomes weak, anemic, and emaciated, and there is no other satisfactory explanation for this group of symptoms, stenosis of the bowel should always be thought of. We may even go further than this, and very strongly suspect the presence of a carcinomatous stricture. Careful consideration of all these points will prevent the grave diagnostic error of regarding such a case as one of simple and harmless constipation. It is bad practice to treat a case of this kind carelessly. The medical man should always consider the possibility of a more serious condition. When careful consideration of the possible causes of habitual constipation raises a suspicion of grave disease in the medical attendant's mind, the patient really gains.

The diagnosis becomes more than a vague suspicion, and may be considered fairly probable, when in addition to simple habitual constipation paroxysmal attacks of colic, with or without visible peristaltic movements of the intestine, develop. In interpreting the significance of paroxysmal attacks of colic, it must at the same time never be forgotten that such paroxysms may also be due to simple fecal accumulation, and that the patient may not be suffering from any typical form of stenosis of the intestine, but merely from simple habitual sluggishness of the bowels (compare p. 103). In cases of this character the accumulated masses of the fecal matter themselves constitute the obstacle to the passage of the bowel contents.

When habitual constipation and, in addition, the appearance of paroxysmal attacks of colic suggest the existence of stenosis, a physical examination of the patient should be made. The suspicion that enterostenosis exists is greatly strengthened and becomes very probable, or may in any given case even become positive, when physical examination reveals conditions which may be directly responsible for the occurrence of the symptoms described ; in other words, when conditions are discovered which may directly or indirectly interfere with the permeability of the intestinal lumen. To this class of lesions belong, for instance, tumors which may either be neoplastic or inflammatory in character ; further, displacement of solid abdominal organs, tumors due to intussusception, chronic peritoneal adhesions, and old external hernias.

Another feature which must be studied in the diagnosis is the character of the dejecta ; the appearance of blood or pus in the motions,

of alterations of the form of the stools in such a way as to constitute so-called " stenosis feces," are important guides in the diagnosis of intestinal stricture.

On the other hand, it must never be forgotten—and special stress should be laid on this point—that the functional symptoms of stenosis (apart, of course, from such pathognomonic functional symptoms as rigidity of loops of intestine and energetic visible peristaltic movements) may all occur in the absence of any real stenosis of the intestine. It will be seen from these remarks that in order to avoid error in the diagnosis great caution and care are necessary in each individual case.

The Diagnosis of Occlusion of the Intestine.—At the commencement of this paragraph we must emphasize the old, important rule which should *always be followed:* In every case which even in the remotest way causes suspicion of intestinal occlusion, examine first all possible hernial rings, and then the rectum. Neglect of this rule has often been bitterly regretted.

Even the most experienced physician will occasionally commit very serious errors in the diagnosis of occlusion of the intestine, which it may be added are sometimes quite unavoidable. Mistakes in diagnosis may not only occur in obscure or undeveloped cases, but also in cases which apparently present a typical clinical picture.

Mistakes in diagnosis in cases of occlusion of the bowel may be made in two directions : in one class of cases the characteristic symptoms of occlusion of the bowel are more or less fully developed, and on these grounds the diagnosis of that condition is accordingly made. The subsequent course of the disease, or the results of surgical interference, or, finally, the data derived from the autopsy, show that all these symptoms were merely functional disorders simulating occlusion of the bowel, without any anatomic basis for this condition. The symptoms, it will be found, were produced by some other affection. In another class of cases the converse error in diagnosis is committed : the presence of misleading symptoms or indefinite physical signs masks the real state of affairs and leads the medical attendant to diagnose some condition other than occlusion of the intestine, which, as shown by the subsequent course of the disease, exploratory laparotomy, or autopsy, is present.

The diagnosis of occlusion is, practically speaking, based on the local intestinal symptoms which result from complete impermeability of the intestinal lumen. The symptoms typical of this condition are absolute constipation, neither flatus nor feces being passed by the bowel, and meteorism and vomiting which may become stercoraceous. And yet all these symptoms— even fecal vomiting—may be present without mechanical or anatomic occlusion of the intestine.

The most important disease which produces stercoraceous vomiting in the absence of occlusion of the bowel is paralysis of the intestine. In any given case, therefore, the first question to be answered is whether or not, as it is now expressed, the condition is one of " mechanical or of

dynamic ileus." In the section on Paralysis of the Intestine this matter was considered in detail and the most important points then brought out will be merely recapitulated here.

All the symptoms of occlusion, including fecal vomiting, may appear after laparotomy or after any operation on the abdominal viscera, particularly on the intestines themselves, in consequence of paralysis of the intestine. The medical man must consider the possibility of paralysis of the intestine in cases where the symptoms come on after an abdominal injury, particularly by some *dull* trauma. Paralysis of the intestine must also be thought of when fecal vomiting persists after a strangulated hernia has been relieved by an operation—that is, after the mechanical obstruction to the onward passage of the feces has been removed. After the reduction of an external hernia by taxis, symptoms of occlusion of the bowel often persist from paralysis of the bowel. In cases of the latter kind, however, it must, in the first place, always be assumed that the reduction of the hernia was really only apparent, and that the mechanical obstruction was in reality not removed. This assumption is always much more plausible than the one that paralysis of the intestine exists and causes the fecal vomiting.

Intestinal paralysis of toxic origin must also be taken into consideration when symptoms of occlusion appear in the course of an infectious disease. A recent observation of Mannaberg's will best demonstrate the extreme importance of this rule : An elderly man, for years a sufferer from arteriosclerosis and cardiac hypertrophy, had one day an attack of illness with slight fever, pain in the left hypochondrium, rapid development of severe meteorism, retching, vomiting, and complete constipation. Gradual increase in severity of all symptoms until fecal vomiting appeared. No trace of peristalsis ; no ascites demonstrable ; nothing palpable in rectum. Opposed to this, quite considerable left-sided pleuritic exudate. Mannaberg diagnosed the case as toxic paralysis of the intestine. Colostomy was followed by death two days later. On section, there was no sign of obstruction in the intestine ; intestinal wall wholly intact ; no anomaly in position of bowels. Profuse, ill-smelling exudate in left pleural cavity ; on the surface of the lower lobe, an abscess about the size of a pea.

Intestinal paralysis from embolism of the mesenteric artery may offer insurmountable obstacles to diagnosis (*cf.* p. 286).

When the symptoms of occlusion occur as enteroparalysis following peritonitis, the diagnosis may become exceedingly difficult. The decision in these cases hinges on the diagnosis of peritonitis. On account of its great practical importance, a fuller consideration of this question will be entered on later.

Other conditions besides intestinal paralysis may simulate occlusion of the intestine. Thus, the symptoms occur at times in the hysterical and nervous, perhaps also in consequence of intestinal paralysis or of actual antiperistalsis In the latter case formed fecal masses may be vomited (*cf.* p. 385). As this has hitherto occurred only in the hysterical and nervous, we cannot fail to arrive at the correct diagnosis.

At other times the individuality of the patient and the usually rather bizarre clinical picture will guide us correctly. But the decision is not always simple, and, of course, it must never be forgotten that hysterical subjects may have genuine occlusion of the bowels.

Hemorrhage and necrosis of the pancreas have often run a course showing the clinical picture of occlusion of the intestine, so that laparotomy was performed. It is only in extremely exceptional cases that even a hypothetic diagnosis of pancreatic disease is possible.

Renal and gall-stone colic sometimes shows the clinical picture of intestinal occlusion even without actual closure of the intestine by the calculus. The same holds true of lead colic and other severe enteralgias, particularly in the so-called flatulent colic. Acute arsenical poisoning has also been mistaken for it. It must be emphasized especially that appendicitis of very acute onset may at first sight simulate the symptom-complex of occlusion. We shall confine ourselves to these references alone, as in this work we cannot enter into the details of these and similar errors.

It is to be remembered that fecal vomiting from gastric, colonic, or jejunocolonic communication may lead to a false diagnosis. Careful consideration of the entire clinical course, and particularly the employment of high rectal injections of liquid colored with a neutral coloring-matter, or the introduction of such a colored fluid into the stomach, will usually help us to a correct conclusion. In the differential diagnosis the forcing of gas into the rectum is of service. Scholz contributes an interesting case from Kraus's clinic. By forcing air into the bowel the sigmoid flexure, descending colon, and stomach were inflated with comparative ease, while it was difficult or even impossible to distend the ascending colon and cecum. The passage of air from the large bowel into the stomach may be attended with a plainly audible noise, and shortly belching from the stomach may occur, the gas having a distinctly feculent odor.

The diagnosis of intestinal occlusion is, on the whole, comparatively easy. Fecal vomiting is a direct indication of it, but there is more difficulty when fecal vomiting is absent, especially when the only localizing symptoms of occlusion are simple vomiting, constipation, and meteorism. It is perfectly obvious that this combination of symptoms, even though occurring together acutely, cannot clinch the diagnosis of occlusion absolutely, for these three symptoms may appear in a number of affections, some of which have been named above, and they are, therefore, too doubtful alone ; they must in consequence be supported by the other clinical symptoms.

In one case we have another and a very definite symptom of occlusion, no matter what other conditions may be simulated. This symptom is localized paroxysmal rigidity of the intestine combined with visible and energetic peristaltic movements of the bowel. As already stated, this occurs exclusively in intestinal stenosis with hypertrophy of the muscular coat of the intestine. If, then, in addition to symptoms that may be due to occlusion of the intestine, but might just as

well be due to some other cause, there is a peristaltic storm, such as has been described above, we may be certain that occlusion from stenosis is the correct diagnosis.

It is, of course, quite unnecessary to emphasize particularly that visible rigidity and peristalsis are in nowise essential symptoms of all forms of occlusion. On the contrary, they are almost constantly absent in the forms with an extremely acute onset, or, in other words, those that occur suddenly in a previously normal intestine (volvulus, internal incarceration, obturation). It is a characteristic symptom only in occlusion from chronic stenosis. This fact is repeated only to prevent possible errors.

To illustrate converse errors, we may state that a diagnosis of meningitis has been made because delirium, vomiting, constipation, and retracted abdomen existed, while in reality occlusion of the jejunum caused the syndrome. Even cholera has been diagnosed where in reality " cholera herniaire " existed. An error of this kind is readily conceivable during a cholera epidemic. At the present time, besides the rice-water stools, the absence of meteorism, and the initial pain, true cholera is distinguishable to a certainty by bacterial examination. It is in the very acute cases of occlusion with severely painful onset that confusion is most probable.

The most serious and the most frequent source of difficulty in the diagnosis of acute occlusion of the bowel is peritonitis. In the first place, it is not always easy—it may, in fact, be quite impossible—to decide whether or not occlusion of the bowel is complicated by peritonitis even when occlusion of the bowel is known to be present. In those cases, of course, in which it is impossible to decide positively whether a definite symptom-complex is produced by general peritonitis (in particular, perforative peritonitis or *peritonitis ex perityphlitide*) or by acute occlusion, the diagnosis is still more difficult. All the symptoms considered to be characteristic of one condition may also appear in the other, and, conversely, certain symptoms may appear in either which are usually considered as essential traits of the other clinical picture. The only way in which a differential diagnosis can ever be made in these cases is by careful and conscientious study of all the symptoms and special features of the case. (For the details of the symptom-complex presented either by peritonitis or by acute occlusion the reader should refer to the sections on these diseases.) One of the most important points in the differential diagnosis is to determine whether the onset of symptoms is very recent or whether it is of some standing, such as several days. From this point of view the individual symptoms of these cases should be analyzed.

Intense spontaneous pain may appear in both affections. The general character of the pain, however, is of considerable importance. When the violent spontaneous pain persists, and in addition there is general tenderness on pressure over the abdomen, and the patient remains absolutely quiet and carefully avoids all voluntary movements, peritonitis is more probable than occlusion of the bowel. True peri-

tonitis may frequently complicate acute occlusion of the bowel very soon after the onset of the first symptoms ; nevertheless, it may be said that even in the most rapid cases an interval of from twenty-four to forty-eight hours must elapse before the symptoms of peritonitis become manifest. If the patient be examined during this period, extremely important points bearing on the differential diagnosis can usually be made out. On the other hand, it must never be forgotten that absence of tenderness on pressure is by no means conclusive of occlusion of the bowel, or opposed to the diagnosis of peritonitis, for cases of peritonitis, even of the acute and purulent form, occasionally present a minimal amount of tenderness on pressure and of spontaneous abdominal pain. Again, on the other hand, meteorism may develop rapidly in some cases of volvulus and internal herniaform strangulation of the bowel, and lead to a considerable degree of tenderness on pressure. Taking all these possibilities into consideration, it may, nevertheless, be stated that pronounced tenderness on pressure always points to peritonitis rather than to occlusion of the bowel.

The character of the meteorism that develops may occasionally be of use in the diagnosis ; in some instances, of course, the existence of meteorism may, on the other hand, complicate matters and make the diagnosis more difficult. When meteorism is definitely localized in the early stages, and inflated and tensely distended loops of intestine can be distinctly felt and shown by combined auscultation-percussion to occupy a circumscribed portion of the abdomen, the diagnosis of volvulus or of internal strangulation of the bowel may be made, and the existence of peritonitis excluded. In other words, when there are visible, palpable, and audible signs of localized meteorism, the diagnosis of peritonitis becomes improbable and that of volvulus or of internal strangulation probable. It is not so easy to decide on the significance of general tympanites, for the excessive abdominal distention naturally makes any careful examination of the abdominal contents impossible. Both in occlusion and in peritonitis general meteorism may occur. It will be seen, therefore, that instead of settling the diagnosis, this sign more frequently has a tendency to obscure it. Some authors maintain that such advanced degrees of meteorism develop more rapidly in the latter disease than in the former, but this view is erroneous. In the first place, tympanitic distention of the abdomen in peritonitis may come on quite slowly, and does not necessarily develop rapidly ; while conversely the abdomen may very rapidly become distended with gas in occlusion. In a case of general meteorism in which the differential diagnosis between peritonitis and occlusion of the bowel has to be made, it should be carefully determined by inspection, palpation, and auscultation whether or not there are any signs of intestinal movements ; absence of all signs of intestinal movement may be considered an index of the presence of peritonitis with paralysis of the intestine ; at this stage " regt sich in der weiten Grabesstille des Leibes nur noch der Schmerz " (Schlange). Even this sign, however, is not

absolutely diagnostic of peritonitis.   This point will be referred to again below.

Tetanic rigidity and even retraction of the abdominal walls are occasionally seen both in the early stages of peritonitis from perforation and in internal herniaform strangulation of the bowel.   This sign, therefore, alone does not furnish us any definite indications as regards the presence of the one or the other condition.

Many writers believe that the presence of a fluid exudate in the abdominal cavity is an important indication of peritonitis and greatly strengthens this diagnosis.   Here too, however, great care is necessary in forming an opinion, for, in the first place, it is well known that in internal herniaform strangulation of the bowel, and particularly in volvulus of the intestine, venous stasis occurs in the mesentery, and may lead to a copious blood-stained transudate, which may fill a considerable portion of the abdominal cavity, and so imitate the fluid exudation of peritonitis.   Conversely, a fluid peritonitic exudate may actually be present and escape detection by physical examination, for in many of these cases, as we have seen above, loops of intestine distended with gas are in close contact with the anterior and lateral parietes of the abdominal wall, so that both anteriorly and in the flanks a hyperresonant note is obtained on percussion.

Fever is correctly regarded as a very important symptom of peritonitis, and in many instances may decide the diagnosis in favor of this disease.   A rise of temperature, however, cannot by any means be considered an absolutely reliable symptom, for, in the first place, true occlusion of the bowel may subsequently become complicated by febrile peritonitis; it has been seen above how rapidly peritonitis with pyrexia may develop after the onset of true occlusion of the bowel.   In a case of this kind, therefore, in which the medical attendant fails to discover that the first onset of the disease was afebrile, a mistake may easily arise, for examination of the patient twenty-four to forty-eight hours after the onset of the disease may show fever due to secondary peritonitis, and may thus lead to a diagnosis of peritonitis alone.   In the second place, even acute, general suppurative and gangrenous forms of peritonitis may run their course without fever, or even with a subnormal temperature.   It is only under the following conditions, therefore, that the temperature can be considered of some diagnostic value—viz., when the temperature of the patient is taken immediately after the onset of the first signs of illness and is then found to be raised; the diagnosis of peritonitis is then justified.

Occasionally indicanuria, or, better, the absence of indicanuria, is of use in the differential diagnosis between peritonitis and intestinal occlusion.   In acute general peritonitis, and this is the form of peritonitis with which we are chiefly concerned here, indicanuria is hardly ever absent; therefore in a case where the diagnosis is doubtful, absence of indicanuria is against the diagnosis of peritonitis and in favor of occlusion of the large intestine.   The presence of indicanuria, however, is if no assistance in the differential diagnosis between occlusion of the

small intestine and peritonitis, for indican is excreted in both of these conditions (compare also the account of indicanuria on pp. 166, 167, and the remarks below on the localization of occlusion of the intestine).

Another useful sign in the differential diagnosis is the presence or absence of visible peristalsis and visible stiffening and rigidity of certain loops of intestine. My personal experience justifies the statement that exaggerated rolling peristaltic movements of the bowel, and especially paroxysmal stiffening of coils of intestine, can be due only to some interference with the lumen of the bowel, and never to acute peritonitis. If, therefore, in any given case exaggerated visible peristaltic movements and paroxysmal stiffening of certain loops of the intestine are present, the diagnosis of intestinal occlusion is justified. But the existence of the latter in an individual case is by no means entirely excluded by this.

On the other hand, I am not inclined to indorse the view advanced by some clinicians that acute peritonitis may be absolutely excluded in all cases in which visible peristalsis and paroxysmal stiffening are present. The advocates of this idea believe that peritonitis always causes paralysis of the intestine, and that it is therefore impossible to see intestinal movements, or even to elicit intestinal contractions by irritating the abdomen (tapping, cold) in any case of acute peritonitis. As I have said, I do not believe that this is always the case, for I have undoubtedly seen cases of stenosis or occlusion of the intestine complicated by acute and even purulent peritonitis, in which visible peristalsis and paroxysmal rigidity and stiffening of certain loops of the intestine were undoubtedly present. In the section on Acute Peritonitis a more detailed account of the influence of this lesion on the movements of the intestine will be given.

I am also unable to indorse the postulate that the cessation of intestinal movements which were distinctly visible up to a certain time always proves that secondary peritonitis has come on. This statement may be true to a limited extent, but it should not be so generally accepted as some clinicians demand. In other words, the diagnosis of secondary peritonitis from the cessation of intestinal movements may be, but is not necessarily, correct, for it is well known that the peristaltic movements of the bowel may stop when the intestine becomes overdistended, and that this overdistention and overstretching of the intestinal wall may be due to the development of meteorism from stasis, even in the complete absence of peritonitis. Further, on p. 588, attention was called to another possible method by which paresis or paralysis of the intestinal muscular coats may occur in the absence of peritonitis—viz., by the direct action of bacterial toxins on the muscular tissue of the intestine.

Unfortunately, the presence of visible intestinal peristalsis or of rigidity and stiffening of certain loops of intestine after tapping the abdomen is particularly likely to fail in cases in which the differential diagnosis between acute occlusion of the bowel and peritonitis is most difficult. This holds true in acute axial rotation and internal hernia-

form strangulation of the bowel, where these symptoms are of no diagnostic value, as they may be completely absent both in these lesions and in peritonitis.

Finally, the etiology of the disease must always be carefully studied in the differential diagnosis between peritonitis and intestinal occlusion. In some cases, of course, no primary cause for peritonitis can be discovered; in others, again, acute ulceration of the stomach or in the intestine may make the diagnosis of peritonitis very probable.

From all this it will be seen that in the present state of our knowledge it must often be quite impossible to decide in any given case whether there is peritonitis or occlusion of the intestine.

THE DIAGNOSIS OF THE SEAT OF THE STENOSIS AND THE OCCLUSION.

The localization of the exact seat of the stenosis or the occlusion is of interest, not only from a theoretic point of view, but also from a practical standpoint. Within recent years the surgery of the intestine has developed so rapidly and has attained such a high degree of perfection that before undertaking surgical interference for the relief of stenosis or occlusion it is highly important to know the exact situation of the lesion. In many cases a positive diagnosis of the seat of the lesion can be made without difficulty; in other cases it is quite impossible to make out exactly where the bowel lumen is stenosed or occluded, while in other cases the most that can be done is to make a doubtful diagnosis. The widest personal experience, the greatest care in examining the patient, and the possession of the greatest diagnostic acumen will often all fail, for the difficulties may occasionally be simply insurmountable.

Generally speaking, localization of the stenosis or occlusion of the bowel is more certain and easier in cases of chronic stenosis. This is self-evident, and it is not surprising that the diagnosis is made correctly in a larger proportion of these cases than in acute occlusion of the bowel in patients whose intestinal tract had been perfectly normal up to the onset of the first symptoms.

The chief problem at issue is always the following : *Is the lesion in the small or in the large intestine ?* Where it appears that the large intestine is involved, it remains to be decided, if possible, in what part of the large intestine the lesion is situated. This question must be approached from several points of view, and a great variety of factors must be studied before a decision can be arrived at. The consideration of this subject will be more interesting and valuable if the diagnostic significance of these various factors is dealt with seriatim. This plan is preferable to giving a general collective description of the diagnosis. If the medical man is familiar with the significance or the lack of value of the individual symptoms bearing on the diagnosis of the site of the lesion, he will necessarily be able to apply this knowledge in making a diagnosis in each special case.

The *physical signs obtained by examination of the abdomen* are probably the most important, and may be considered first. They are the following :

Abdominal distention and changes in the shape of the abdomen, caused by the accumulation of fluids, chiefly gaseous, in certain loops of intestine.

When changes in the size and the contour of the abdomen are absent, it is evident that the diagnosis cannot be advanced by these signs. Occasionally, however, the complete absence of any distention or alteration in the shape of the abdomen may be of use in arriving at a diagnosis of stenosis of the duodenum or of the upper part of the jejunum, for in these two conditions the meteoristic distention of the intestine is rare, whereas dilatation of the stomach is quite common. On the other hand, the presence of excessive abdominal tympanitic distention, especially when it is uniform throughout the abdomen, is of no value as regards the exact seat of the lesion in the bowel ; in fact, as it makes the examination of the abdomen very difficult and occasionally quite impossible, it interferes with rather than helps in the diagnosis.

More or less local meteorism is of special importance and value. It is almost impossible to give a verbal description of these forms of meteorism, as this gives only an imperfect and incomplete impression of the true condition of affairs. A few pictures of this condition, drawn from nature, are therefore appended (see Plates IV.–XX.). The anatomic conditions existing in each of the illustrative cases are given in the legend appended to the plates. It seems quite unnecessary to give a more detailed description of the conditions shown. I will, therefore, confine my remarks to a few general axioms based on the study of a large number of observations.

In general, the position and arrangement of the distended portions of the intestine correspond to their normal position. It is true that there are frequent exceptions to this rule, but this does not invalidate the general significance of this rule. It is much more important to study the position and the arrangement of the distended loops of intestine than their circumference and their size. Distended loops of the small intestine are usually somewhat smaller and less voluminous than distended loops of the large intestine, but these differences in size are only relative and cannot be absolutely relied on in the diagnosis, since the small intestine is sometimes distended to almost astonishing degrees. Leichtenstern points out that in stenosis of the colon the distended loops may occasionally move to and fro as a whole, and refers this to the fact that this part of the intestine has a longer mesocolon than others.

In stenosis of the sigmoid flexure complicated by meteorism from stasis the upper and lateral aspects of the abdomen are usually distended. The distention of the lateral aspects of the abdomen is generally called flank meteorism (Plates VI., X., XVII.). When the distention of the bowel is limited to certain sections of the colon, flank meteorism may be unilateral (see Plates VII., XIV., XV., XX.). This may occur even when the stenosis is in the sigmoid flexure, for only a small segment of the bowel above the stricture may be distended. Unilateral

meteorism may, of course, also occur when the stenosis is higher up in
the colon. In other instances the transverse colon alone is distended;
for example, when the stenosis is in the splenic flexure (see Plates VIII.,
IX.); it is only in exceptional cases of stenosis of the colon that the
flank meteorism is so slight as in Plate XIX. In this plate the normal
concave indentation of the flanks is absent in the front view, but it can
hardly be said that the flanks protrude so as to form a convex line.

In judging of the clinical significance of flank meteorism and of dis-
tention of the colon in general, a number of different points must be
carefully weighed and considered. In very corpulent individuals, for
instance, the amount of subcutaneous fat may be so excessive that
meteorism in the flanks, even if it exists, may be obscured. This point
must, of course, be carefully considered in any case of suspected occlu-
sion of the bowel in an obese subject. Another factor that must be
remembered is that in many individuals congenital or acquired anoma-
lies in the position of the colon exist. In cases of this kind the devel-
opment of meteorism in displaced segments of the large intestine may
produce very peculiar and puzzling conditions.

Another fact of great importance deserves special mention. It occa-
sionally happens that the flanks are distended and the upper portion of
the abdomen is prominent, thus imitating obstruction in the sigmoid
flexure. Subsequent examination shows that in spite of these signs
there is no distention of the colon, but only local meteorism of the
small intestine. I remember, for instance, a case in which there was
a characteristic protrusion of the left flank, great protrusion of the
whole upper part of the abdomen, slight protrusion of the right flank
and of the lower part of the abdomen; the whole protruding area gave
a very deep, meteoristic note on percussion, while below the level of
the umbilicus the note was high, clear, and tympanitic; no trace of
peristaltic movements; there was evidence of ascites on change of posi-
tion. This case simulated stenosis of the colon in the sigmoid region,
but at the autopsy the colon was empty and collapsed. The only dis-
tended bowel was a portion of the jejunum, the distention beginning 6
to 7 cm. below the duodenum, which was due to axial rotation of
the mesentery. Above and below this point the small intestine was
collapsed, and in addition there was secondary purulent peritonitis. It
will be shown in a subsequent paragraph that even in cases of this kind
it is possible to arrive at positive decisions as regards the true seat of
the lesion. The factors which determine this diagnosis will be consid-
ered below.

I have already mentioned the fact that anomalies of position occur-
ring in the abdominal viscera may make the recognition of the true
state of affairs exceedingly difficult in some cases. This does not
apply only to congenital and acquired anomalies in the position of the
abdominal organs, but applies with equal force to displacements brought
about by meteoristic distention of organs which were originally situated
in their normal position. This variety of displacement is seen particu-
larly in certain forms of local meteorism. In all diseases of the intes-

tine, therefore, accompanied by local meteorism the most bizarre varieties of anatomic displacement of the abdominal viscera may be seen. The case, for instance, just quoted shows that bulging of the flanks may occur in local meteorism of the jejunum. When the large intestine is affected, it is usually very important to determine the position of the sigmoid flexure. Plates VII. and XIX. show how greatly the sigmoid loop may become distended even in simple cases of ordinary meteorism from stasis, and that in this condition the apex of the sigmoid flexure may extend into the right hypochondriac region. The distention of the sigmoid flexure becomes even more extreme in volvulus of this portion of the bowel. In a preceding section attention was called to the fact that in this lesion the two branches of the sigmoid flexure are in close proximity to each other, so that the two portions of the bowel appear as a single tumor on inspection, palpation, and on percussion and auscultation combined. A tumor of this kind formed by volvulus of the sigmoid flexure and approximation of its two branches usually occupies the left lower portion of the abdomen at first, but may eventually become so large as to fill the whole abdominal cavity.

On comparing the results of occlusion of the large intestine and *the small intestine*, it will be found that there is a great difference on inspection. Plates IV., V., XI., XII., XIII., and XIV. illustrate a few very striking peculiarities; in all these cases the distended loops of intestine are seen in the center of the abdomen; flank meteorism is completely absent, and appears only in cases where the colon is also distended (Plates X., XIV., XX.); the different loops of intestine form a tangled mass of convolutions and are arranged in a similar manner as the loops of the small intestine under normal conditions; lastly, the distended loops of small intestine are always smaller than the loops of distended large intestine; it is only in exceptional cases that the inflated loops of small intestine are approximately as large as those of the large intestine (Plate XII.).

Plates V. and XVI. show a very interesting result of stenosis of the small intestine which occasionally occurs and may be best described as an *organ-pipe arrangement* of the distended loops of intestine. So far I have seen it only in cases where the distended loops were fixed and anchored by numerous old peritoneal adhesions.

It is comparatively easy to distinguish distention of the large from distention of the small intestine in chronic stenosis of the bowel. In acute occlusion of the intestine, however, in which enormous degrees of local meteorism come on very rapidly, the differential diagnosis is frequently quite impossible. In cases of this kind it often happens that the various loops of intestine, after becoming fully inflated and distended with gas, are so placed that it is quite impossible to recognize them from their anatomic position. In cases of this character the intestinal loops, whether they belong to the large or the small intestine, are distributed irregularly throughout the abdominal cavity and, provided they are not anchored or fixed by preëxisting adhesions, in obedience to a universal physical law, rise upward.

It need hardly be mentioned that occasionally other visible and palpable abnormal constituents of the abdomen, especially tumors, may be of use in making a diagnosis of the site of stenosis or occlusion; particularly when these tumors are in some way (either as cause or effect) related to the lesion to be localized.

It is comparatively rare that *percussion and auscultation* of the abdomen in these cases give any information of value, but occasionally the diagnosis is helped by these methods of examination. Meteoristic loops of intestine give the characteristic meteoristic note on percussion —that is, a very low and loud note, which is not tympanitic. Occasionally this meteoristic note has a metallic ring which can he brought out by simple percussion, provided it is not too forcible. This metallic note can almost always be obtained when auscultation and gentle percussion are performed at the same time. The metallic note, however, is only occasionally of value in the localization of the obstruction; namely, when it is constantly present in the same circumscribed part of the abdomen. Its significance is as follows: Since local meteorism, just like meteorism from stasis, is most marked in the immediate neighborhood of the obstruction, it is clear that the obstruction to the passage of the contents of the bowel must be situated somewhere near the circumscribed area in which the metallic and meteoristic note is constantly heard. This sign, however, should be made use of only with the greatest care and caution. Even when the note is constantly heard in one part of the abdomen there is evidence as to the part of the intestine which is involved in the process, or, in other words, whether the obstruction is situated in the small or the large intestine. Meteorism, when not strictly localized, but diffuse and giving rise to a metallic meteoristic note all over the abdomen, is, of course, quite valueless in the diagnosis of the site of the obstruction.

I wish to call attention to another sign obtained by percussion which, so far as I know, has never been mentioned before, and sometimes gives valuable information as regards the localization of the stenosis. The sign is the following: In a normal subject the percussion-note in the upper lumbar region behind is more or less dull, high, and flat; in stenosis of the large intestine the percussion-note here is frequently loud and deep; when the stenosis is situated in the sigmoid flexure or the descending colon, this loud and deep percussion-note is elicited in the upper lumbar region on both sides, and when the stenosis is localized in the splenic flexure or in the transverse colon, only in the upper lumbar region of the right side. Occasionally a study of this peculiar change in the normal percussion-note may indicate the correct diagnosis. In the case described above (on pages 606 and 607) the configuration of the abdomen led me to suspect that the obstruction was low down in the colon, but a loud percussion-note could not be obtained in the posterior lumbodorsal region, and, as a matter of fact, the condition was axial rotation of the jejunum. Another case presented the typical picture of vigorous peristalsis of the small intestine, while no peristalsis of the large intestine was visible. Examination of the two

PLATE IV.
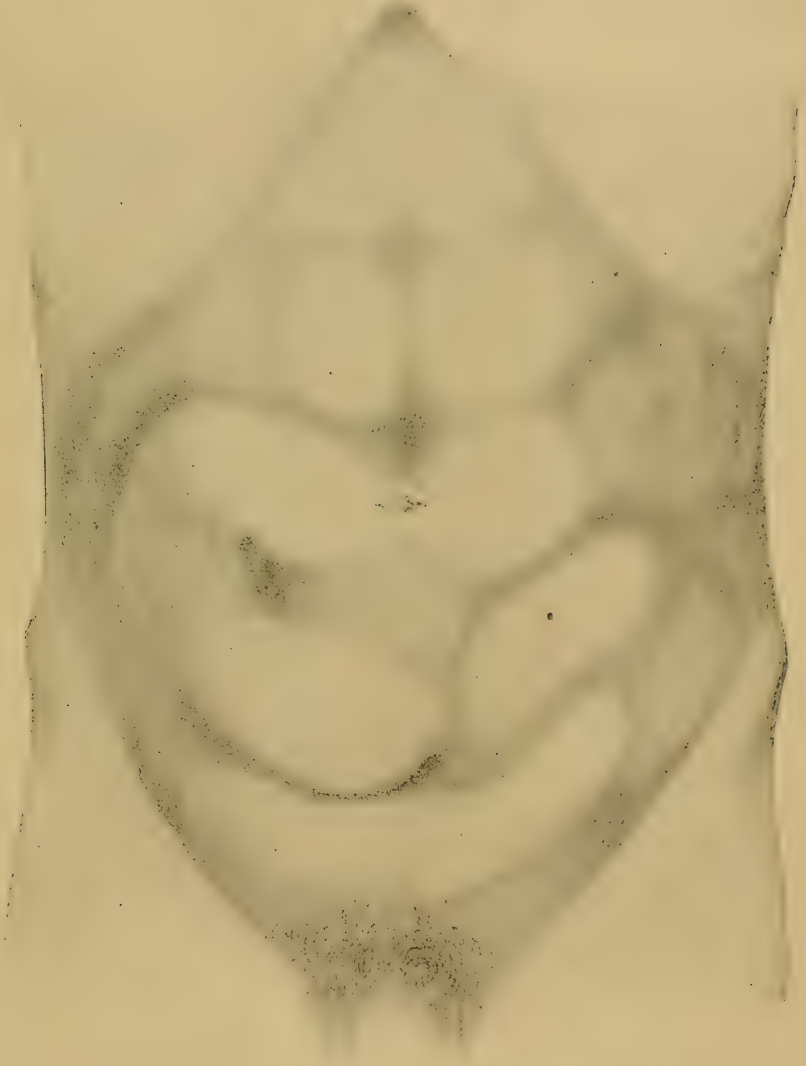
STENOSIS OF THE LOWER ILEUM IN CONSEQUENCE OF PERITONEAL ADHESIONS.

PLATE V.
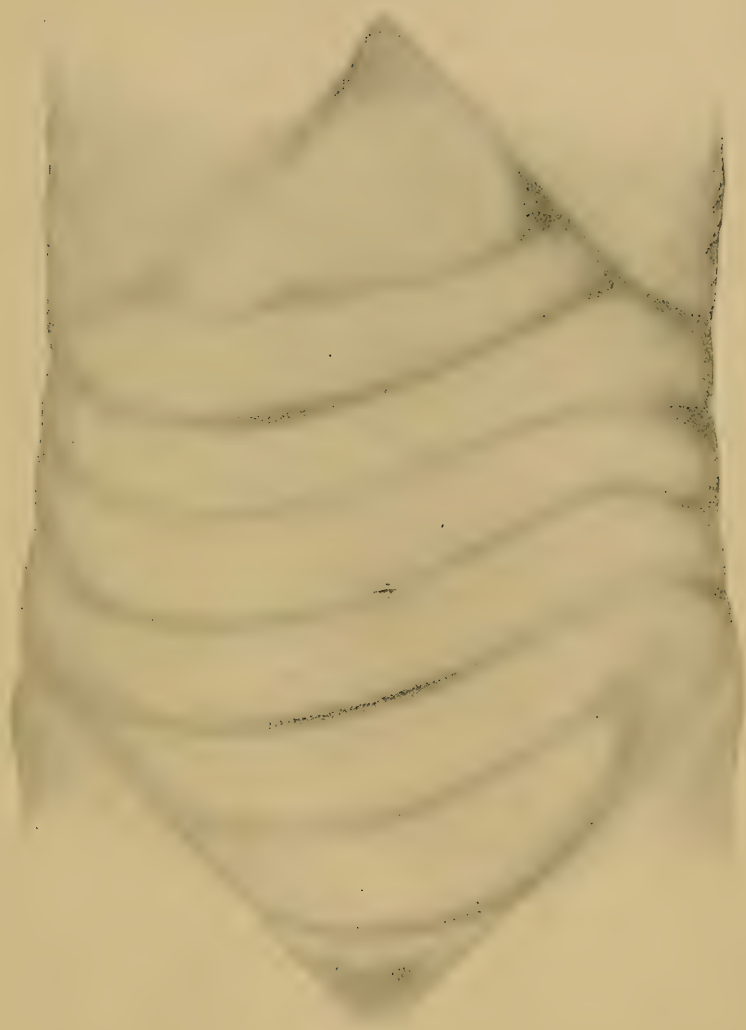
CHRONIC PERITONITIS WITH PERISTALSIS OF THE SMALL INTESTINE.

PLATE VI.
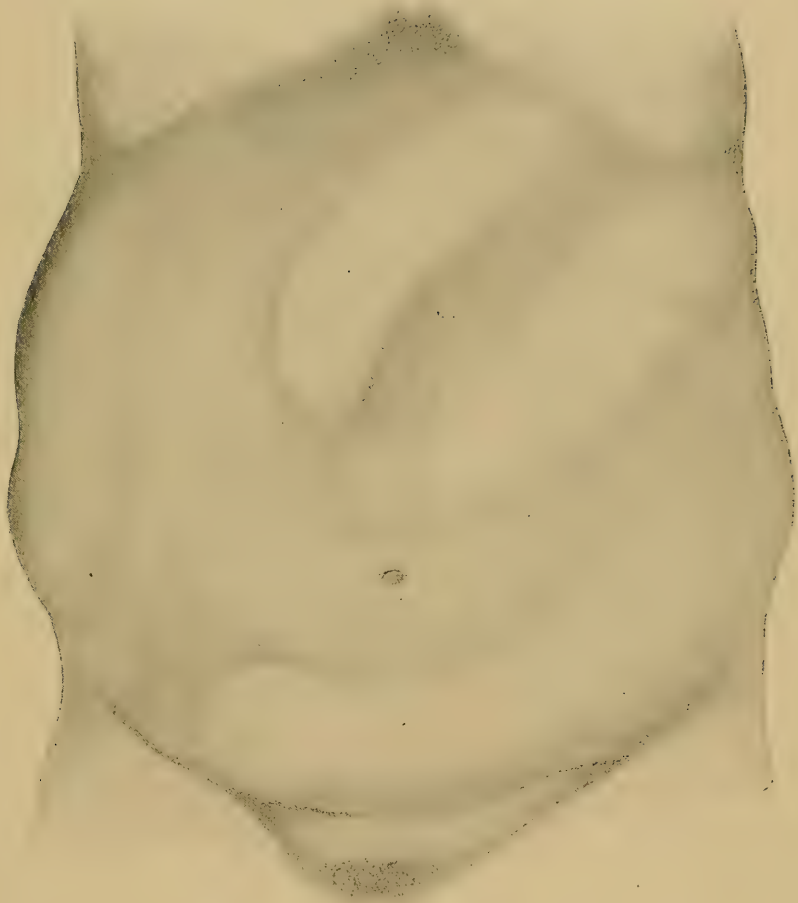
STENOSIS OF THE SIGMOID FLEXURE IN THE REGION OF THE LEFT
ILEOPECTINEAL LINE.

PLATE VII.

STENOSIS FROM CARCINOMA OF THE LOWER PART OF THE SIGMOID FLEXURE.

PLATE VIII.
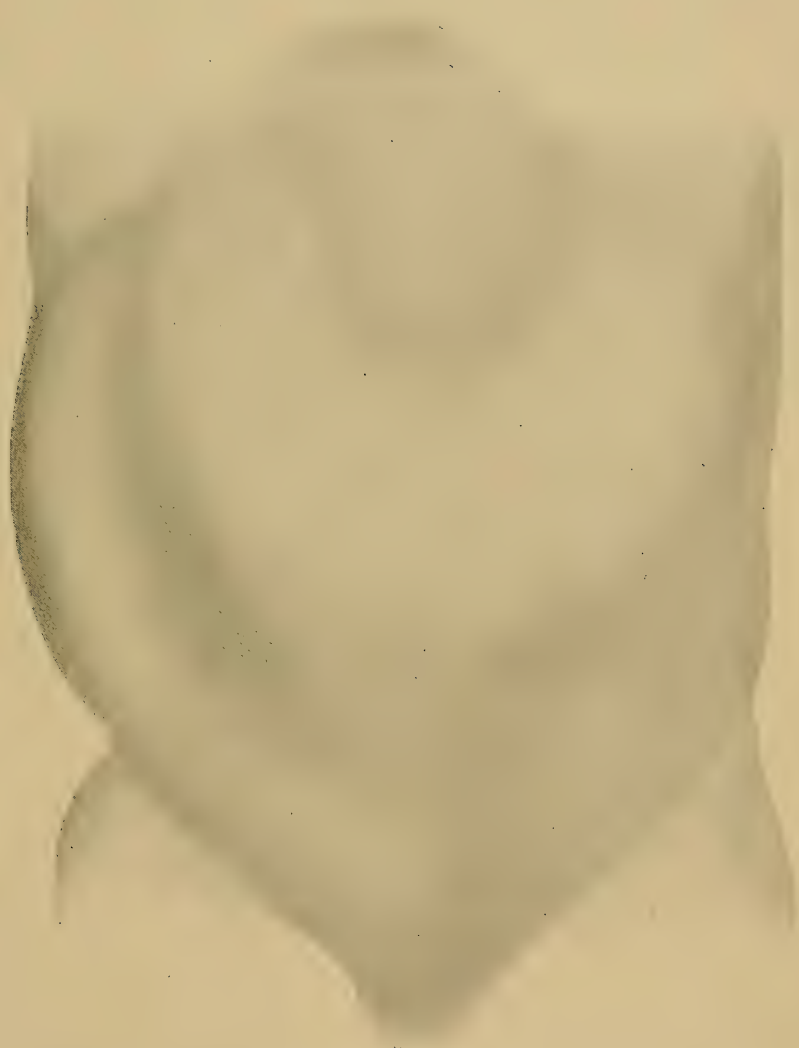
CICATRICIAL STRICTURE DUE TO DYSENTERY IN THE REGION OF THE SPLENIC
FLEXURE OF THE COLON (FRONT VIEW).

PLATE IX.

The Same Case as Plate VIII. in Three-Quarter Profile.

PLATE X.
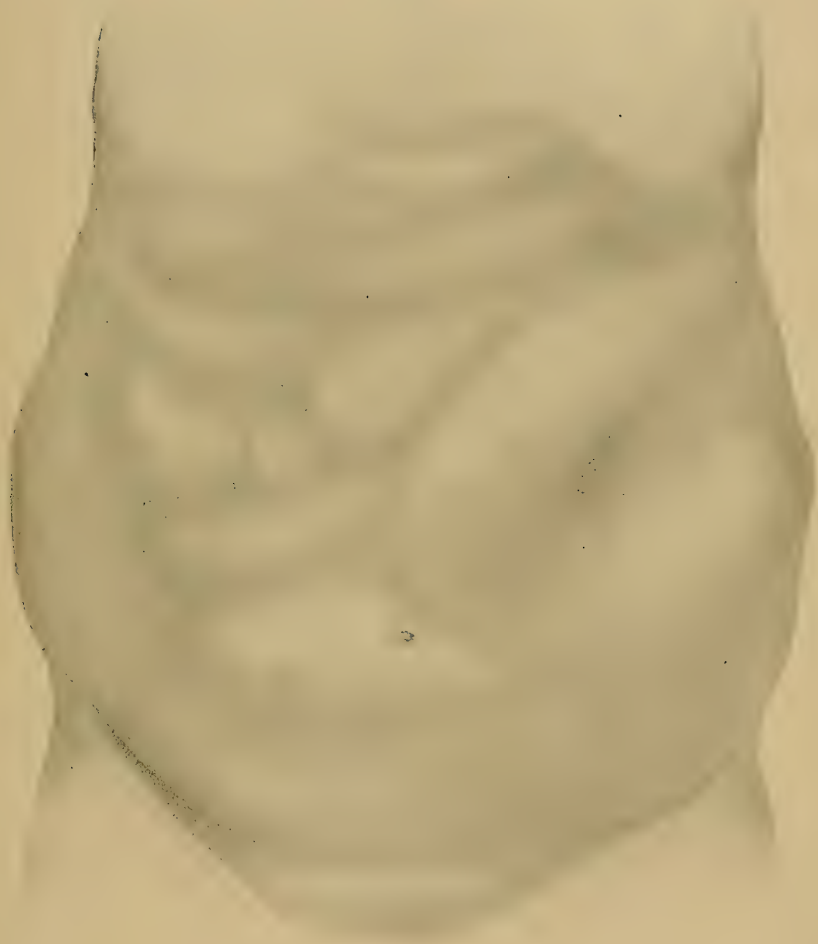
Stenosis of the Sigmoid Flexure with Peristalsis of the Large and
Small Intestine.

PLATE XI.

INTESTINAL STENOSIS FROM UNKNOWN CAUSE (CURED), SITUATED PROBABLY
IN THE NEIGHBORHOOD OF THE CECUM. (VIGOROUS PERISTALSIS OF SMALL
INTESTINE. FECAL VOMITING.)

PLATE XII.
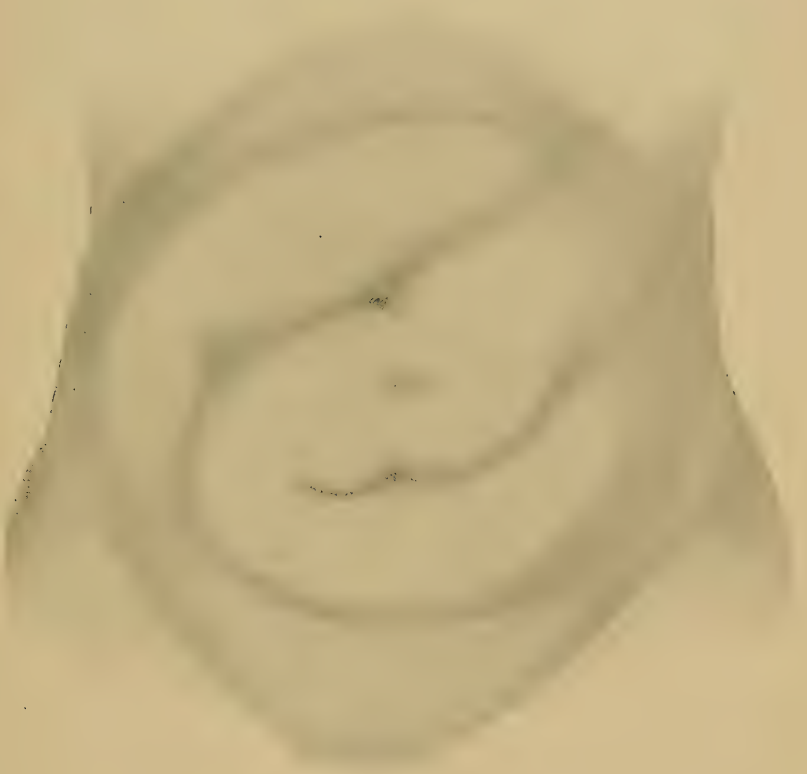
Stenosis of Unknown Origin in the Small Intestine (Discharged Uncured), with Marked Inflation from Knotting of the Intestine.

PLATE XIII.

VIGOROUS PERISTALSIS OF THE SMALL INTESTINE. ILEUS-LIKE APPEARANCE.
KOPROSTASIS. CURED BY MASS-IRRIGATION.

PLATE XIV.

CARCINOMA OF THE HEPATIC FLEXURE OF THE COLON. ILEUS. PERISTALSIS IN THE ASCENDING COLON AND SMALL INTESTINE.

PLATE XV.

STENOSIS OF THE ASCENDING COLON IN THE REGION OF THE
HEPATIC FLEXURE.

PLATE XVI.
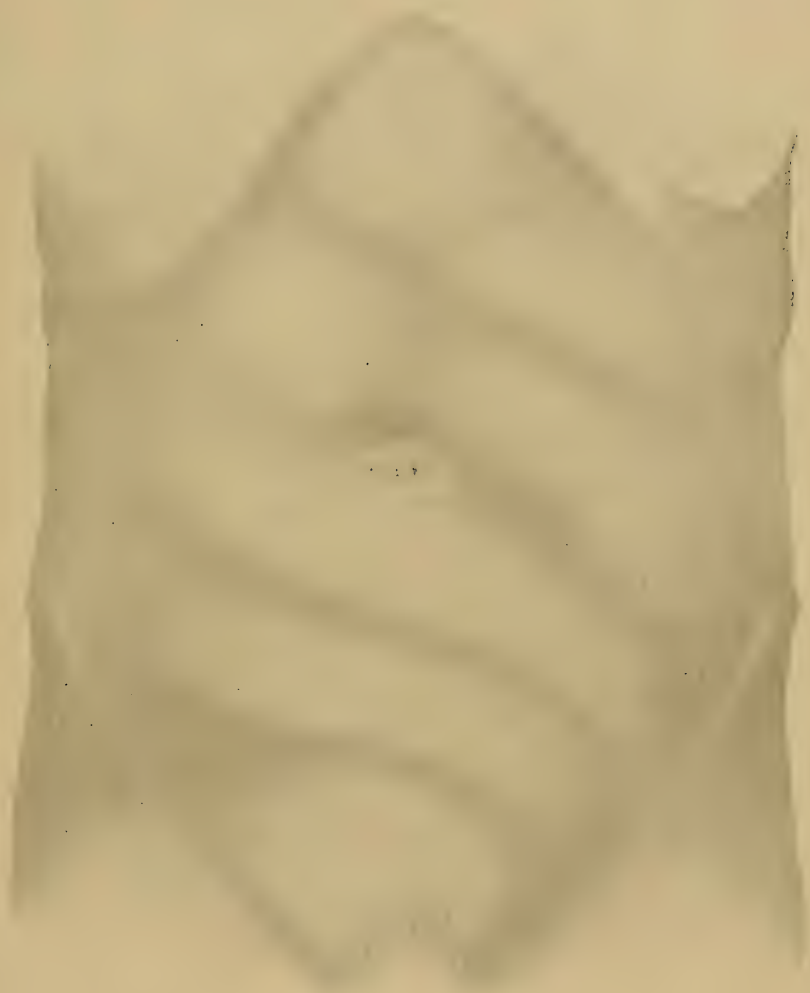
Stenosis of the Lower Ileum (Following Peritonitic Adhesions).

PLATE XVII.
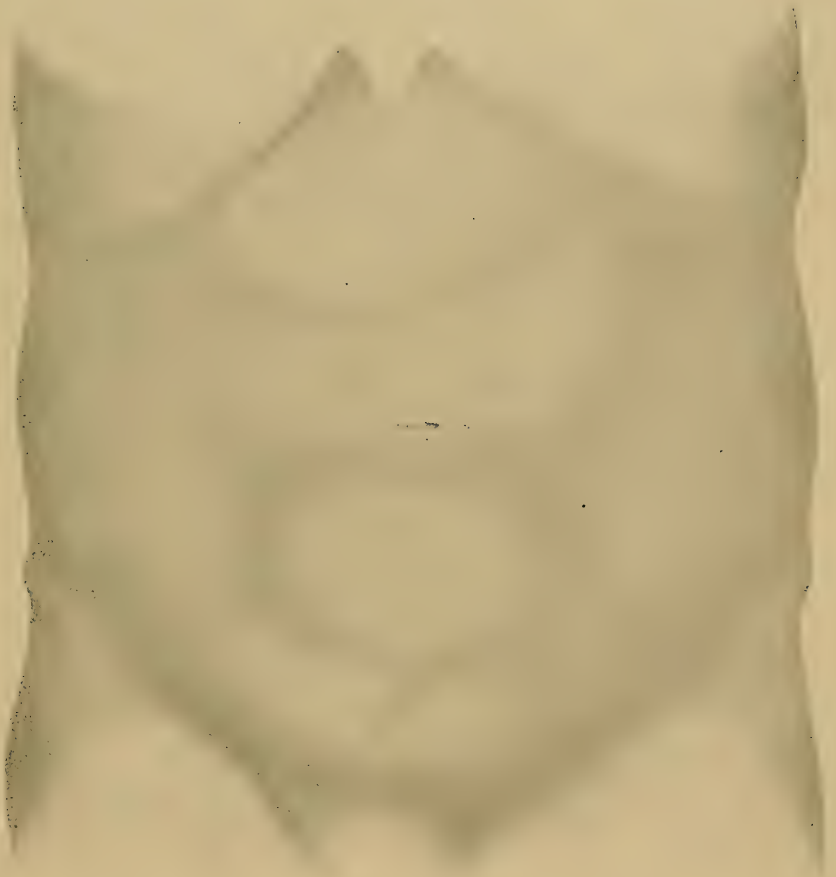
STENOSIS OF THE SIGMOID FLEXURE (FOLLOWING DYSENTERY).

PLATE XVIII.
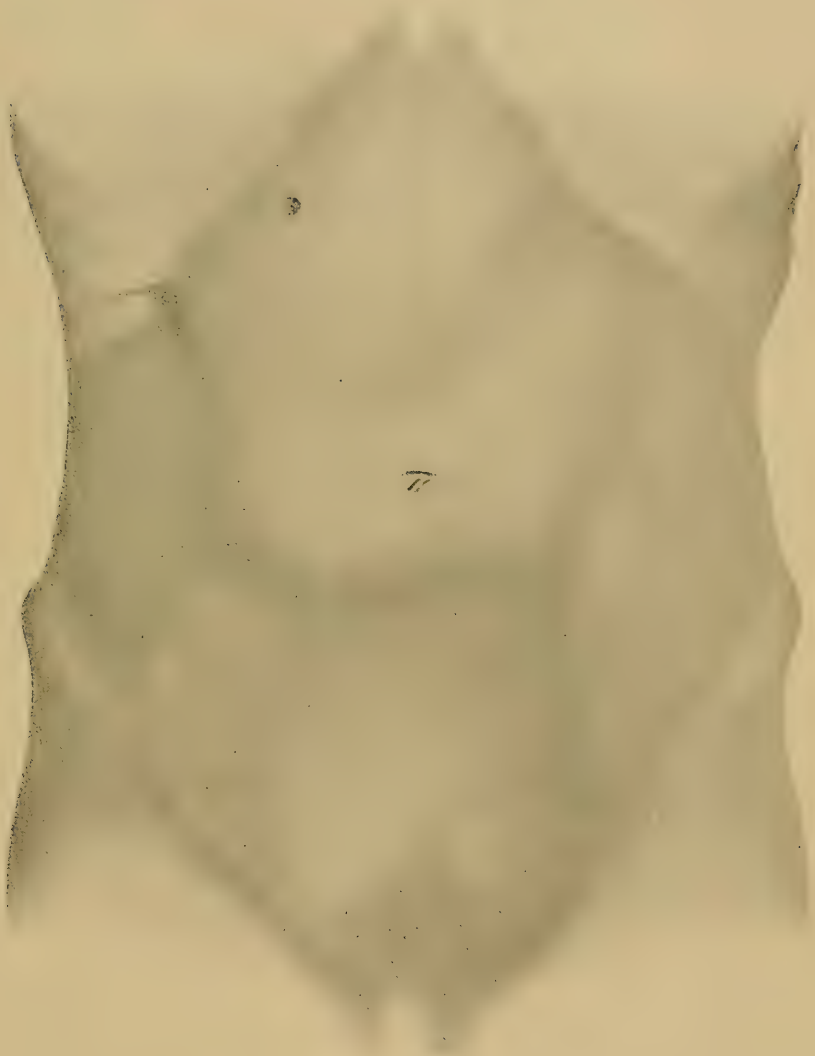
CHRONIC ILEOCECAL INVAGINATION.

PLATE XIX.
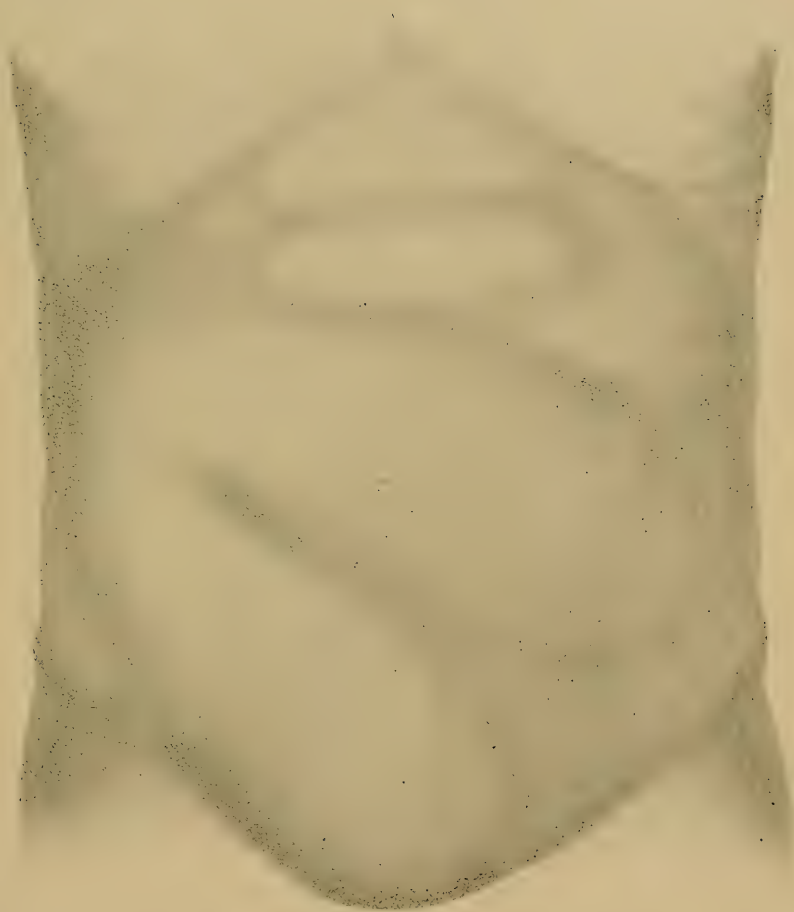
Stenosis from Cicatrix in the Lower Part of the Sigmoid Flexure.

PLATE XX.

Stenosis (Cicatrices following Tuberculous Ulcer) in the Portion of
the Ascending Colon Opposite the Hepatic Flexure (Peristalsis
in the Ascending Colon and Small Intestine).

flanks gave negative results; they were certainly not concave, and could hardly be said to be markedly convex; in this case a deep and loud percussion-note, however, was heard in both lumbodorsal regions, and laparotomy showed that the obstruction was due to kinking of the sigmoid flexure, the whole of the large intestine and a large portion of the small intestine being enormously distended.

Occasionally the discovery of visible peristalsis and a paroxysmal stiffening and rigidity of certain loops of intestine may enable a correct decision as regards the seat of the obstruction to be made. Study of the plates I have appended shows at once that in nearly all the cases the position, the arrangement, and the form of the loops of intestine that are in a condition of peristaltic unrest or of tetanic rigidity enable us to decide with a great degree of certainty whether these pathologic movements are in the small intestine or in the large intestine. It is also worthy of note that the movements of the colon are usually slower than those of the small intestine, where they are generally very much quicker and restless. Occasionally peristaltic movements and tetanic rigidity stop with absolute regularity in some one part of the abdomen, such as the ileocecal region. When this occurs, it may be concluded that the obstruction is situated somewhere in the region of the cecum, especially when the visible peristaltic movements possess the features typical of the small intestine. In arriving at this interpretation special care must be taken to avoid the following error: In cases of occlusion of the bowel of several days' duration in which the obstruction of the intestinal lumen gradually came on in the course of progressive stenosis, we are not justified in localizing the obstruction exactly where the peristaltic movements of the bowel stop. In carcinomatous occlusion of the sigmoid flexure, for instance, stiffening and rigidity of the bowel may be seen in the first days of the disease, apparently in the descending colon; at the expiration of three to five days, however, these movements may cease and the transverse colon be completely at rest, whereas the rigidity and peristalsis are confined chiefly or entirely to the ascending colon. The explanation of this peculiar change is that those portions of the bowel situated above the obstruction eventually become fatigued and paretic from excessive work and from overdistention.

The significance of visible peristaltic movements and paroxysmal tetanic rigidity of the bowel when present is exceedingly important in the diagnosis of the site of the obstruction. Unfortunately, visible peristalsis and paroxysmal stiffening are absent, especially in those cases where any possible assistance in making the diagnosis is most wanted, as in the acute forms of occlusion of the bowels, axial rotation of the intestine, and internal herniaform strangulation of the bowel.

Second in importance to the various signs just described are *certain methods of examination* which can be employed in making a diagnosis of the site of occlusion of the bowel. Although the value of some of these methods of examination was formerly greatly exaggerated, at any rate to some extent, some of them may be of use in individual cases.

Rectal irrigation and enemata were formerly commonly employed in

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order to localize the seat of the obstruction. Brinton in particular has outlined the indications for this method of diagnosis with great care, and has described the method of employing them in diagnosis. The idea was to estimate the extent of intestine situated below the obstruction from the quantity of fluid which the bowel would retain, but practical experience shows that the value of this method is very slight indeed. Careful consideration of the possibilities of error shows that the method is decidedly defective. The amount of fluid that the intestine can hold is dependent on a number of factors which vary very considerably in any given case, such as the length of the colon, the state of contraction of the intestine below the obstacle, and the elasticity of the bowel. The elasticity of the bowel is sometimes normal, or may be modified by morbid changes in its wall. The amount of fluid which can be introduced into the intestine will naturally vary according to the elasticity of its wall. Another factor which may influence the amount of fluid that the intestine will contain is the degree of intra-abdominal pressure, which exerts a marked influence on the passive distensibility of the intestine. In addition it must be remembered that in stenosis of the intestine the primary cause of the stenosis may be an anatomic lesion which interferes with the passage of the bowel contents downward, but does not interfere with the passage upward of rectal enemata. Under these conditions, therefore, conclusions which are entirely erroneous might be drawn as to the seat of the stricture, since the large amount of fluid which could be introduced into the bowel would point to the stenosis being very much higher up in the bowel than it actually is. The only condition on which more or less reliable conclusions can be drawn from this method of diagnosis is when repeated enemata are given and only a minute amount of fluid can be driven into the rectum and is very quickly returned. In such cases the obstruction may be assumed with great probability to be low down in the bowel. With regard to this point it must be remembered that the rectum with its ampulla is capable of holding quite a liter and even occasionally a liter and a half of fluid.

Another manipulation which has been employed, but is just as unreliable as the preceding one, is *sounding the rectum*. This manipulation may give rise to erroneous conclusions in two ways : sounding the rectum may create the impression either that the obstruction is situated lower down or higher up than it actually is. The sound, for instance, may be caught in a fold of rectal mucous membrane or in some rectal diverticulum, or again it may be stopped by a plug of fecal matter ; in either case it would be concluded erroneously that the obstruction is situated low down ; or, conversely, the sound may bend upon itself or be curled up in the rectum, or again it may really pass up as far as the lower end of the sigmoid flexure, and if this part of the colon has a long mesocolon, the sound may be pushed upward for a considerable distance inside the sigmoid flexure, so that its point may even be felt in the umbilical region. The obstruction may thus appear to be situated above the sigmoid flexure, whereas in reality it may be very low down.

A very important means of assistance in the diagnosis of the situation of intestinal obstruction is *simple digital examination of the rectum.* This method of examination should, in fact, never be omitted in attempting to localize the seat of the obstruction.   All cicatricial strictures, neoplasms, intussusceptions, fecal tumors, and foreign bodies giving rise to obstruction, when situated low down in the rectum, and conditions which compress the rectum from without and so narrow it, can be diagnosed by a simple digital examination, which may be supplemented by the use of the rectal speculum or the rectoscope.

[Ballooning of the rectum, in which the intestine is dilated to the utmost and empty, though it may occur in other conditions, is often met with in association with stricture of the colon, especially when the descending colon or the sigmoid flexure is involved (Treves). —Ed.]

Vaginal examination may also supply information of value in the diagnosis of the seat of the obstruction.

G. Simon strongly advocates the *examination of the rectum by inserting the whole hand,* and has worked this method out very thoroughly. The majority of clinicians, however, regard it as so serious a proceeding that they hardly ever employ it now for the purpose of determining the seat of the obstruction.   A method which sometimes shows whether or not the obstruction is in the large intestine and which was warmly recommended by Treves is *auscultation of the ileocecal region while a rectal enema is being given.*   He argues that if the sounds heard indicate that the injected liquid readily passes into the right side of the abdomen, the obstruction is not in the colon and that the lumen of that part of the bowel is freely permeable.   In order to obtain good results from this method the diagnostician must have some experience and some practice in order to be able to interpret correctly the sounds.   It is frequently very difficult to differentiate the sounds produced by the passage of water through the ileocecal region from spontaneous borborygmi. Another point to remember is that in cases complicated by meteorism the sounds are frequently heard over wide areas of the abdomen and are often transmitted for long distances ; thus they may be heard on the right side even when they are produced on the left side.

[In the later edition of his work on intestinal obstruction, 1899, Treves[1] says : " I made a somewhat extensive inquiry into this method of diagnosis, and carried out experiments upon the dead body, and made numerous investigations in cases of actual obstruction.   At first I was disposed to think the measure of service in determining the seat of the obstruction, but further experience convinced me that auscultation of the colon in cases of intestinal obstruction was absolutely valueless as a means of diagnosis."—Ed.]

The study of the **functional intestinal symptoms** forms a third method of analyzing these cases and frequently gives valuable information.

Pain is quite unreliable as a guide in the diagnosis of the seat of

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[1] F. Treves, *Intestinal Obstruction,* second ed., 1899.

the lesion. In the first place, it must be remembered that pain is often entirely absent; in other cases it is too general and widely spread over the abdomen to be of any use in localizing the lesion; in other cases it is manifestly produced by conditions other than occlusion of the bowel *per se*—viz., tonic contraction of the bowel, with colicky pain and severe meteoristic distention of the bowel—which produce such widely distributed pain, or pain in such distant parts that no information is obtained as to the seat of the obstruction. Pain is only of use in the diagnosis of the seat of the obstruction when it is strictly localized and constant in one region of the abdomen. But even then great care is necessary not only in acute, but also in chronic, cases of intestinal obstruction. In discussing individual cases above I have repeatedly called attention to the fact that the pain of chronic peritonitis complicating obstruction may occasionally be felt in the left side of the abdomen, whereas the primary seat of the disease may be in the ileocecal region. In order to illustrate how very misleading the situation of the pain may be in cases of acute occlusion of the bowel, the following example may be mentioned: A patient with acute intestinal obstruction of several days' duration was to undergo an operation for the relief of his symptoms. There was absolutely nothing to show the seat of the obstruction. The abdomen was distended by enormous meteorism, and palpation was quite impossible, but the patient complained of constant and very violent pain in a strictly circumscribed area situated a little to the right of the umbilicus (in addition to this pain there was the diffuse pain produced by the abdominal tension and meteorism). An incision was made in the linea alba, and on examining the point of the localized pain, the surgeon discovered and divided a peritoneal band. The symptoms of occlusion, however, persisted, and on the following day the abdominal cavity was opened a second time and a volvulus of the sigmoid flexure was found. The patient succumbed ten hours after the operation. Experiences of this kind naturally inculcate extreme caution in basing a diagnosis on the situation of even distinctly localized pain. I believe that circumscribed pain in this disease is only of localizing value in diagnosis when it is not only spontaneously felt by the patient, but in which it can always be elicited by pressure in the same part of the abdomen.

The majority of clinicians state that, in acute occlusion of the small intestine, pain appears earlier in the disease, is more violent and more persistent than in occlusion of the large intestine, and, generally speaking, this is correct. In individual cases, however, so many exceptions to this rule occur that its diagnostic significance is comparatively small. For instance, volvulus of the sigmoid flexure, as we know, is always accompanied with very violent pain early in the disease, and is a lesion of the large intestine in which this rule would not apply. Apart from all this, an estimation of the greater or less relative severity of pain is a very uncertain matter and depends entirely on individual judgment. Attention should be called here to a fact already mentioned in a preceding section—viz., that very violent pain, localized strictly in the

dorsal region, is believed to be specially frequent in cases of axial rotation of the small intestine.

Although *vomiting* is of no positive value in the diagnosis, it may occasionally be of some use in deciding the seat of the lesion. As a rule, it can hardly be said universally, vomiting is one of the symptoms of occlusion of the small intestine, particularly of the acute forms; and is more constant, persistent, and severe than in occlusion of the large intestine. This point is best illustrated by the symptom-complex presented by volvulus of the sigmoid flexure, on the one hand, and of internal hernia of the ileum or the jejunum, on the other hand. The chief reason why vomiting is more frequent, constant, persistent, and severe in lesions of the small intestine than in lesions of the large intestine is that the former part of the bowel possesses many more nerve-fibers and a much more irritable nervous apparatus than the large intestine.

The old view that fecal vomiting is pathognomonic of obstruction in the large intestine is, as has repeatedly been emphasized in preceding sections, quite erroneous. It has been seen that fecal vomiting may occur in any form of obstruction of the intestine, and wherever the obstruction is situated, even in occlusion of the jejunum high up; in fact, the rule may be reversed to the effect that experience shows that feculent vomiting is more frequently observed and occurs earlier in the course of the disease in cases of acute occlusion of the small intestine (internal strangulation) than in occlusion of the large intestine (volvulus of the sigmoid flexure).

Very little can be learned as regards the situation of the obstruction from *examination of the stools*. When a patient with symptoms of obstruction of the bowel has marked tenesmus, the seat of the obstruction should always be looked for in the rectum or the lower part of the colon. Blood in the stools should suggest that the lesion is situated low down and below the cecum; this occurrence, in addition, allows certain conclusions to be drawn as to the anatomic character of the process, and from this indirectly as to the probable site of the lesion. When the feces have the character of stenosis feces, which have been described above, and the general conditions found make it probable that this peculiar formation of the fecal cylinder is due to narrowing of the intestinal lumen, the stenosis should be looked for in the rectum, the sigmoid flexure, or the lower portions of the colon.

Extensive tenesmus indicates that the lesion is in the rectum or lower colon; but caution must be exercised here too, as it has been noted, even though very rarely, in ileac invagination.

Another functional sign which may be made use of in the diagnosis of occlusion of the bowel is *indicanuria*. Before utilizing this symptom in localizing the position of the lesion it must always be remembered that indicanuria, however marked it may be, is quite valueless as a basis for any conclusions as to an intestinal lesion in the presence of acute peritonitis, or even when there is any suspicion of the presence of acute peritonitis. The same applies to those cases in which occlusion of the

bowel is not complete and in which there is a combination of the symptoms of stenosis and of diarrhea.

The fact that occlusion of the small intestine leads to a great excretion of indican in the urine was first established by Jaffé. This statement is perfectly correct, for in my experience indicanuria is never absent in cases where there is a stricture of the intestine above the ileocecal valve. The observations of others have been to the same effect, and it safe to assert that if indicanuria is absent, the obstruction is not situated in the small intestine.

Jaffé, in addition, remarks that in occlusion of the small intestine indicanuria is not only pronounced, but also appears rapidly after the onset of the first symptoms of occlusion—that is, on the second or third day. At this time, he believes, the amount of indican excreted is quite considerable, and on this point again my experience fully tallies with his. The two following cases illustrate the validity of this statement. In one patient a mesenteric cord had compressed the ileum above the cecum—diagnosis being made at the autopsy. On the fourth day of the disease (when the patient was admitted to the hospital) there was very marked indicanuria. In another patient a tightly stretched cord of connective tissue, situated in the vicinity of the left inguinal canal, tightly compressed a loop of the small intestine (operated in Billroth's clinic; recovery). In this case indicanuria was noticed on the second day of the disease. These facts as to the excretion of indican, therefore, are of special value in the diagnosis of acute occlusion of the intestine. Our knowledge of this subject may be summed up as follows:

If, in an individual who was perfectly healthy until the onset of symptoms of acute occlusion of the bowel (without symptoms of peritonitis), a considerable degree of indicanuria is present on the second or third day after the onset of the disease, the diagnosis points to the seat of the obstruction being in the small intestine.

Conversely, in cases with symptoms of complete occlusion of the intestine, in which, on the fourth or fifth day, or even later after the onset of the disease, no appreciable increase in the excretion of indican can be determined, the seat of the lesion must be looked for in the large intestine. Jaffé, however, has correctly called attention to a fact which I have also often verified—viz., that in cases of stenosis of the colon running a chronic course indicanuria may also occasionally appear. In these cases the increased excretion of indican is due to stagnation of fecal material in the small intestine. A summary of our knowledge as to the value of indicanuria in the local diagnosis of occlusion of the bowel reads as follows:

After excluding all sources of error,—peritonitis, old diseases of the intestine,—the presence of indicanuria during the first days of the disease indicates that the occlusion is situated in the small intestine, and the absence of indicanuria at this time that the occlusion is in the large intestine. If the disease is chronic and runs a more protracted course, the absence of indicanuria also indicates that the lesion is situ-

ated in the large intestine, but the presence of indicanuria under these conditions does not justify any decision as to the seat of the obstruction.

The excretion of the conjugate sulphates in the urine need not be considered here, as, for reasons given in another section, it is irrelevant to the question under discussion.

Finally, a few general points may be briefly mentioned which may occasionally be of use in determining the seat of the occlusion.

In all cases in which the symptoms of acute occlusion of the bowel come on with very violent pain and vomiting, and in which the course of the disease is very severe and a marked degree of collapse rapidly develops, it is usually assumed that the seat of the occlusion is in the small intestine; whereas, on the other hand, in cases where the intensity of the general symptoms and the whole course of the disease are less violent, it is assumed that the occlusion is probably in the large intestine. This diagnostic formula applies to many, but not by any means to all, cases; it can hardly be said, therefore, that this means of distinction possesses the dignity of a general rule of diagnosis. Treves has called attention to the fact that in many cases the symptoms produced by the different kinds of occlusion (acute or chronic) have been confounded with the symptoms produced by obstruction of different parts of the intestine; in other words, the differential diagnosis has been made more from the character of the obstruction than from the point of view of its situation. This confusion can be readily understood, for in those cases of occlusion of the bowel which run a very acute course the obstruction is usually in the small intestine, whereas in cases running a more chronic course, the occlusion is more often in the large intestine.

The rapidity of the course, and in part the gravity of the general symptoms, are, it is true, influenced to a certain degree by variations in the seat of the occlusion in the bowel. This, however, is true only to a certain degree, and applies only to acute occlusion. In discussing the general symptomatology of occlusion of the bowel, I have already called attention to the limitations of this statement. The chief reason why occlusion of the small intestine produces more serious and more violent symptoms is the fact that this part of the bowel contains a more abundant and a more sensitive nervous supply, so that the reflex symptoms in general (collapse) are more pronounced than in corresponding lesions of the large intestine. In addition to the direct involvement of the intestine, however, the secondary involvement of the peritoneum and the mesentery must always be considered. When these two structures are involved to any great extent in the morbid process,—in other words, when the primary lesion, in addition to producing occlusion of the bowel, causes wide-spread and severe strangulation,—the same violent course and serious general clinical picture may be presented in occlusion of the large intestine as in occlusion of the small intestine. That this can actually occur is proved by the syndrome presented in volvulus of the sigmoid flexure. Conversely,

the onset of the disease need not be so severe in all cases where the small intestine is involved. This is shown, for instance, by cases of the kind described on p. 581. These facts show that it is impossible to make general statements as regards the diagnostic significance of a violent course and of very severe general symptoms in determining the seat of the obstruction. The rule quoted above, therefore, does not hold good absolutely, and too rigid reliance on this formula may lead to very serious errors in diagnosis.

Occasionally the diagnosis as to the seat of the trouble may be almost or even quite positive when the anatomic nature of the occlusion or of the lesion that produced stenosis of the bowel is recognized. As regards this point, the history of the case may be of some use in the diagnosis of the situation of the obstruction.

After this description of the general features of the local diagnosis it would be superfluous to consider again the diagnosis of the seat of occlusion or stenosis of the small intestine, on the one hand, and of the large intestine, on the other, and unnecessary even to summarize again what has been said. A critical study of each case and consideration of the different points of view mentioned will enable the medical man to arrive at a definite decision as to the seat of the lesion in almost every case where this can be done. I shall, therefore, omit a detailed description of the differential diagnosis between lesions of the small and the large intestine capable of causing stenosis or occlusion of the bowel, and shall devote a few lines to the localization of obstruction or stenosis of the duodenum and of the rectum.

The diagnosis of stenosis of the duodenum, a lesion which is comparatively rare, has become so exact within the last decade or so that in many cases it can readily be made. In the first place, a careful study of the etiology of this disease is often the source of valuable information, for the etiologic factors of duodenal stenosis are usually special and distinct. It may be said that in no other segment of the bowel is stenosis of the lumen so often due to compression from without. This depends partly on the fixed position of the duodenum and partly on the frequency with which lesions capable of compressing the duodenum, particularly carcinoma, occur in neighboring organs. Gerhardi and Hagenbach have collected a large number of cases of stenosis of the duodenum, and have shown that diseases of the pancreas are the most prolific causes of compression from without and of stricture of this segment of the bowel. The lesion of the pancreas, again, which most frequently produces this effect, is carcinoma; in exceptional cases inflammation of the pancreas may also produce duodenal stenosis. Next in order of frequency are certain forms of constricting peritonitis due to pericholecystitis (Hochhaus and others). In the third place, enlargements of the retroperitoneal lymphatic glands are a frequent cause of obstruction. In other cases the stricture of the duodenum may be due to a new growth arising in the duodenal wall itself, or constricting cicatrices following simple duodenal ulcer. Boas reports a case of congenital stenosis of the duodenum; a few exceptional cases have been

recorded in which acute obturation of the duodenum was due to impaction of a large gall-stone. We may particularly mention acute arteriomesenteric occlusion at the duodenojejunal junction.

[Congenital stricture of the duodenum usually occurs close to the entrance of the bile-duct into the duodenum, generally above, but occasionally just below, the biliary papilla ; Cordes [1] has collected 57 cases. There may be a septum, with or without a perforation in the center, running across the bowel. The septum is composed of the muscular as well as of the mucous and submucous coats. Its position points to the conclusion that it is connected with the budding-off of the hepatic diverticulum ; stricture in this situation is, therefore, analogous to stricture of the ileum at the point where Meckel's diverticulum comes off and to congenital atresia of the esophagus at the point where the pulmonary diverticulum occurs (Shattock). [2] Complete interruption of the duodenum may be found at this spot. Congenital stricture, obliteration, or complete interruption may also occur at the juncture of the duodenum and jejunum. Ducros [3] collected 11 cases—3 of stenosis, 5 of obliteration of the lumen, and 3 of complete interruption of the intestine at this point. Reference has been made on p. 482 to stricture of the third part of the duodenum by the pressure of the superior mesenteric vessels in the mesentery, described by Albrecht [4] and Byron Robinson. [5] This condition, which is part of visceroptosis, is specially likely to occur when the patient lies in the dorsal position.—ED.]

The symptoms of duodenal stenosis may be either acute or chronic. The disease, as a rule, runs a chronic and only exceptionally an acute course. In acute occlusion of the duodenum produced by compression by the mesentery of prolapsed loops of the small intestine, or from impaction of a gall-stone, the general symptoms characteristic of acute occlusion of the bowel may appear—namely, sudden and very severe collapse, and even stupor, and profuse, non-feculent vomiting, distention of the upper abdominal region, and, especially, distinctly perceptible acute gastric distention, without distention of the intestines. These symptoms, of course, are in no way characteristic or pathognomonic of obstruction in the duodenum. In one case, however, with this collection of symptoms, Schüle made a correct diagnosis after repeatedly washing out the stomach and finding that each lavage contained large quantities of fresh bile. After the disease had persisted for four days a large gall-stone was found in the motions, and the patient made a rapid recovery.

Chronic forms of stenosis of the duodenum must be divided into two groups : in the one, the stenosis is above the entrance of the common bile- and pancreatic ducts into the duodenum ; in the other group, it is below this point. The diagnosis of the former lesion can probably never be made, because the symptoms correspond in all their

[1] L. Cordes, *Arch. Pediatrics*, 1901, vol. xviii., p. 401.
[2] S. G. Shattock, *Trans. Path. Soc.*, vol. xli., p. 93.   [3] Ducros, *Thèse de Paris*, 1895.
[4] P. A. Albrecht, *Virchow's Arch.*, vol. clvi.
[5] Byron Robinson, *The American Practitioner and News*, August 15, 1900, p. 125.

essential features with those of stenosis of the pylorus. The latter form, however, in which the stenosis is situated further down in the duodenum and below the point of entry of the common bile- and the pancreatic ducts, presents so many peculiar and characteristic features that the diagnosis can usually be made, provided the patient is examined with sufficient care. In this instance both the duodenum and the stomach are greatly dilated, a condition which can readily be discovered by the physical signs presented; in addition, however, there are other very characteristic features. Leichtenstern was the first to call attention to the occurrence of copious vomiting of bile and bilious material. Cahn showed that, on siphoning the stomach, the last portions of liquid drawn off usually consisted of an abundant quantity of almost pure bile. Other observers have corroborated these statements and have shown that the abundant and continuous entrance of bile into the stomach must be considered a characteristic, almost a pathognomonic, sign of stenosis of the duodenum below the entrance of the common bile-duct into the bowel. Boas showed, moreover, that, in addition to bile, the pancreatic juice passes into the stomach and duodenal digestion takes place within the gastric cavity, as proved by the fact that a distinct hydrolysis of fat may occur in the stomach. The stomach-contents in a case of this kind will be found to be neutral or alkaline in reaction. This is important because it shows that the condition is not dilatation of the stomach due to stenosis of the pylorus; for in this condition the stomach-contents are always acid. In addition, the typical acid products of fermentation are absent in the stomach-contents in cases of duodenal stenosis, whereas they are, as a rule, present in cases of pyloric stenosis. Riegel, even before these publications, had called attention to the chemic changes in the stomach-contents, and to the great variations which can be observed in the chemic composition and properties of the siphoned stomach-contents. He explained these differences and this irregularity by assuming that the alkaline juices of the intestine regurgitated backward into the stomach. Another important sign of duodenal stenosis (Riegel, Hochhaus) is that the stomach, even when it has been completely emptied the evening before, may be found next morning to contain large quantities of fluid material—as much as three liters. A phenomenon which is practically identical with this is that occasionally after the stomach has been completely emptied of all its fluid contents, the patient may be seized by a paroxysm of vomiting and bring up large quantities of liquid material. In cases of stenosis low down in the duodenum the motions are usually colorless and free from bile.

Only a few words need be added as to the diagnosis of stenosis of the rectum. The diagnosis of this lesion can almost invariably be made with absolute certainty, provided the decisive examination—namely, digital exploration of the rectum—is not omitted. In the paragraphs on the general clinical aspects of stenosis of the intestine, and in the section on Carcinoma of the Rectum, I have already pointed out the functional disturbances produced by these lesions, which should suggest

digital examination of the rectum. In order to avoid repetition, the reader should refer for these indications to the sections mentioned. When functional disorders of this kind exist, especially obstinate constipation (occasionally interrupted by diarrheic motions which may contain mucus, pus, or blood), tenesmus, and the development of hemorrhoids, the medical attendant should always carefully explore the rectum with the finger. He will usually find a stricture either immediately above the anus or higher up, and in many instances will be able to determine whether the stenosis is due to a cicatricial stricture, to a carcinoma, to some foreign body, or to something compressing the rectum from without—for instance, a hypertrophied prostate, swellings or tumors of the female sexual organs or of the urinary bladder, tumors of bones, or inflammatory swellings of the perirectal tissues, etc. Occasionally ocular inspection of the rectum through the rectal speculum, or occasionally the skilful manipulation of the sound by an experienced hand, may give further information as regards the particular character of the stenosis of the rectum.

THE DIAGNOSIS OF THE ANATOMIC NATURE OF STENOSIS OR OCCLUSION OF THE BOWEL.

Some authors have expressed themselves to the effect that the determination of the anatomic character of intestinal obstruction is, properly speaking, superfluous. These writers argue that the most important thing from a practical point of view is to determine the physiologic condition of the intestine—that is, to find out whether it is paralyzed or whether it is still capable of peristaltic contractions. I do not agree with this view. The motor powers of the intestine are, of course, of the greatest significance in prognosis, and it is very important to know the state of this function in treating these cases. But in order to come to any definite conclusion as regards the efficiency of the intestinal contractions, it is necessary to understand the anatomic nature of the primary process producing the occlusion; it is necessary, moreover, to know whether the stenosis of the bowel is due to some process which has been developing slowly and chronically, or whether it is merely an acute termination of a chronic process, or, finally, whether the occlusion occurred acutely in an intestine which was previously perfectly normal. Apart from all this, however, the recognition of the anatomic process responsible for the symptoms is very important from the point of view of prognosis, and in many cases directly influences the treatment for the cure or the relief of the disease.

It is unnecessary to enumerate here all the symptoms which characterize the different anatomic lesions capable of interfering with the passage of the bowel contents, as special and detailed attention was given to all these factors in the sections on these lesions. A general review of the symptoms produced by the different conditions producing stenosis shows that they resemble one another in many of their most essential features. This being the fact, it can readily be understood how exceedingly difficult—in fact, how absolutely impossible—it is in many cases

to make a diagnosis of the anatomic character of the primary morbid process that is producing stenosis of the intestine. I have often failed in attempting this diagnosis. Nevertheless, a conscientious attempt should always be made to determine the exact anatomic nature of the lesion. A brief sketch will be given here of the best method to be pursued in order to make this diagnosis.

When, in any given case, the medical attendant has reason to suspect, from the general symptoms, that the clinical picture is due to some occlusion of the intestine, the two following methods should invariably be carried out :

First, all the hernial apertures of the body accessible to palpation should be carefully examined.

Second, the rectum and the vagina should be carefully examined by the finger. The anatomic diagnosis of the lesion may frequently be made directly by one or the other of these methods of examination, or, when the diagnosis cannot be made at once, some signs, at least, are usually forthcoming to indicate the lines on which the diagnosis should be followed out ; in other words, a positive result on digital exploration or on palpation of the hernial openings will solve the diagnosis at once, whereas negative results limit the diagnostic possibilities by exclusion.

A third rule, which all physicians who have had much experience in this field of diagnosis will indorse, is the following : In any case where there is an old hernia, even though there is no evidence that it is the direct cause of the symptoms (by strangulation or inflammation), it should always be borne in mind that it may be the indirect cause of the symptoms of occlusion—viz., as the starting-point of old peritoneal adhesions which may, in many ways, affect the permeability of the bowel passage. Such adhesions may produce constriction of the bowel from without, or kinking of the bowel, or may lead to the formation of false ligaments, and so to internal herniaform strangulation of some segment of the intestine. An old hernia, therefore, must always be considered a very suspicious lesion until examination of the patient shows that the symptoms of occlusion of the bowel are, without question, due to some other cause.

If it can be shown that the hernia is not the primary cause of the trouble, and that the stenosis is not in the rectum, the patient should be submitted to the ordinary strictly methodic method of general examination. In the first place, a very careful history, with full attention to all details, should be elicited ; this will show whether or not the patient was in perfect health up to the onset of the symptoms ; whether or not certain intestinal symptoms developed before the beginning of the present trouble, or whether the functions of the bowel were perfectly normal up to the onset of the disease—in other words, whether or not the symptoms of occlusion developed acutely or slowly.

When the onset of the disease was sudden and appeared in a hitherto healthy individual, the following conditions must be thought of : acute occlusion, volvulus, internal strangulation of the bowel, kinking of the bowel, intussusception, obstruction of the bowel lumen by gall-stones

or some foreign body, and very acute peritonitis. It rarely occurs that patients in whom symptoms of acute occlusion of the bowel supervene on gradually progressive chronic stenosis of the intestine are completely free from all symptoms of intestinal disorder prior to the onset of the disease. Acute occlusion of a chronic stenosis of the bowel may, therefore, in the majority of these cases, be excluded. It need hardly be mentioned that the various possible conditions enumerated above are by no means excluded when the patient has had certain functional disorders of the intestine before the onset of the symptoms of occlusion ; the point to be emphasized, however, is that if all these symptoms are absent, the various diseases enumerated above must all be taken into consideration, and that a negative history makes the diagnosis of one of these diseases more probable.

Obstruction of the bowels by impaction of a foreign body can either be excluded or definitely diagnosed at once from the history, provided the patient is in possession of his mental faculties.

Blocking of the lumen of the intestine by a gall-stone is probably a very rare occurrence in individuals who have enjoyed perfect health up to the time of the onset of the disease. Almost without exception a history of some definite symptoms which can be attributed to attacks of gall-stone colic will be elicited, or if not distinct gall-stone symptoms, at least signs pointing to the conclusion that something pathologic was going on in the region of the liver for some time before the onset of the symptoms of occlusion of the intestine. [This view is by no means universally held ; thus Mayo Robson [1] says : " In many cases there is absolutely no previous history to guide one, and it is quite impossible to say whether or not the attack is dependent on the cause in question or on a volvulus, band, or internal hernia."—Ed.]

The next factor to be considered is the age of the patient. When symptoms of occlusion of the bowel occur in children under ten years, the diagnosis of acute intussusception of the bowel can be made almost with certainty, for this is practically the only lesion occurring at this early age capable of producing occlusion of the intestine. Cases occurring between the ages of ten and forty are usually due to the same lesions—namely, acute intussusception or acute internal herniaform strangulation of the bowel. When the patient is over forty, the diagnosis of axial rotation and knotting of the intestine is more probable. It need hardly be mentioned that there are exceptions to this rule as regards the age of the patient.

No great diagnostic importance can be attached to the sex of the patient ; one thing, of course, must always be remembered, that blocking of the intestine by gall-stones is much more frequent in females than in males, since cholelithiasis is much more common in the former.

Volvulus, internal strangulation of the bowel, intussusception, and very acute peritonitis occur at any age, and are fairly evenly distributed through the decades. All these diseases, moreover, have a similar onset. As a rule, they begin with violent pain and an attack of

[2] A. W. Mayo Robson, *Diseases of the Gall-bladder and Bile-ducts*, 1897, p. 87.

vomiting. One of the most important problems to be solved is whether the patient really has mechanical occlusion of the bowel or whether all the symptoms of occlusion are due merely to paralysis of the intestine, which, in its turn, may be caused by peritonitis or by some other primary lesion.

In a previous section attention was called to the enormous difficulties which often surround this question of diagnosis, and the various points of importance in the differential diagnosis were described in detail. It was specially pointed out that the very first onset of the disease is frequently of great importance in settling this difficult question, and that it is of the utmost importance to make out whether or not the disease began with fever; if very soon after the onset of the first symptoms—that is, within the first hours after the development of pain and vomiting in a perfectly healthy subject—there is an elevated temperature, we are justified in assuming that in all probability the case is one of primary peritonitis and not of mechanical occlusion of the intestinal lumen. At the same time, it must never be forgotten that even very acute forms of peritonitis may occasionally develop without pyrexia. Cases of this kind baffle the diagnostic powers of the most skilful and experienced physicians, and no one can be blamed for errors of diagnosis, and in assuming an occlusion of the bowel in these cases which at an operation or on autopsy are found to be due to peritonitis. [The presence of leukocytosis is in favor of peritonitis; the value of a blood count is well seen in appendicitis, the presence of leukocytosis showing that inflammation is spreading to the peritoneum or that there is a local abscess. The presence of leukocytosis, however, does not exclude acute mechanical obstruction, for a leukocytosis of 75,000 may be present (Osler[1]), and, of course, peritonitis may supervene in acute obstruction, and, conversely, in very severe peritonitis leukocytosis may be absent (Bloodgood,[2] Da Costa[3]). Acute pancreatitis has often been regarded as acute intestinal obstruction. It comes on suddenly, with agonizing pain in the epigastrium, which becomes distended, rigid, and tender; vomiting is rarely stercoraceous, and the degree of collapse is very marked. Cyanosis is sometimes a striking feature. The clinical picture is so characteristic that the day has passed when a correct diagnosis should be regarded as a good guess (Thayer[4]). It has been suggested that acute pancreatitis may possibly be characterized by the presence of a fat-splitting ferment in the urine. Opie[5] has proposed that the urine should be tested to see whether it can, when neutralized with potash, decompose carefully purified ethylbutyrate. The value of this test remains to be more extensively proved: it was positive in a case of acute pancreatitis examined after death by Opie. Another test, which has been described by Mayo Robson[6]

[1] Osler, *Practice of Medicine*, p. 535, fourth ed.
[2] Bloodgood, *American Medicine*, 1901, vol. i., p. 306.
[3] Da Costa, *Clinical Hematology*, 1902.
[4] W. S. Thayer, *American Medicine*, March 1, 1902.
[5] Opie, *Johns Hopkins Hosp. Bull.*, May, 1902, No. 134, p. 117.
[6] A. W. Mayo Robson, "Pancreatitis," *Phila. Med. Jour.*, June 1, 1901 (Cammidge's test).

and is sometimes known as "Cammidge's test," may be mentioned, although no full or exhaustive account of it has yet been published. It is found that if the urines from cases of pancreatitis were boiled for a short time with some oxidizing agent, the phenyl-hydrazin test gave the characteristic (for sugar) delicate, yellow, needle-shaped crystals arranged in sheaths and rosets. A full account of this test and its important diagnostic bearings will soon be published by P. J. Cammidge. Acute pleurisy and pneumonia not uncommonly give rise to abdominal symptoms, and may be mistaken for acute peritonitis due to perforation of the stomach, intestines, or appendix ; this subject has recently been dealt with by H. L. Barnard.[1] In rarer instances acute obstruction has been diagnosed in cases of pneumonia before the characteristic physical signs have become evident.—ED.]

The possibility or probability of simple paralysis of the bowel must also always be considered, especially when the symptoms of occlusion develop under the conditions described in detail in the section on Paralysis of the Bowel. (For a summary of these different factors and the important essential points in the differential diagnosis the reader should refer to p. 585 and to the above-mentioned section.)

In the absence of any signs of simple paralysis of the bowel, or of peritonitis with paralysis of the bowel, the question arises whether the condition is volvulus, strangulation, intussusception, or blocking of the bowel.

In the first place, as has been said, the age of the patient must be taken into account, and, in addition, a careful physical examination of the abdomen and an examination of the functional powers of the different organs must be carried out. From these examinations many points of value in the diagnosis can be obtained.

The presence of local meteorism, provided an examination of the patient can be made early in the disease, may occasionally give us definite information, particularly as to the diagnosis of volvulus of the sigmoid flexure. This diagnosis becomes very probable when there are distended, tensely elastic, but absolutely quiescent loops of intestine, which rise upward from the left lower portion of the abdomen. When there is local meteorism in some other portion of the abdomen,—for instance, when it is limited to the upper or the middle portions of the abdomen,—axial rotation, or less frequently internal herniaform strangulation of the small intestine, must be thought of. It is probably impossible to diagnose the nature of the strangulation of the small intestine—that is, to determine whether it is due to strangulation underneath a Meckel's diverticulum or a false ligament, or whether it is due to the strangulation of an internal hernia and to what form of internal hernia. It is possible that in rare and exceptional cases a diagnosis of strangulation of a diaphragmatic hernia or of a Treitz hernia can be made.

When there is a palpable tumor possessing all the characteristics of an intussusception described above, the diagnosis of invagination of the bowel may occasionally be justified.

[1] H. L. Barnard, *Trans. Clin. Soc.*, 1902, vol. xxxv., p. 122.

Of the functional disturbances bearing on the differential diagnosis of the various forms of acute intestinal obstruction pain is the least significant, for it occurs in all of them. The severity of the pain varies in each case, but in nearly all the different forms it is constant and uniform and one of the initial symptoms. Occasionally the original seat of the pain indicates the position of the lesion. Pain starting in the left hypogastric region points to volvulus of the sigmoid flexure; when the pain starts from the right hypogastric region, the ileocecal form of intussusception should be thought of, and when it starts from the middle or upper parts of the abdomen, internal strangulation or axial rotation of the small intestine is probable.

Vomiting, which also occurs in all these forms of acute occlusion of the bowel, is most common and constant in obstruction and in strangulation of the small intestine. Here, too, it is most violent and occurs at an early stage of the disease. It is approximately just as violent, and appears about as early in axial rotation and in knotting of the bowel, whereas in intussusception, especially in adults, it appears much later in the course of the disease and may even be completely absent. Another factor which must be considered in estimating the significance of vomiting is that in strangulation and in axial rotation of the small intestine it hardly ever relieves the patient's distress, whereas in volvulus of the sigmoid flexure it nearly always brings transitory relief of some of the most distressing symptoms. Another difference between the vomiting of strangulation and of axial rotation of the small intestine, on the one hand, and volvulus of the sigmoid, on the other, is that in the former case the vomited material becomes stercoraceous comparatively often and early in the course of the disease, whereas in volvulus of the sigmoid, intussusception, and in obstruction of the bowel by gall-stones, stercoraceous vomiting is comparatively rare, and when it does occur, appears late in the course of the disease. A form of vomiting which is worth special mention is the vomiting of abundant quantities of bile in cases of obstruction of the upper part of the intestine by gall-stones.

In internal strangulation of the bowel, in volvulus, and in gall-stone obstruction, absolute *constipation* is the rule. In intussusception, on the other hand, absolute constipation is only occasionally seen, and quite frequently there is diarrhea, either alone or alternating with constipation. The passage of blood with the motions points to intussusception with some degree of probability, for in volvulus of the sigmoid flexure it is rare, in internal strangulation of the bowel exceptional, and is never seen in gall-stone obstruction.

The same diagnostic conclusions as regards the form of occlusion of the bowel can be drawn from tenesmus as from the admixture of blood with the stools.

Prostration and sudden collapse are two signs which occur in all forms of acute occlusion of the bowel, but, more frequently, constantly and earlier in strangulation of the bowel than in volvulus. In intussusception and blocking of the bowel prostration and sudden collapse may be entirely absent.

The history that the acute symptoms supervened in an individual who had for some time previously shown signs of interference with the bowel passage (constipation occasionally alternating with diarrhea, repeated attacks of colic) makes the diagnosis of a gradually progressive stenosis of the bowel probable; and it may further be assumed that the stricture has advanced to the point of complete occlusion of the lumen of the bowel, either mechanically or dynamically—*i. e.*, owing to insufficiency of the muscular coat of the intestine.

This category, in which complete intestinal obstruction supervenes on the partial, more or less advanced stenosis of the intestine, is exceedingly important from a practical point of view. By a very careful examination of the patient it may sometimes be possible to differentiate this form of occlusion from the other acute forms of intestinal occlusion which develop suddenly in cases where the intestine was perfectly healthy up to the onset of the first symptoms of obstruction. The following points are of assistance in making this differential diagnosis:

The history in nearly all these cases shows that attacks of colic and other signs pointing to stenosis of the bowel have existed for some time. As Naunyn rightly points out, the recurrence of these paroxysmal signs of stenosis—and these recurrences may occasionally assume the character of symptoms of occlusion—are of value only in the diagnosis of stenosis of the bowel when they appear at short intervals and in rapid succession; long intervals, even several years, between the different attacks of colic, etc., show that the attacks have no bearing on the diagnosis of chronic progressive stenosis of the bowel. In exceptional cases stricture of the bowel exists without any signs or symptoms, especially in the small intestine. It can readily be understood, therefore, that when occlusion of the bowel supervenes on a chronic and progressive stricture, it may imitate the sudden onset of acute occlusion in a previously perfectly normal bowel.

It is, however, often possible to make a correct diagnosis even in these cases. Careful study of a case of this form of apparently acute occlusion often shows that the strictly localized violent pain which is so characteristic of genuine acute occlusion, and usually ushers it in, is absent. It is true, as I can state from my personal experience, that this initial pain may be present in some instances. The general prostration and collapse are not, as a rule, so severe in cases of occlusion supervening on chronic stenosis as in cases of genuine acute occlusion. This can readily be understood when we remember that in the former category strangulation of the bowel and the mesentery does not occur, for the peritoneum and the mesentery are not involved in the process. The most valuable sign in the differential diagnosis is the presence or absence of visible peristaltic movements and tetanic rigidity and stiffening of certain loops of bowel. This sign is rarely seen when very acute occlusion occurs in the normal intestine, whereas it is very frequent in acute occlusion occurring in stricture of the bowel in which the muscular coat of the intestine is hypertrophied.

When it appears that the patient is not suffering from acute occlu-

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sion of a previously normal intestine, but from occlusion supervening on a gradually progressive stenosis of the bowel, the question arises as to the primary character of the cause of the stenosis. The alternatives are a malignant neoplasm, stricture due to cicatricial contraction following ulceration, constriction or compression of the intestine from without by a tumor or by bands of adhesions, obstruction of the bowel by fecal tumors, or chronic intussusception. From the descriptions given of the symptomatology of these different diseases it will be seen that it is simply impossible in many cases to make the differential diagnosis at the bedside. In many instances we must be content with determining that stenosis of the bowel exists. When a tumor is palpable, the diagnostic possibilities become limited, although even then a differential diagnosis may often be impossible. A case illustrating this was mentioned in a preceding section. In some of the cases, however, the clinical picture and the objective signs are sufficiently marked and distinctive to justify a more or less positive opinion as to the nature of the occlusion and of the underlying cause of the obstruction of the bowel lumen. When the history of a patient advanced in years shows that there has been obstinate constipation for years, obstruction is probably due to fecal tumor, especially in the absence of cachexia and in the presence of a tumor which is freely movable and possibly of doughy consistence. This diagnosis is further strengthened if the shape and size of the tumor vary before and after the bowels are open. But the absence of this change does not exclude a fecal tumor.

A tumor due to carcinoma is usually hard and solid. The tumor of chronic intussusception of the bowels is softer, and, moreover, presents one peculiar feature which may be considered characteristic— viz., repeated palpation at frequent intervals shows that sometimes the tumor feels hard and sometimes soft. This must be assumed to be due to rigidity and tetanic contraction of the affected portion of the bowel. In the account of the different disease-forms the slight variations in the clinical picture which occasionally decide the diagnosis in favor of this or that type of tumor were described in detail. Occasionally these apparently insignificant modifications in the syndrome, combined with the characteristic signs of chronic stenosis, give us definite information as to the nature of the tumor felt. Chronic intussusception and carcinoma cannot, of course, be excluded, even in the absence of a tumor.

The presence of tumors compressing the intestine from without can, in the great majority of cases, be detected if the patient is examined with sufficient care.

When no tumor at all can be felt, either because there is none or from the presence of meteorism, all the processes described above may be present, and, in addition, all the symptoms of stenosis of the bowel, due to cicatricial constriction of the bowel, to anatomic abnormality, or to peritoneal adhesions constricting the bowel from without. In all cases of this kind the history and a number of accidental circumstances may provide important information. If it can be shown that the stricture is in the small intestine, cicatricial (particularly

tuberculous) stricture and constriction due to peritoneal adhesions are more probable than any other condition, for carcinoma is very rare in this part of the bowel. In stenosis of the large intestine, on the other hand, all these factors, as well as dysenteric and syphilitic strictures, must be thought of. The finer differentiation between the two latter forms of stricture must depend on the history. Other possible causes of stenosis which must be considered in stenosis of the large intestine are cicatrices from stercoral ulcers, especially when there is a history of chronic constipation, and, lastly, annular carcinoma. If there is a history of chronic pelvic inflammation, of old hernia, or of perityphlitis and similar diseases, peritoneal adhesions must always be thought of. It is only rarely possible to prove beyond doubt that such a diagnosis is correct. In the great majority of cases the diagnosis is not more than one of probability; but a correct diagnosis has often been made either by a fortunate guess or from a lucky combination of accidents.

It is evident, therefore, that even under ordinary circumstances, and apart from the fact that a careful examination of the patient is sometimes quite impossible, the diagnosis of the causes producing stenosis of the bowel is exceedingly difficult; in addition, however, extraordinary circumstances which cannot be predicted may make the diagnosis in certain cases still more difficult. A considerable number of cases, for instance, are on record in which two or even more factors obstructing the passage of the bowel contents existed simultaneously in one individual, such as multiple intussusceptions, multiple cicatricial strictures, multiple peritoneal constrictions and adhesions, or, to quote a case described above, strangulation of the bowel by a band in one place and volvulus of the sigmoid flexure. Hochenegg has described, under the name "combination-ileus," a form of occlusion of the intestine in which, besides a chronic progressive stenosis of the large intestine, there was also stenosis of the small. After the former was relieved, symptoms of occlusion soon recurred.

The above sketch of the methods of diagnosis to be followed in deciding the seat and the nature of the occluding agency shows how exceedingly difficult the recognition of the anatomic character of the obstruction may be. The only way in which the medical man can arrive at conclusions satisfactory to himself and of value to the patient is by careful study and analysis of each individual phenomenon presented; he must never forget that any given case may deviate considerably from the general and commonly accepted symptomatic scheme. The medical man must also always be prepared for entirely unexpected conditions, even when he has apparently considered all the possible complications. I consider the determination of the nature and seat of an intestinal obstruction to be the most difficult task of anatomic diagnosis.

TREATMENT OF STENOSIS AND OCCLUSION OF THE INTESTINE.

THE following remarks on the treatment of stenosis and occlusion of the bowels are written by a physician and not by a practising surgeon. I do not feel justified, therefore, in passing judgment on the different operative procedures *per se*, nor on the method and technic of operations. From my point of view as a physician, I shall describe the various forms of medical treatment in stenosis and occlusion of the bowel as distinctly and fully as possible from my personal experience and that of other clinicians. The course of treatment will be described until the patient's condition justifies his being transferred to my surgical colleagues.

Every medical man who has debated over many cases of occlusion of the bowel realizes how often it is necessary to decide at once the important question of treatment. The majority of medical men who are in frequent contact with emergency cases of this kind have formulated a set of rules for their own guidance. As these rules are evolved from the personal experience of a great many individual men, it is not surprising to find that the treatment of stenosis and occlusion of the bowel varies very greatly at the present time. This is to be regretted, for it is precisely in this class of diseases that human life frequently depends on the clear judgment and the rapid decision of the medical attendant. It is, therefore, very desirable that general fundamental rules should be formulated for the treatment of this disorder. These rules, which would, of course, be chiefly formulated for those who have only a moderate amount of experience, are more necessary in occlusion of the bowels than in any other disease.

For thousands of years medical men indulged in fruitless, barren, and theoretic speculations as to the nature of intestinal occlusion. Of recent years the study of pathologic anatomy has thrown much light on the conditions existing in this disease, and has shown that all the methods of treatment employed by the older physicians were valueless and quite incapable of influencing the course of the disease. Within recent years the ancient so-called *systems* of cure, which were long in vogue and were exceedingly crude, have been gradually eliminated from our armamentarium ; unfortunately, however, no improved methods of treatment were substituted for them. The natural result was general therapeutic pessimism. For a time, enemata, purgatives, and a few other means of this character were employed. If these failed to bring relief, the patient was usually left to his fate.

Gradually, however, a clear insight was gained into the physiologic conditions obtaining in cases of stenosis and occlusion of the bowel, and the functional perversions which resulted from these accidents were soon understood. One of the direct results of this increased knowledge, and possibly in part of the mass of objective *clinical* observations on record, was the introduction of opium preparations in the treatment of intestinal occlusion. In addition, we soon learned to employ a number

of important bloodless mechanical methods of treatment. Gradually, then, the so-called internal treatment of occlusion of the bowel developed into a set of rules and regulations which were uniformly adopted with only slight modifications by the great majority of medical men; and it is consequently possible to enunciate a few general rules for the treatment of this disease. Notwithstanding, however, the great advances of recent years, the results of internal treatment are still exceedingly unsatisfactory.

At this period surgical technic began to be perfected. The discoveries of Lister made operative interference in a large number of abdominal diseases feasible, and it seemed probable that possibly operative treatment of this almost hopeless disease might be successful. The advisability of surgical interference in stenosis and occlusion of the bowel, which had up to this time been advocated only in exceptional cases, and more to satisfy scientific theories than in the expectation of therapeutic results, was now more and more insisted upon by competent surgeons. The discussion of the treatment of intestinal occlusion at present hinges on questions of a surgical nature.

We shall now attempt the description. My conclusions will be based exclusively on the experience and investigation in this field. From the exigencies of space in a work like this the description of this subject must necessarily be short and concise, and hence minute details will be omitted. It is also very important that in discussing this question the personal equation should be eliminated as much as possible, for the experience of any one individual can hardly be considered valid and representative. I shall endeavor, therefore, to summarize and to reconcile, as far as possible, the views of a number of individual observers. In view of the tremendous number of publications on the subject, it is impossible to mention authors' names in all instances.

Before any treatment whatever is begun in occlusion of the bowel, it is absolutely necessary that the *indications* for the particular form of treatment to be employed should be perfectly clearly understood, otherwise all therapy is crude empiricism. It is utterly useless to try this, that, or the other remedy, or to rely on the chance effects of one or the other means. It must also always be fully realized what may be expected from our remedies and from the mechanical adjuvants to the treatment that we employ. Before prescribing medicines or before adopting manipulative treatment, as definite a diagnosis as possible must always be made, and the natural course of the disease, when uninfluenced by treatment, should be clearly recognized. Finally, we must have a clear conception of the physiologic factors and their perversions which are responsible for the clinical symptoms and conditions.

From this point of view the indications for treatment in stenosis of the bowel and in occlusion will first engage our attention.

It is, relatively speaking, an easy matter to determine the indications for treatment in all those conditions produced by slowly progressive chronic stenosis of the intestinal lumen. In nearly all these cases, with the exception of fecal accumulation and a few isolated instances

due to other causes, a radical cure, or at least a partial reëstablishment of function, can be brought about only by surgical interference. This applies to carcinomatous and cicatricial strictures, and to constriction of the intestine due to peritoneal adhesions. These morbid conditions, which lead to stricture of the bowel, of course never undergo spontaneous cure, and cannot be relieved by so-called internal measures. The same applies to the cure or relief of nearly all those pathologic lesions which compress the intestine from without. Only a few of them are, under certain conditions, amenable to treatment by other than surgical methods—as, for instance, the replacement of displaced organs and the reduction of inflammation in certain inflammatory swellings.

In all these cases the physician can determine the time for operation if he has succeeded in recognizing that the symptom-complex presented is due to a gradual increase of the intestinal stenosis. As soon as a correct diagnosis is made, and fecal accumulation or one of the other causes which cannot be removed by operative measures have been excluded, an operation is indicated, provided a radical cure of the condition seems desirable, and provided the operation is technically possible and is not contraindicated for any possible reason. For example, in cases in which neoplastic growths exist but without metastases or wide-spread adhesions in the neighborhood, and in which, finally, the general condition of the patient is good, an operation may be conscientiously advised.

When a case of chronic stenosis gradually or suddenly develops into complete occlusion of the bowel, the same indications for treatment exist as in acute occlusion of the bowel occurring in an intestine which was normal and permeable up to the onset of the first symptoms of occlusion ; in both instances radical measures must be employed at once in order to save the life of the patient. Whether or not the operation that is performed under these conditions is to bring about a radical cure—that is, complete removal of the cause of the obstruction—is another question, and must be decided separately.

Other general indications for treatment in cases of stenosis of the bowel are almost self-evident, such as the necessity of withholding all articles of diet which might mechanically fill up the intestine, and of a most careful regulation of the bowels. Patients of this kind should always have regular evacuations.

The general indications for treatment in cases of acute occlusion of the bowel are very complicated, and differ radically from the indications existing in cases of chronic and progressive stenosis. The difficulties begin with the diagnosis. In cases of chronic stenosis there is always time to examine the patient repeatedly and to think the case over at leisure ; in acute occlusion the symptoms develop with such rapidity that a final decision must often be made within a few hours. A physician in private practice is usually much more fortunately situated as regards these cases than a physician in hospital practice, for, as I have shown above, the very first onset is the only characteristic and

pathognomonic point in many of these cases, so that an examination of the patient during the first twelve to twenty-four to forty-eight hours usually determines the diagnosis, and so gives the indications for treatment. Later in the disease the well-known distressing clinical picture is presented. In all the cases there are a certain number of phenomena which are common to all cases of obstruction of the bowels, namely, collapse, vomiting, complete obstruction of the bowels, and abdominal distention. Under these circumstances, as I have shown by several examples, it is quite impossible to examine the patient in a satisfactory manner, and all attempts to obtain information as to the anatomic nature of the primary lesion are futile. In many of these cases the diagnosis rarely advances further than the preliminary question—whether the intestinal obstruction is due to some mechanical obstruction or to motor paralysis of the bowel. This question is particularly difficult to answer in cases complicated with peritonitis.

When a diagnosis of mechanical occlusion of the bowel can be made, the next question to be answered is as to the anatomic nature of the obstruction present in each case, the treatment necessarily varying according to the type of obstruction. It is self-evident that the subsequent treatment, especially operative interference,—and this is the most important question,—is dependent on this diagnosis. We must decide as soon as possible whether an operation is indicated at all, whether the operation should be performed at once, and, in particular, whether we are justified in advising an exploratory laparotomy on the chance of finding the obstruction and removing it.

A variety of possible types of mechanical obstruction varying in their anatomic nature must be considered. It must be determined whether there is internal strangulation of the bowel, volvulus, knotting of the intestine, kinking, intussusception, or obstruction by a gall-stone or by some other foreign body. Finally, it must be decided, if possible, whether the case is one of chronic stenosis or of fecal accumulation in which the occlusion of the bowel has suddenly become complete.

The last three of these six conditions, we know, occasionally recover without operative interference, or spontaneously, or at least with the aid of certain bloodless measures and manipulations. As regards the first three of the six groups (internal strangulation, volvulus, acute kinking), the first fundamental question to be settled is, whether a restitution to normal or relief of the alarming symptoms is possible without operative interference. A negative answer to this question establishes the first and the most important indication for treatment, for it is then clear that the only way of saving the patient's life is by an operation for the relief of the obstruction. Having once decided this, the operation should be done immediately, with the slightest possible loss of time, for a delay of only a few hours always reduces the chances of recovery.

Many authors, particularly surgeons, deny the possibility of spontaneous restitution to normal, or even of a partial restitution, by measures short of operation in internal strangulation, volvulus, or kinking of the

bowel, and, therefore, invariably advise operation, and, moreover, that it should be performed as soon as possible.

Other observers—and these include chiefly physicians and but few surgeons—advocate another view, namely, that a spontaneous relief of the occlusion is possible in these three dreaded forms of the disease, particularly in the early stages. They believe that the mechanical causes of the occlusion can be influenced by bloodless measures in very many cases, and that some of these manipulations should be tried before operating, so as to bring about a spontaneous relief of the symptoms. Curschmann is one of the most emphatic advocates of this view, which is indorsed by many other experienced observers. Personally, I believe that in a certain percentage of the cases in which the anatomic conditions are particularly favorable, and in early stages of the disease, internal strangulations, volvulus (of 180 degrees), or slight degrees of kinking can be relieved spontaneously. From a very thorough study of the literature on the subject, and from considerable personal experience in this field of medicine, I do not hesitate to express the opinion that such spontaneous cures do occur in these conditions ; on the other hand, I do not think that it is possible that total axial rotation, knotting of different loops of intestine with one another, very firm and large strangulations, or severe degrees of kinking of the bowel can ever undergo spontaneous cure. Why I advocate early operation in all these cases will appear further on.

In addition to the indication for treatment derived from a knowledge of the anatomic conditions,—an indication which may be called *indicatio morbi*,—a second series of very important factors must also be considered in treating these patients.

Many patients with occlusion of the bowel pass into a condition of shock and become collapsed. These two conditions must be treated and carefully considered when planning the lines of treatment. Stress need hardly be laid on the necessity of distinguishing between the initial shock and the state of collapse which comes on later in the course of the disease. Both of these forms of shock or collapse must be treated directly.

Another consequence of occlusion which requires special treatment is the enormous increase of the intra-abdominal pressure. This should be relieved, as it exerts a most deleterious influence on the anatomic conditions that are causing the trouble. Cases are on record in which measures intended to do no more than to reduce the intra-abdominal pressure produced an amelioration of the symptoms and ultimately a cure of the disease. It is clear, therefore, that this important symptom should always be carefully considered in planning the general lines of treatment in occlusion of the bowel.

The preceding remarks are general in character, as it seems advisable to preface the more special and practical remarks that are to follow with these general considerations in order to make the subject more lucid and clear. For the purpose of making the discussion more comprehensive and more systematic, acute occlusion of the bowel will be con-

sidered separately from stenosis, and internal treatment will be separated from surgical treatment. Here and there, of course, it will be impossible to avoid treating one or other of these therapeutic measures from different points of view.

TREATMENT OF OCCLUSION OF THE INTESTINE.

NON-OPERATIVE TREATMENT.

The therapeutic measures available in occlusion of the bowel may be subdivided, as we have seen, into two great groups : on the one hand, so-called internal treatment, including simple puncture of the intestine ; on the other hand, surgical treatment, which implies opening of the abdominal cavity or of the intestine by the knife.

In each individual case of occlusion of the bowel the first and the most fundamental question to be decided is whether or not internal treatment should be employed. It must always be decided whether good results can be expected from such internal treatment ; whether it is at all permissible, or whether the disease has progressed so far that it would be wrong to persist in internal treatment, and, lastly, whether an operation is indicated, and whether it should be done at once.

The physician should always remember that no general principles of treatment exist in this disease that are applicable to all cases, or, at best, that such principles can be applied only in a very limited degree. Here more than in any other disease the medical man must individualize and must consider the peculiarities of each case in deciding the general plan of treatment and the methods he wishes to employ in order to bring about a cure.

In many cases the first examination of the patient will remove all doubts as regards the advisability of an immediate operation ; in other words, it is at once clear that surgical interference is the only procedure that can save the life of the patient. These cases will be referred to below.

In other instances bloodless measures may be indicated at first, and should be tried, for there is no doubt that a considerable proportion of even severe cases with all the symptoms of occlusion of the bowel fully developed may recover without an operation ; in fact, many observers state that one-third of all their cases of occlusion of the bowel recovered without an operation. In view of this the medical attendant is not justified in denying the patients the benefit of the doubt, and should always inform them of the possibility of recovery without an operation. A further argument in favor of bloodless procedure in cases not calling positively for immediate operation is the fact that the operation itself is not entirely without danger.

Some of the enthusiastic advocates of immediate operation in all these cases claim that the majority of instances in which patients with occlusion of the bowel recovered spontaneously without an operation were really cases of simple fecal accumulation. This I deny most decidedly, and believe that those who make statements of this kind greatly exag-

gerate and have no grounds for their arguments. There can be no doubt whatever that many of the cases of occlusion of the bowel which get well spontaneously are cases of internal strangulation, kinking, axial rotation, intussusception, or complete blocking of the bowel by gall-stones and foreign bodies.

The most difficult task for the physician is to determine when he is justified, in view of the existing conditions, in employing internal treatment and in refraining, for the time being, from operative interference. What are the factors which determine this decision?

In the first place, the general condition of the patient must be carefully considered. I believe that internal treatment should be tried only, in fact is indicated only, in those cases of occlusion of the bowel in which there are no symptoms of strangulation; further, that internal treatment should be employed only when the heart's action and the pulse are normal; in particular, when the arterial tension and the frequency of the pulse are not increased. The local symptoms of occlusion, such as meteorism and vomiting, are not so important in this respect.

Generally speaking, the action of the heart is specially favorable in cases which run a very moderate course; in other words, when the disease is not ushered in with symptoms of violent shock.

When at the onset the diagnosis is quite uncertain, bloodless measures should always be attempted, provided, of course, the action of the heart is good. The chances of success by operative measures are favorable in proportion to the evidence which the surgeon can gain from his examination of the patient as to the exact position and nature of the obstruction, and consequently of the surgical technic to be employed. Internal treatment, therefore, is specially indicated when the diagnosis is uncertain, because a certain amount of time is gained while this treatment is being carried out, and it is always possible that repeated examinations of the patient may furnish very important information as regards the indications for operative treatment; by thus delaying while the diagnosis is obscure, we may hope to improve the chances of the patient should an operation become necessary after all; for the surgeon may thus be in a position to perform it with a more satisfactory knowledge of the conditions he expects to find and to relieve by surgical interference.

When, on the other hand, the diagnosis is perfectly clear, internal measures should be more extensively employed in some of the forms of occlusion,—namely, blocking of the lumen of the bowel by gall-stones, or occlusion of the intestine by fecal accumulation,—whereas in other forms, as in axial rotation, kinking of the bowel, and internal strangulation, internal treatment should not be attempted. In some of the latter cases it may be necessary to advise some manipulative treatment or the administration of some remedy in order to quiet the patient and to satisfy his friends. While this treatment is being carried out, preparations for the operation should be made. In intussusception of the bowel, finally, internal measures should at least be tried for a time. In those cases that call for operative treatment it may still be advisable to delay and to administer internal measures for a little time, chiefly

because, as I have shown above, the possibility of spontaneous recovery can never be excluded, although, as has been pointed out, the probability of such a favorable issue is almost infinitesimally small. This applies specially to those cases in which the symptoms are so definite that they justify a positive diagnosis of axial rotation, kinking, or internal strangulation. If the diagnosis, however, is absolutely certain, the technic of the operation can be so carefully planned and consequently the chances of success be so much improved that it is not well to hesitate any longer than is necessary in cases of this character. When, in a given case, it is decided that internal treatment is justified and is begun, the question always arises, When should this treatment be stopped, and when should the patient be operated upon? The medical attendant should have well-defined views on this subject, and should arrive at his decision on the ground of certain general principles. Every given case, of course, must, nevertheless, be considered on its own merits, as each individual case presents new and unexpected problems. As I have said in discussing the various bloodless methods of treating this disease, some authors condemn their use altogether; such as, to mention the most important ones in use, the administration of opiates and lavage of the stomach. It is claimed that both the patient and the medical attendant are lulled into a false sense of security, while at the same time the morbid process is allowed to continue in an insidious manner and gradually to progress from bad to worse, and that thus the proper time for operative interference is allowed to pass by. I quite admit this danger, but I also believe that it can be avoided if the patient is continuously watched with the greatest care, special attention being paid to the action of the heart. It is, however, true that even with the most rigid observation of the patient the heart may suddenly fail. Against unexpected heart failure, which often happens, we are, I fear, entirely helpless.

The medical attendant should base his treatment on the fundamental principle that all internal measures or bloodless remedies should be administered with a definite object in view, and, it may be added, in rapid succession. All the measures that we are familiar with should be given, according to individual indications, in the course of the *first day or two;* in exceptional cases, provided the pulse is good and the patient's general condition remains favorable, *for three days.* Cases are undoubtedly on record in which a resolution of the occlusion occurred under this treatment even after obstruction had existed for six to eight days; on the other hand, however, it is just as true that the chances of success by operative interference are less favorable the longer the operation is delayed.

An account will now be given of the various bloodless measures that can be employed:

The Diet.—All authors agree as to the regulations that must be imposed in regard to the amount and quality of food and drink to be taken. In the treatment of occlusion of the intestine the regulation of the diet constitutes an important feature.

In cases of acute occlusion of the bowel all food should be completely withheld from the very beginning. The withdrawal of food becomes a self-evident rule in cases in which the patients vomit a great deal and are tortured by constant nausea, so that they themselves refuse to eat anything. Even, however, when the patients do not express an aversion to food, they should still be forbidden to eat anything whatever. The reason for this withdrawal of food is that, in the first place, its ingestion would be useless and not fulfil the purpose for which the food is given—namely, to nourish the patient. We know that digestion and absorption are reduced to a minimum in those portions of the bowel that are situated above the occluded area, and even that food which is converted into an assimilable form is very frequently vomited. This may be called the negative indication for the withdrawal of food. The positive indication is that the administration of solid food and liquid material *per os* may directly exacerbate the process we are trying to relieve. The entrance of food or drink into the stomach usually precipitates vomiting anew, and, in addition, adds to the quantity of material filling up the intestine above the obstruction. This overfilling of the intestine is the more important factor of the two, for the bowel becomes overloaded and distended; at the same time, of course, the intra-abdominal pressure is increased, and in this way the possibility of a spontaneous resolution of the occlusion is rendered more difficult. Finally, overloading of the bowel favors the occurrence of insufficiency of the muscular wall of the bowel, and thus leads to a more rapid completion of the vicious circle, and, finally, on account of the distention, favors a more rapid onset of peritonitis.

The best way in which to alleviate the torturing thirst from which these patients frequently suffer is to give them small pieces of ice, with the instruction to allow them to dissolve slowly in the mouth. It may even be permissible to give them very small quantities of iced water (with or without brandy) to swallow.

[Treves[1] condemns, without reservation, the sucking of ice, as it introduces an unknown quantity of cold water into the stomach of a patient who is already probably not recovered from collapse. He allows the patient to rinse the mouth with cold water as often as he likes, and occasionally to chew a minute piece of lemon. The mouth must be kept very clean, and if dry, the tongue may be occasionally painted with boroglycerid and water. Intolerable thirst is best treated by enemata of warm water (99° F.). There seems to be no evidence that subcutaneous or intravenous transfusion is more efficacious than enemata.—ED.]

In patients with profuse vomiting, in whom, consequently, the percentage of water in the tissues is being reduced and the quantity of fluid in the vascular system is also being diminished, small quantities of water, or water mixed with wine or brandy, must be repeatedly injected into the rectum. Curschmann, Kocher, and others recommend subcutaneous infusion of physiologic salt solution in these cases,

[1] Treves, *Intestinal Obstruction*, second edition, 1899.

and the last-named author wishes, in fact, to substitute these measures for fragments of ice by the mouth, in order to obviate any increased strain above the occlusion.

While it is true that complete withdrawal of food is necessary during the first days of the disease, we must, nevertheless, know what to do in those cases that run a more prolonged course, especially in intussusception of the bowel, in which, sooner or later, the question arises as to how these patients are to be fed. The best way in which to administer food under these circumstances is by the well-known nutrient enemata.

Internal Laxatives.—Formerly laxatives and purgatives were very generally employed in the treatment of occlusion of the bowel, but modern views as regards this treatment have changed considerably, and, fortunately, there is general unanimity of opinion among clinicians. The results of thorough investigation of this subject, obtained from practical experience, and the theoretic conclusions obtained from experimental research, are in agreement.

In all forms of acute, subacute, and chronic complete occlusion of the bowels laxatives definitely do harm, and should, therefore, be rigorously avoided. The only form of occlusion of the bowel in which laxatives are permissible is in blocking of the lumen of the bowel by masses of fecal material. It is only, therefore, when the diagnosis of fecal impaction is absolutely certain, that the administration of laxatives *per os* is justifiable. When this diagnosis is not perfectly clear, the medical attendant should most religiously avoid the administration of any laxative whatever to a case in which the symptoms point to intestinal obstruction. Universal experience shows that purgatives and laxatives not only do no good, but make the patient's condition worse both subjectively and objectively ; in fact, the fatal issue has often been precipitated in cases where recovery would probably have occurred had this treatment not been adopted.

The old view as to the mode of action of purgatives and laxatives in cases of occlusion of the bowel was that they stimulated peristalsis, and that the increased peristalsis and activity of the muscular coat of the bowel overcame the obstruction. Let us inquire what the true condition of affairs is when purgatives are given in a case of occlusion of the bowel. In chronic stenosis of the bowel which has progressed so far that the occlusion of the intestinal lumen is complete, inspection of the patient's abdomen alone shows that enormous peristaltic waves and movements and stiffening and rigidity of the bowel-wall occur ; in other words, that the intestinal wall is performing the maximum propulsive effort that it is capable of. It can hardly be imagined that the irritation exerted by purgatives could increase these efforts. In cases of acute occlusion of the bowel supervening in a healthy person whose intestine was normal and permeable up to the time of the occlusion, the peristaltic movements and the exaggerated muscular efforts of the intestine are not, it is true, visible through the abdominal wall. This is due to the fact that in this form of occlusion the increased work performed by the bowel is not so great, for the simple reason that the muscular

coat of the bowel is not in the same condition of hypertrophy as in cases of chronic stenosis, although its contractions are also very active. I have been able to demonstrate this by experiments, and have also been able to determine by auscultation of the abdomen at the bedside that very active borborygmi can be heard under these conditions. This increase in the peristaltic movements of the bowel is detrimental to the patient in one respect. When the obstruction persists in spite of this increased peristalsis,—and this is almost always the case,—the increased intestinal movements will do direct harm. When the intestine immediately above the obstruction is performing particularly active and forcible propulsive movements, it can readily be understood how a large quantity of bowel contents will be propelled onward as far as it will go. In this way the portion of the bowel situated immediately above the occlusion will gradually become distended with bowel contents. Overdistention and paralysis of the muscular coat of the bowel-wall follow, and the mechanical occlusion of the bowel becomes complicated by paralysis of the intestine.

No proof, moreover, has ever been adduced to show that violent peristaltic movements in any way tend to remove the mechanism that produces the occlusion of the intestine; on the contrary, intussusception, axial rotation, and internal hernial strangulation of the bowel are probably unfavorably affected, at least to judge from what is known as to the causal mechanism of these lesions. A foreign body obstructing the intestinal canal will probably not be any more forcibly driven on by exaggerated peristalsis than by normal peristalsis. In intussusception it is frequently possible to determine by direct observation that the lesion is made more complicated and more obstinate by the administration of a purgative; the intussusception tumor can be felt to become progressively harder and larger—the exact opposite is noticed in cases that are treated with opium. Purgatives, moreover, increase the pain and the vomiting. Sometimes vomiting does not become profuse until purgatives are administered, or becomes stercoraceous only after the administration of purgatives. Collapse may also be accelerated in this way, and a few cases have been put on record in which the administration of purgative drugs seemed to be immediately followed by perforation of the bowel.

It is true that even now a case is occasionally reported by some good clinical observer in which the obstruction was removed by the administration of a purgative even after fecal vomiting had set in, and some cases are stated to have been cured in this way. But, let me ask, who can prove that these were really cases of strangulation of the bowel, of axial rotation, kinking, or intussusception, and that they were not simple cases of obstruction of the bowel by fecal material? Are we justified in administering purgatives on the mere chance that the diagnosis is doubtful, and that possibly the case is one of fecal tumor? Should the patient be given the benefit of this doubt, and, on the slight chance of their being successful, or in the light of the fact that the great disadvantages of purgatives in the majority of cases are well

established, ought purgatives to be avoided? On the whole, I think that the most conservative and the most rational rule as to this point is to withhold the administration of purgatives in all cases of acute occlusion of the bowel which are probably or certainly due to strangulation, axial rotation, intussusception, or kinking, and also in all those cases in which the diagnosis is doubtful.

In all cases, however, where the diagnosis of fecal obstruction can be positively made, purgatives are distinctly indicated; but caution is necessary, especially when there is any reason to suspect that there is a considerable degree of intestinal insufficiency, or possibly of well-developed intestinal paralysis. In cases with insufficiency or paresis purgatives may do harm in the same way as has been described above; they propel masses of fecal material onward, so that an obstruction gradually forms which can no longer pass through the paralyzed segment of the bowel. In this way the intestine is stuffed and stretched, and the paralysis from distention is increased by the mass of fecal material driven into the paralyzed portion from above.

Metallic Mercury.—For several centuries metallic mercury has been credited with great virtues in the treatment of occlusion of the bowel. While it is still prescribed occasionally, it may be said that its rôle in this respect is played out. I feel justified in indorsing the view that the administration of metallic mercury is a method of treatment which can very well be dispensed with, and that, in fact, it must be considered dangerous. If large doses of metallic mercury really exert the mechanical effect that is attributed to them and expected of them, it would do harm in a much larger proportion of the cases in which it is given. The fact that it does not possess these powers renders it less harmful than it might be. It was formerly an article of universal belief that mercury, by virtue of its weight, could remove the obstruction in the lumen of the bowel. For this purpose, then, large doses of from 100 to 1000 grams were administered without any clear conception either as to the nature of the obstacle that this amount of mercury was expected to overcome or as to the manner in which it could force a passage through the impermeable portion of the intestinal canal. As a matter of fact, postmortem examinations show that metallic mercury is nearly always found in a finely divided state scattered throughout the intestine, and not in bulk. Moreover, when mercury is given to a patient with intestinal obstruction which subsequently passes away, the mercury is not passed in the dejecta at once, or even in appreciably large quantities, but continues to be found in a finely divided state in the bowel motions for days, weeks, and even months after its administration. This shows clearly that mercury administered in bulk cannot possibly exert the effect it is supposed to have in virtue of its weight. What slight effect it does exert by its bulk must be considered reflex, for it seems that a large quantity of mercury introduced into the stomach is capable of producing reflex movements of the intestinal wall. This, at least, is the assumption made by Traube. Leichtenstern calls attention to the fact that the presence of mercury in the

stomach, provided it remains there for some time, may produce disten-
tion of the viscus, and in this way cause arrest of vomiting; this is a
favorable effect following the administration of mercury specially em-
phasized by many of the older observers. In isolated cases it may,
however, actually happen that the metal accumulates in large quantities
above the obstacle. This I consider to be very dangerous, for it may
cause the death of the patient by producing rupture of the segment of
the bowel immediately above the obstruction, especially when this por-
tion of the intestine is inflamed or gangrenous. Examination of the
cases reported in the past, and even at the present time, of the cure of
" ileus " by the administration of metallic mercury, shows that they are
far from clear, and more than this, that they are so rare that the use of
this remedy is hardly justified on the ground of these reports and obser-
vations alone.

[Treves, after quoting from Matignon's thesis [1] cases of fecal accu-
mulation in which obstruction was relieved after the administration of
metallic mercury, says : " M. Matignon's cases appear so clear that in
any case of fecal accumulation that has resisted the action of aperients,
enemata, massage, etc., the use of metallic mercury in large doses would
seem to be worth considering, especially as the mode of treatment
appears to be attended by no special risk." McK. Harrison [2] has re-
cently reported 2 cases of acute intestinal obstruction which recovered
after this form of treatment.—ED.]

Opium and Opium Preparations.—When opium was first
introduced in the treatment of occlusion of the bowels, a great deal of
opposition was raised and was felt in Germany, where Pfeuffer was an
energetic advocate of the opium treatment of occlusion. The treatment
is now no longer condemned, and all adverse criticism has gradually
been silenced, especially among internists. Quite recently it appears
that certain surgeons are again entering the lists very energetically
against the use of opium in occlusion of the bowel. They point out
that the effect of opium is to obscure the clinical picture, cause apparent
improvement (while, in reality, the mechanical occlusion with all its
deleterious effects still persists), and thus the time favorable for opera-
tion may pass by.

The favorable effect of opiates on the subjective condition is decep-
tive. The distressing fear, the anxiety, the restlessness, the nausea,
and the miserable vomiting of the patient are all improved by the
administration of opium. The violence of the pain is mitigated, and a
condition of relative well-being and comfort is produced. If opium is
given at the right time,—i. e., at the very onset of the disease,—the
effect on the initial collapse is remarkable and surprising. The pulse-
rate improves, the pulse-beat becomes fuller and stronger, the tempera-
ture of the surface of the body rises from subnormal to normal, the
hippocratic expression disappears, turgescence of the cutaneous capil-
laries is reëstablished, and anuria passes off. All these factors must

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[1] Matignon, *Thèse de Paris*, 1879, No. 340.
[2] J. McK. Harrison, *Brit. Med. Jour.*, 1902, vol. i., p. 1023.

undoubtedly be attributed to the sedative action of opium. There can be no doubt that many of these symptoms are reflex, and that a remedy like opium can reduce reflex irritation by quieting both the nervous centers and the peripheral nerves. It can also be easily understood how opium can reduce the severity of the initial collapse when we remember, as has been shown above, that this condition of collapse is brought about by the exceedingly violent irritation of the sensory nerves of the intestine, of the peritoneum, and of the strangulated mesentery.

Experimental results and theoretic consideration lead us to the further possibility that opiates may diminish the deleterious effect of excessive intestinal movements by quieting peristalsis. It may be further assumed that this produces conditions favoring spontaneous resolution of the occluding mechanism in many cases—*e. g.*, invagination, volvulus, twisting into knots. Of course, the exact nature of these conditions is not yet physiologically discernible. It has been covered by the expression that the "incoördinated" peristalsis is regulated.

Nevertheless, the majority of surgeons are opposed to the use of opiates. They declare that this method of treatment should be avoided because it produces an apparent improvement, or euphoria, which, by concealing important symptoms, hinders diagnosis, and because it causes the patient, and not infrequently the physician also, to postpone operation until the favorable period for it has passed.

In course of time I have come to incline more and more to the surgeon's belief, and though I cannot even now advise an entire abandonment of opium in the treatment of occlusion, I must change the views, expressed in the first edition of this work, and say that only under the following conditions are the opiates useful or allowable :

1. In the very beginning of the affection—*i. e.*, in the first twenty-four, at most thirty-six, hours.

2. Even in this period it is permissible only when a positive diagnosis cannot be made at once and there is violent pain.

3. If internal incarceration or volvulus is at once probable, opiates are to be avoided despite great pain, and immediate advantage should be taken of the only reliable remedy for such cases—operation.

If the patient absolutely refuses operation, opiates are allowable and indicated according to the symptoms. It is needless to say that they may in this case be pushed to euthanasia.

The method of administration varies. If opium is administered internally, it is frequently vomited, and even if it is retained in the stomach, the absorption of the drug by the mucous lining of the stomach and intestine seems to be reduced, or even completely inhibited, in occlusion of the bowel. Subcutaneous injections of morphin are more reliable, and, therefore, are always to be preferred. Another convenient way of administering opium, and a method that I can recommend, is the administration of suppositories of morphin or extract of opium. The best preparation for administration by mouth is tincture of opium.

If a case of occlusion of the bowels is to be treated with opiates early in the course of the disease, the individuality of the patient and

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other concomitant circumstances must always be carefully studied ; the drug should be given in frequently repeated doses until an effect is produced ; the initial doses should not be too small. I advise giving from 0.02 to 0.03 gram of the tincture or the extract of opium every one or two hours, graduallly increasing the dose in the course of twenty-four hours up to 0.5 to 1.0 of the tincture or extract. Morphin should be given in corresponding doses. The subsequent development of the case will show whether to continue the use of opium, and for how long a time.

[Treves gives the following rules as to the employment of morphin in acute intestinal obstruction : (1) Morphin must be given to relieve the pain, which is, without doubt, one of the most terrible forms of human suffering, and should be given with as little delay as possible. (2) The least amount which will effect this end should be the amount given. In an adult with quite acute symptoms $\frac{1}{4}$ grain should be given hypodermically, and if the pain is not subdued, the subsequent doses should be not more than $\frac{1}{6}$ grain, given at as long intervals as possible. In an adult with symptoms not of the most extreme degree, the initial dose may be $\frac{1}{4}$ grain, to be repeated in one or two hours if absolutely necessary. The advice of some surgeons that in any case the dose should be small—$\frac{1}{10}$ to $\frac{1}{6}$ of a grain—is not in accord with his experience, and he suggests that the advocacy of the small dose is possibly based on an exceptional experience of patients very susceptible to morphin. He insists on the advisability of using " tabloids," in order to avoid the possibility of giving a morphin solution which, from keeping, has developed some apomorphin, which is well known to be a ready emetic. No advantage is to be gained from combining atropin with morphin ; in fact, it may add to the thirst and painful dryness of the mouth. —Ed.]

Atropin.—Belladonna was employed in the treatment of ileus over a century ago, and then was completely forgotten for a number of years. Recently it has been again recommended, and that it should be readily accepted by practical physicians is easily conceivable. Unfortunately, the available material, which has been collated and critically reviewed by Honigmann, is not yet sufficient to justify positive judgment. We cannot reason from theoretic grounds, as our knowledge of the action of belladonna on the sound intestines is not at all complete. We have only clinical observation to go by, and those reported to date are mostly very incomplete and obscure. One thing is quite certain, that of the cases that progressed favorably under atropin treatment not one was a case of strangulation-ileus. Recoveries seem to have occurred only in cases of so-called dynamic ileus, in occlusion from gall-stones, in cases of renal and biliary calculous colic, with symptoms of occlusion, and in occlusion from fecal obturation. My only personal experience was a case of gall-stone occlusion, which recovered under atropin treatment. It cannot yet be decided whether or not the indications for atropin treatment are the same as those for opium, but under no circumstances must we allow atropin treatment to interfere with the indi-

cations for operation. In this connection the same remarks hold true as concerning the use of opium. Whether atropin gives better results than opium, and if so, under what conditions, is yet to be decided.

Individual physicians, it is true, have from the start administered doses far in excess of the maximum, but it is always best to begin with small doses, on account of the symptoms of intoxication frequently observed; caution is ever advisable. Honigmann warns against large doses in elderly patients. It is always administered subcutaneously— 0.0005 to 0.001 gm. a dose, seldom 0.002 gm. a dose, to a total of 0.003 to 0.005 gm. a day.

[Ostermaier,[1] who found that cases of hernia underwent spontaneous reduction after its use, believes that it acts by constricting the blood-vessels in the wall of the strangulated bowel and so reducing swelling. —Ed.]

Lavage of the Stomach.—Kussmaul first introduced this method into the treatment of acute occlusion of the bowels. It rapidly gained general recognition, and is now employed by the majority of physicians. Apart from surgical methods, I consider the evacuation of the stomach by siphonage, or lavage of the stomach, to be one of the most effective measures that we possess. The value of this method depends essentially upon the following factors : In the first place, enormous quantities of stomach-contents are frequently removed in cases of this kind; this reduces the intragastric pressure and so favors regurgitation into the stomach of some of the intestinal contents which are under much higher pressure (Oser). It can readily be shown that a very active regurgitation of intestinal contents into the stomach actually occurs under these conditions, for siphonage of the stomach two or three hours after the first evacuation of the stomach-contents will always produce large quantities of material that must have entered the stomach in the mean time. The external evidence of this effect are changes in the outline and the tension of the abdomen; the patients also usually feel better, for the constant gagging and vomiting of fecal material soon stop. As a further valuable effect of the irrigations, Kocher states that the indirect emptying of the bowels is "a valuable means for the temporary relief of the state of collapse from intoxication."

The effects of siphonage of the stomach and removal of its contents are not only palliative and symptomatic, but unquestionably also occasionally contribute to an actual cure of the disease. A considerable number of cases in which this happy result was obtained are on record. The first two cases that Kussmaul himself studied possess sufficient historic interest to be quoted here. In one of his patients complete occlusion of the bowel had existed for a week; the patient was vomiting fecal material and had lapsed into a dangerous state of weakness. The stomach was washed out five times within twenty-four hours, and the occlusion of the bowels disappeared and the patient finally recovered. In a second case the occlusion of the bowels had existed for nine days and was producing very serious symptoms. An enterotomy had been

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[1] Ostermaier, *Münch. med. Wochenschr.*, September 9, 1902.

decided upon as a last resort. In this case the stomach was thoroughly evacuated once, and almost simultaneously the lumen of the bowel became patent. The curative effect of lavage of the stomach must undoubtedly depend on the fact that removal of large quantities of stomach-contents produces conditions, as we have seen above, which favor the cure of the occlusion of the bowel. When the stomach-contents are removed under these conditions, the pressure in the intestine is reduced, and excessive peristaltic movements that may be going on are in this way also stopped. In addition, the pressure exerted by the distended and inflated loops of intestine upon one another is reduced or entirely relieved, so that it can easily be imagined that a return of strangulated, kinked, or twisted segments of the bowel into their normal position may be facilitated and rendered possible. This explanation of the good effects of lavage of the stomach in occlusion of the bowels seems to be quite rational.

Too much should not, of course, be expected from this procedure; it is enough that it has been known to produce a cure in some cases. Unfortunately, it is quite impossible to predict in advance whether any given case will be benefited by this procedure. Curschmann occupies a position that is slightly at variance with that of many other authors. I agree with his view that the good effects of lavage of the stomach are more apparent in occlusion of the small intestine, especially when the seat of obstruction is high up and near the stomach, than in occlusion of the colon. Many surgeons object to lavage of the stomach for the same reason that they object to the use of opiates—namely, on the grounds that a misleading condition of euphoria is created both in the mind of the patient and of the medical man. Of course, the physician should never allow himself to be lulled into a sense of false security even when the symptoms seem to improve after lavage of the stomach. He should, moreover, observe and watch the case with particular care under these conditions, in order not to allow the proper time for operative interference to pass. Some clinicians object to this method on the ground that aspiration pneumonia may occasionally result. Ewald has refuted this objection, however, by showing that the aspiration of the stomach-contents into the lungs can readily be avoided by tightly squeezing the stomach-tube close to the mouth, and then withdrawing it rapidly with one long movement.

One other rule should be observed in washing out the stomach in cases of occlusion of the bowel—namely, not to adopt this treatment too late in the course of the disease. It is quite unnecessary to wait until stercoraceous vomiting appears, for it is a matter of experience that the stomach may contain fecal material long before fecal vomiting occurs. Another point to be remembered is that when siphonage of the stomach is not sufficient, the stomach should be washed out many times—if necessary, every two or three hours for a time. Occasionally careful measurement of the amount of material withdrawn on each occasion will show how rapidly bowel contents regurgitate from the overdistended intestine into the stomach. The following phenomenon is sometimes

seen : the water pouring from the tube may be perfectly clear, when suddenly it becomes mixed with more fecal material. It is often astonishing what enormous quantities of material can be aspirated from the stomach, in some instances as much as five liters having been removed at one time. The general condition of the patient and his strength should, of course, always be carefully considered, and washing out the stomach should be omitted if the patient is too much exhausted. This point hardly calls for particular emphasis. Lavage of the stomach, like opium treatment, should not be postponed too long, but should be employed early in the disease.

[Treves says that his experience is such that he would advise its use as a routine detail of treatment in every case in which it can be tolerated, and that the danger of the patient vomiting under an anesthetic and the passage of the vomited material into the lung may, to a great extent, be met by washing out the stomach before the operation.—ED.]

Rectal Enemata.—Since time immemorial, a variety of measures have been given by the rectum in occlusion of the bowels in the belief that they could exert an effect on the intestinal obstruction. The chief means employed have been injections of different fluids. The older authors believed that the injection of fluid into the rectum might be of benefit by reaching as far as the obstacle and removing it directly (fecal tumors), or at least by stimulating the tissues in its vicinity so that a restitution to normal was brought about (axial rotation, invagination, incarceration). The exact mechanism by which the latter effect was brought about no one has definitely stated, and it is not easy to gain a clear understanding of some of these older views, nor to find out whether the different writers believed that peristaltic movements of the bowels were stimulated, or that the increase of the pressure from below exerted a beneficial effect.

Against the value of this method of treatment many arguments have been adduced. Here we find theory opposed to theory. Those who object to the treatment of occlusion of the bowel by rectal enemata argue that any increase in the intra-abdominal pressure is more apt to prevent the solution of intestinal occlusion than to favor it. They claim that the distention of the part of the intestine situated below the obstruction undoubtedly causes an increase of intra-abdominal pressure, and in this way acts directly upon the loops of intestine situated above the obstruction and interferes with the resolution of the mechanism that caused the occlusion. They also claim that when, as occasionally happens, the enema is not returned, the danger of the situation may be greatly increased. Again, it is difficult to understand how peristaltic movements which may possibly be stimulated in the section of the bowel situated below the obstruction, can bring about the solution of an axial rotation, of a knot in the intestine, of internal incarceration, or of kinking of the bowel. Finally, it might be objected that the fluid or gas injected never reaches the occluded spot if it is situated above the ileocecal valve unless more or less deep anesthesia is produced, for it is well known that in the living the resistance of this valve can be over-

come only by excessive pressure. It is also well known that very large enemata may occasionally induce dangerous collapse in individuals who are weak and exhausted.

So much for the theoretic arguments *pro* and *con.* in this method of treatment. The only true criterion as regards the efficacy of any method of treatment is practical experience; examination of the reported cases and the results obtained from rectal treatment in occlusion of the bowel show that in all cases of occlusion of the bowel due to fecal accumulation rectal injections are of the greatest benefit, and, in fact, may be considered an indispensable adjuvant to the treatment. There can be no doubt that this method directly causes the intestine to become permeable, especially when it is supplemented by the administration of purgatives by the mouth. Injections of fluid into the rectum not only stimulate the peristaltic movements of the bowel, but—and this is much more important—cause softening and liquefaction of the hardened masses of fecal material that constitute the obstacle to the passage of the contents of the bowel. Another condition in which rectal enemata are of the greatest value is chronic stenosis of any kind. Here the large quantities of bowel contents that are arrested above the stricture become liquefied and can pass on. The question is not positively decided whether the injection of fluids into the rectum is beneficial in cases of acute occlusion of the intestine occurring in a perfectly normal bowel (axial rotation, knotting, internal incarceration, kinking). Possibly this method of treatment may be of use when a gall-stone becomes impacted and obstructs the large intestine. In invagination the method has also been tried, and it has been shown to be effective in a few isolated cases of this lesion in which the invagination extended as far as the large intestine. In these cases large amounts of fluid or of air were forced into the rectum under considerable pressure and led to the disappearance of the invagination of the bowel.

To judge from my personal experience, the value of large injections is very slight in the great majority of cases of acute occlusion of the bowel; many clinicians, in fact, do not employ this method of treatment, for the reason that so many objections can be advanced against it. It need hardly be mentioned that large rectal enemata or the inflation of the bowel with air should he strenuously avoided in any case in which there is good reason to suspect the presence of ulceration or of fragility of the intestinal wall.

In giving rectal injections, the patient lies on the left side or in the knee-elbow position, and water of different temperatures is used. Some clinicians use pure water, others add certain ingredients, while others employ oil instead. The temperature of the pure-water injections may be lukewarm, cool, or ice-cold. There is no question that ice-water stimulates the peristaltic movements of the bowel; whether it is capable of doing this to such a degree as is usually imagined is, I consider, doubtful, from the results of experiments I have performed on this question. As the ice-water gradually becomes warmer in the bowel, its power to stimulate peristaltic movements decreases. Lukewarm and

slightly cool injections do not stimulate the peristaltic movements of the bowels at all by their temperature—they merely act by distending the intestine ; consequently the greater their bulk, the more active the peristaltic reaction (this, of course, applies only to moderate quantities of fluid ; if the quantity of fluid injected is so great that the intestinal wall becomes overdistended, peristalsis cannot occur). We know from experience that comparatively large quantities—viz., from 1 to 2 to 5 liters—of fluid must be used in order to produce any real stimulating effect on peristalsis. If oil is employed, $\frac{1}{2}$ to 1 liter should be injected. Injections of ice-water are entirely condemned by some observers, and I am of opinion that they should be used with care in exhausted patients.

Rectal injections with irritating solutions are also occasionally recommended. The most popular are the infusions of senna and salt solution, the latter being preferable. It is useless, of course, to use salt-water enemata, which are too weak ; the strength of the solution should be at least 5 to 7 or 8 per cent. Irritating enemata, especially those containing a good deal of salt, may pass high up into the intestine. This is due to the fact that they produce antiperistaltic movements wherever they come in contact with the intestinal wall, and are thus carried upward. That this is actually the case was shown by my animal experiments, and also by clinical observation in patients. Even small enemata (not exceeding 400 c.c.) may thus reach the lower part of the ileum. Enemata composed of strong salt solutions, therefore, are a means of reaching intestinal obstructions situated high up in the bowel. Another advantage of these comparatively small injections over the large injections that are usually recommended is that they do not increase intra-abdominal pressure. The value of this factor cannot be overestimated, particularly when we remember that occasionally a large amount of fluid is injected into the rectum, but is retained and not returned by the patient. Finally, I consider irritating salt-water enemata to be of considerable value in invagination of the bowel. In the course of my experiments on animals I have repeatedly seen physiologic intussusception disappear after the injection of irritating salt-water solutions into the rectum. The mechanism was the following : The salt-water solution was injected into the rectum, and wherever it happened to be in the bowel, started antiperistaltic movements ; in this way the salt-water solution was gradually propelled toward the invaginated portion of the bowel. As soon as the antiperistaltic waves reached this portion, the invagination disappeared.

Of late years, **inflation of the rectum with gas** has been practised (Runeberg, Ziemssen, Curschmann). This method of treatment is not new, and was even used by the physicians of antiquity. Everything that can be said for and against injections of water applies with equal force to inflation of the bowel with gas. This procedure, too, is most useful in certain cases of deep-seated invagination of the bowel. The advocates of gas inflation claim that this method is fully

as effectual as the injection of water, and, at the same time, less dangerous. Other authors again maintain exactly the opposite.

Inflation of the bowel with gas is performed by introducing a rectal tube into the rectum, connecting it with the air compressor, and pumping air into the bowel. In order to promote the escape of air as soon as it is desired, Curschmann advises inserting a T-tube between the rectal tube and the air-compressor. One limb of the T-tube is inserted into the rectal tube, the other one into the tube leading from the air compressor, and the third into a short piece of rubber tubing that can be opened or shut by a pinch-cock.

In connection with air inflation injections of carbonic acid gas may be mentioned. They can be given, on theoretic grounds, for the treatment of the same conditions as gas inflations and water injections; from a practical point of view approximately the same applies for and against them as in the case of water and gas inflation. Carbonic acid enemata are given by connecting a siphon with the rectal tube and allowing the carbonated water to run into the rectum. Another method of performing inflation of the bowel with carbonic acid gas is to administer first a solution of 20 grams of bicarbonate of soda, and then a solution of 15 grams of tartaric acid, repeating the dose alternately several times.

Formerly it was customary to inflate the rectum and bowel with tobacco smoke and to inject infusions of tobacco-leaves, but this treatment has now been entirely abandoned.

Massage.—Massage has long been employed in the treatment of this disease, and has been warmly recommended by many authors. We possess sufficient experience in regard to the efficacy of this treatment to enable us to form a fairly conclusive opinion as to its value. In all cases of strangulation of the bowel, in axial rotation and knotting, in internal incarceration, and also in kinking of the bowel, massage must be absolutely condemned, even in the hands of an expert masseur, while it is also contraindicated when there is the slightest probability of peritonitis or when the occlusion of the bowel has existed so long that the intestinal wall may be gangrenous or very fragile. It is always a difficult matter to determine when the intestine may be fragile or gangrenous, and it is well to remember that this condition may develop very early in the course of the disease. Massage has been successfully employed in a few cases of very recent invagination of the bowel, especially when the patient was placed under an anesthetic at the same time; but even in this condition great care is necessary. The intussusception tumor may often be felt to grow harder from contraction under the stimulus of palpation, and not to become reduced. At all events, injections of fluid into the rectum and inflation of the bowel with air or gas are more likely to do good than massage.

In obturation of the bowel by fecal accumulation careful massage performed by an expert manipulator may occasionally be of value. In this condition it may act in two ways: it may either mechanically lead to onward passage of the accumulated masses of fecal material, or it

may stimulate contraction in the muscle-fibers of the intestinal wall in cases of moderate muscular paresis.

A few cases are also on record in which it was possible to push obturating gall-stones and other foreign bodies forward by massage of the parts.

Electricity occupies approximately the same position in the treatment of occlusion of the bowels as massage ; in fact, the efficacy of electric treatment is less and its sphere of usefulness still more circumscribed ; nevertheless, many authors claim to have witnessed good results from the application of electricity. I do not believe that many medical men at the present day would risk the waste of valuable time involved by electric treatment, particularly in cases of occlusion of the bowel due to strangulation, axial rotation, or invagination ; in all cases of occlusion of the bowel, moreover, that are due to impaction of foreign bodies or obturation of the intestinal lumen by gall-stones electricity is absolutely useless. The only condition in which electricity might be of some value is in paresis of the muscular coat of the intestine, such as is occasionally seen in coprostasis. Here I believe that electricity, in combination with other suitable measures, as irrigation of the bowel and the administration of purgatives, may be of some slight value. A number of " cures of ileus with electricity " are on record, but a critical study of all these reports reveals that in the great majority of the cases occlusion was due to fecal accumulation.

It is customary to employ chiefly the faradic current and only rarely the galvanic current. The faradic current is applied in such a way that both electrodes are placed upon the abdomen. In other cases one of the electrodes—it does not matter which in applying galvanism— is placed upon the abdomen and the other one introduced into the rectum.

[Treves points out that it is difficult to see how electricity can do any good in acute strangulation, and that if it increases peristalsis, it may do harm.—ED.]

Ice, Priessnitz Compresses, and Warm Poultices.—All these different applications are frequently made to the abdomen, but are naturally quite incapable of influencing the course of a case of occlusion of the bowel in any way. They are only employed symptomatically in order to relieve pain. It is impossible to give definite directions as to the best temperature of the various compresses : the subjective sensations of the patient must determine this point. It is always well to consult the patient as to his personal comfort or discomfort, and to modify the temperature of the applications accordingly. This is fully justified, as the temperature itself exercises no influence whatever on the course of the process. It is possible that in cases of peritonitis cold applications should be preferred.

One method of treatment remains to be mentioned here which, strictly speaking, belongs to the field of surgery, but is commonly included among the so-called bloodless measures—viz., direct puncture of

the intestine. Puncture of the intestine allows the escape of gas from distended loops of bowel, and so reduces the intra-abdominal pressure in the same way as in siphonage of the stomach. It is clear that this reduction of the intra-abdominal pressure must be followed by all the favorable consequences that we have spoken of in different places, and consequently fulfils a very important indication in the treatment of occlusion of the bowels. The patients, as a rule, experience a considerable relief of their subjective symptoms; in addition, to judge from the reports of competent observers, genuine cures have followed this procedure, and must be regarded as analogous to the cure of occlusion after siphonage of the stomach. [A large number of cases will be found in J. W. Ogle's [1] monograph.—ED.]

Notwithstanding these favorable effects of gas puncture, there are serious objections against this procedure. The main danger, of course, is the possibility of peritonitis. It is true that in the great majority of cases puncture of the intestine is harmless. Occasionally, however, it does lead to the development of peritonitis, and produces local gangrene of the intestine around the point of puncture. Lastly, the point of puncture may not close completely, especially when the intestine is over-distended and paralyzed. In this way the entrance of gaseous or liquid bowel contents into the peritoneal cavity is permitted. Curschmann is particularly emphatic that puncture of the bowel should never be undertaken in cases of occlusion of the intestine in which symptoms of intestinal paresis, peritonitis, or gangrene of the intestine are threatening. Theoretically, this rule is excellent, but practically it is impossible to follow, inasmuch as these conditions frequently cannot be diagnosed, and it is consequently impossible to know when and when not to perform puncture. Naunyn and Graser, the most recent writers on this subject, therefore advise omitting direct puncture of the intestine in all cases in which the patient may possibly consent to a laparotomy, and to perform it only in those cases in which the patient positively refuses an operation. [Treves, after pointing out the haphazard nature of this proceeding and its dangers, agrees with the opinion expressed by the last-mentioned writers.—ED.]

Curschmann has described the best method of performing direct puncture of the intestine. A long, hollow needle of the same caliber as the needle of a Pravaz syringe is carefully disinfected, and the cock at one end closed. It is then rapidly inserted into the most distended loops of intestine. After the point of the needle has entered the intestine, the other end is connected with a rubber tube which leads into a bottle filled with a solution of salicylic acid. This bottle in its turn is inverted over a basin containing the same solution. Sometimes only very little gas will escape from one loop of intestine, so that puncture of the bowel must be repeated in different places. A very important point to remember is never to hold the needle tightly, but to allow it to follow the movements of the bowel.

[1] J. W. Ogle, *On the Relief of Excessive and Dangerous Tympanites by Puncture of the Abdomen,* 1888.

OPERATIVE TREATMENT.

I have already mentioned that about one-third of all the cases of occlusion of the bowel that are treated by drugs and mechanical measures—that is, without operative interference—recover. It is manifest, therefore, that the mortality is about from 65 to 70 per cent., a percentage which is so enormous that it urgently calls for the discovery of better methods of treatment. From the very nature of the problem operative interference is the only method that can be considered.

The advisability of the plan of opening the abdomen, finding the obstacle, and removing it is self-evident. As a matter of fact, the recognition of the true anatomic character of the different forms of occlusion of the bowel soon led to the adoption of this method. At first operative treatment, while it was regarded theoretically as the logical sequence of improved knowledge of the disease, was rarely performed in practice, inasmuch as the operation was highly dangerous before the era of aseptic surgery. At the present day the dangers from the operation proper are very much reduced, and the question arises whether or not the advocates of early laparotomy in all cases are not right after all. It might fitly be argued that it is wrong to jeopardize the life of the patient by not operating, for, as we have seen, the chances of recovery under bloodless treatment are only one-third; while we know that it is often possible to remove the obstruction by immediate operative interference.

If it were really true that the operation itself is without danger, that it never leads to fresh complications, and that, if undertaken under apparently favorable conditions, it would save the life of the patient with almost absolute certainty, these arguments would be valid, and it would be the duty of the physician immediately to give the patient the benefit of operative treatment. If the results of operative treatment were really so favorable, it would not be the duty of the physician, as I emphasized above, to try all bloodless measures before advising operation. Even if it were impossible in some instances to remove the obstruction by an early laparotomy, the patient, provided the operation itself were devoid of danger, would be in no worse position after operation than before.

We see, therefore, that it is very important to determine whether or not laparotomy is without danger and incapable of producing untoward effects and disagreeable consequences in cases of acute occlusion of the bowel. Unfortunately, experience shows that laparotomy performed in cases of occlusion of the bowel, particularly when symptoms of strangulation are fairly well developed, constitutes a much more serious measure than laparotomy performed for other causes in other conditions. Modern surgeons, it is true, can usually prevent septic infection and peritonitis, provided peritonitis has not developed as a direct result of the occlusion. Apart from the danger of septic infection and of peritonitis, however, there are other dangers in every laparotomy for occlusion of the bowel. These dangers are peculiar to this condition, and result from the necessary and unavoidable technical

manipulation of the bowel. Among the disagreeable and objective features that must be considered are the following: palpation and manipulation of loops of intestine which are rendered vulnerable by the morbid process, the great protrusion of large masses of intestine from the abdominal wound, and loss of heat from large portions of the exposed loops of intestine. Secondary consequences of all these factors are intestinal paresis, and, above all, reflex effects on the nervous system and the circulatory apparatus. All these possibilities must be taken into consideration; in fact, the effects produced by manipulation, exposure, and cooling of the bowels are frequently so serious that the operation *per se* may be as formidable a matter as the disease itself.

The dangers of surgical interference in occlusion of the bowel are chiefly influenced by one factor, which, at the same time, furnishes the main indication for performing laparotomy. This is shown by theoretic considerations and also by practical experience. The factor that I refer to is the following:

All observers agree that the results of laparotomy as regards finding and removing the obstruction are better when the operation is performed as early as possible in the course of the disease. The reasons for this are self-apparent. The collapse and shock always increase as the disease progresses and the longer the occlusion persists; the patients lose strength as a result of the morbid changes in the intestinal wall with their consequences, peritonitis begins, intra-abdominal pressure rises more and more, there is danger of paralysis and gangrene of the intestine, and the initial shock, which may possibly have been moderate and insignificant, gradually develops into a serious state of secondary collapse; in other words, the powers of resistance of the patient to surgical interference decrease *pari passu* with the duration of the disease. It is, of course, quite impossible to determine at what time in the disease these various dangerous factors will make their appearance. As each case varies, special consideration must be given to each, but, generally speaking, it may be stated that the longer the occlusion persists, the greater the danger of operation.

On this basis we can readily understand Naunyn's statistics in regard to the results of operative treatment of occlusion of the bowel. Naunyn collected 288 cases which were operated upon, and found that the best results were obtained when the operation was performed within the first or second day after the onset of the occlusion. The percentage of cures among those operated upon during the first two days was 75; among those operated on the third day the percentage of cures was very much lower—about 35 to 45; the percentage of cures in cases operated upon after the third day was about the same, even when the operation was performed as late as the second week or later still.

The dangers of operative interference can hardly be altogether avoided, even in the first two days, but we may say that the dangers increase in proportion to the duration of the occlusion.

Against the fact that with every passing hour the chances of successful operation are diminished, all other arguments must fail. One

circumstance which greatly influences the prognosis in cases that are operated upon is the local diagnosis. When the seat of the occlusion can be made out, and the surgical technic can be so arranged as to reach and remove the cause of the obstruction at once and without manipulation of the intestine, the prognosis is better. In those cases where the seat of the lesion, and possibly its nature, are not clear, the surgeon is necessarily in the dark as to the technic to be pursued and the part of the abdomen to be reached. Exploratory laparotomy, therefore, in the absence of any positive diagnosis, is a much more dangerous procedure than laparotomy for the removal of a definite lesion in a definite portion of the intestinal tract. The chances of success from laparotomy are greater when the physician is able to localize the occlusion in this or that portion of the bowel, and can settle on its nature before operation. One might think that a localizing diagnosis could be made better by examining the patient repeatedly, and that for this reason operation might well be delayed somewhat. But as a matter of fact, the diagnostic advantages thus gained are insignificant when compared with the serious dangers that such delay inevitably adds to the operation. Moreover, from personal experience, I have found that if a comparatively accurate localization is not possible at the first examination, it will be even less likely at subsequent ones, chiefly on account of the increasing meteorism. For the fulfilment of the first indication in intestinal occlusion, as given by Kocher, namely, evacuation of the intestine above the point of occlusion, no precise diagnostic localization need be made.

We repeat : Success of operation increases in proportion to the early date at which it is undertaken. The view taken by most surgeons seems to me as correct : the high mortality of operation does not justify delaying it ; on the contrary, it should induce earlier operation, particularly in acute cases with symptoms of strangulation. All forms do not demand laparotomy equally. Further on, the individual cases will be specially discussed in this connection, and here we shall remark in a general way as follows : We must proceed somewhat expectantly when obturation from gall-stones and foreign bodies is recognized ; when a diagnosis of kinking is rendered very probable by only moderate intensity of the symptoms, and when there is fecal accumulation. All other forms of occlusion—and they constitute the great majority of the cases met with—conform to the rule given above.

Now, how is the question of operation to be settled early in the course or in the first few days of the disease when the diagnosis is quite obscure, all that is known being that the patient is not suffering from peritonitis, though it is assumed that there is an acute occlusion of the bowel, without, however, anything being known as to the anatomic character or position of the occlusion of the intestine ? A situation of this kind constitutes the horns of a dilemma, for the early stage of the disease is in favor of operation, while the obscurity of the diagnosis, on the other hand, makes operative interference undesirable. What are we to do ?

Under conditions of this kind the cases of occlusion must be divided into two great categories according to the general symptom-complex and clinical course of the disease.

In one category we have cases that run a moderate course, and in which the general clinical picture is not excessively severe, symptoms of strangulation not greatly developed, those of occlusion dominating the situation. In cases of this character we must pursue the following train of reasoning: Acute occlusion of the bowels is not a disease that is invariably and unavoidably fatal. A certain proportion of the cases, even though they seem to be exceedingly grave, occasionally recover on internal treatment; on the other hand, laparotomy, especially when it is undertaken without any very definite evidence as to the position and the character of the occlusion, is often a very dangerous remedy, and one that may precipitate the fatal issue. Schönborn expresses himself in regard to the difficulties of the situation, and particularly in regard to the advisability of delaying operation under these conditions, as follows: "Supposing the case were that of your own child, or supposing that some person who was very near to you developed symptoms of this kind; would you feel justified in performing a laparotomy within the first twenty-four hours even though this operation would be comparatively less dangerous at this period than later in the disease?"

In these cases an attempt should certainly be made to treat the patient with so-called internal, bloodless measures. This treatment, however, should be carried out according to definite indications, rapidly and consistently. Such measures as siphonage of the stomach, the administration of opiates, and possibly inflation of the bowel with air and injection of fluid into the rectum should be first employed.

If this form of treatment is decided on, the patient should be most carefully watched by the physician, who should constantly be on his guard. The action of the heart, and the character and rate of the pulse, should be observed with the greatest care, for the character of the heart and the pulse frequently indicate when it becomes necessary to call in the surgeon. There is no better or more reliable guide to the correct time at which an operation becomes necessary than the state of the heart, which, of course, is shown by the quality of the pulse. The rapidity, the tension, the volume, and the fulness of the pulse must all be considered. When the pulse-rate is not too quick and the tension, fulness, and volume are fair, the prognosis as to the results of an operation is comparatively favorable, so that under these conditions, if an operation is decided upon, the chances of success are better. As soon as the pulse-rate begins to increase and the tension to decrease even slightly, no time should be lost in performing the operation if the patient's life is to be saved.

Stress must again be laid on the great importance of making a differential diagnosis between the symptoms of strangulation and the symptoms of occlusion in the decision whether operation is necessary. Symptoms of the latter conditions, such as great distention of the abdomen and fecal vomiting, may be present, and yet the case be not particularly

urgent; when, on the other hand, symptoms of strangulation are fully developed, as manifested by the general appearance of the patient and by symptoms of collapse, particularly by changes in the heart's action, no time should be lost. It may be considered a general rule that under the latter conditions operative interference is immediately called for.

One other point may be mentioned here which is of no direct scientific or technical value, but is more of psychologic interest; this factor may, however, under certain circumstances, exert a considerable influence on the course of the disease and the termination of the case. Every medical man who is confronted with a critical situation of this kind should always call the attention of the patient and of his friends to the possible necessity of performing an operation and the probable outcome of such a serious measure. If he waits too long before informing the patient and his friends of the necessity of performing an operation, much valuable time may be lost at a critical moment, because naturally the patient is suddenly surprised and may be frightened, and will require some time to overcome the natural hesitancy he will feel to so serious a method of treatment.

If, after due consideration of all the circumstances of the case, an operation is decided upon, the difficult question remains to be decided, in what way to operate. The exact decision of this question must be left to the surgeon, but I may be allowed to make the following remarks here. Many surgeons of wide experience in this subject (to mention among many others von Bergmann, Czerny, Mikulicz, Schede, Schönborn, and Kocher) are very cautious in regard to performing a radical laparotomy for the purpose of finding and removing the obstruction in any case of occlusion of the bowel in which the diagnosis is more or less obscure. Some advise that the operation should be limited to enterostomy—in other words, to the production of an artificial anus. This, they claim, should be the immediate object of the operation, and should be performed as soon as the abdomen is opened without attempting to look for the affected segment of the bowel. If, on opening the abdomen, the obstruction can be discovered without trouble, there is, of course, no reason why the occluded spot in the intestine should not at once be treated *secundum artem*. But, as Curschmann says, "if a small or medium-sized incision is made through the abdominal wall in the linea alba as near as possible to the suspected seat of the obstruction, and if, after a short and careful palpation of the intestine through this wound and without allowing the bowel to prolapse, the affected spot cannot be discovered," enterostomy and the formation of an artificial anus should always be attempted. The artificial anus should be made by sewing the nearest loop of distended intestine to the abdominal wound. Many surgeons call attention to the fact, moreover, that the formation of an artificial anus occasionally directly leads to a cure of the disease. Whenever this occurs, it may be attributed to the reduction in the intra-abdominal pressure and the release of tension in the neighborhood of the occlusion.

In the second category the diagnosis is quite uncertain and the cases

run an extremely acute course, with most severe symptoms of shock at the onset. Here the patients rapidly become collapsed, the pulse is small and rapid, and violent reflex vomiting appears. Are these cases to be operated upon or not? Many surgeons are opposed to operation, owing to the collapse. From the standpoint of the physician, however, we can only agree with those who would advise immediate laparotomy followed by a search for the obstruction. If necessary, of course, a brief attempt may be made to relieve the symptoms and to release the occlusion with morphin and washing out the stomach. If the administration of morphin does not improve the symptoms of collapse, a laparotomy is indicated.

Cases of this kind, as we know, almost invariably prove fatal unless an operation is performed, and therefore every patient of this kind who recovers after an operation may be regarded as saved by it. I do not think that the shock can really be considered a contraindication for operation, for, as Schede correctly remarks, patients who succumb to very acute attacks of occlusion of the bowels never come out of the state of collapse at all. For this reason not to operate is naturally the same as abandoning the patient to his fate without any attempt to save him ; for the shock is the direct result of the occlusion of the bowel itself (axial rotation, internal strangulation), and the mechanism responsible for the occlusion, and also for the shock, can be relieved only by operation. In this way shock is not only not produced, but, on the contrary, is relieved by surgical interference.

Finally, the question of operation must be considered in cases in which the occlusion has persisted for several days. Here again two factors must be taken into account in deciding as to the advisability of surgical interference ; in the first place, the diagnosis of the anatomic character of the occlusion ; in the second place, the general state of the patient. As regards the latter indication, the reader should refer to the rules formulated above ; here it may merely be insisted on that the action of the heart is specially important in this respect. All that has been said above as regards the prognosis of operative treatment and the necessity for operation in different anatomic forms of occlusion of the bowel applies with equal force to cases of occlusion of some days' duration. These details will be referred to in a subsequent paragraph.

In many patients who would have recovered without difficulty from a laparotomy if it had been performed early in the disease such a severe measure is quite out of the question at this period. They are so much exhausted by pain, vomiting, want of nourishment and sleep, and occasionally by autointoxication, that the extensive surgical procedure, with the prolonged manipulation of the intestine that is necessary, may directly precipitate a fatal issue. The production of an artificial anus is probably all they can bear in their exhausted condition. It may, therefore, be very difficult to decide in cases of this kind whether to perform laparotomy or to make an artificial anus.

The presence or absence of peritonitis must also to some extent influence our decision as to the advisability of an operation. Views

on this matter have of recent years undergone decided change. Formerly the majority of surgeons regarded any operation in cases complicated by general peritonitis as a mistake, for they claimed that the prospects of success were infinitesimal. Here and there, it is true, isolated cases are reported which recover even though peritonitis existed; but under these circumstances an operation must be considered an extremely risky procedure. To-day diffuse peritonitis is occasionally treated by immediate operation, and not only that, but Kocher, for example, advocates the same step for cases in the first stage of which a certain differentiation between occlusion and peritonitis cannot be made, his ground being that the lack of differential diagnosis constitutes no bar to operation.

When the peritonitis is local and mild in degree, the results of surgical interference are often somewhat more favorable. Operation has been advocated more extensively by surgeons, chiefly on the grounds that slight degrees of local peritonitis are rarely diagnosed before the operation is undertaken.

[Treves says there was a time when the existence of peritonitis was supposed to contraindicate any operative interference in acute intestinal obstruction. At the present day (1899) the question of peritonitis enters but very little into the problem. In really extensive peritonitis of the usual low type the case is practically hopeless with or without operation, the condition of the patient being probably one of rapidly increasing septicemia. Operation, if declined as hopeless, would be declined not on account of peritonitis, but on account of the deplorable condition of the patient. When peritonitis is present, the more brisk and active its manifestations, the brighter are the prospects of operation. The least satisfactory cases are those associated with that low form of general peritonitis which presents but feeble manifestations and which is a more or less inevitable feature in the last stages of any case of fatal intestinal obstruction.

Lockwood [1] has paid special attention to the operative treatment of diffuse septic peritonitis; many cases due to perforation of the appendix, intestine, or stomach resemble cases of mechanical obstruction.—Ed.]

TREATMENT OF STENOSIS OF THE INTESTINE.

In cases of stenosis of the bowel that run a gradually progressive course, treatment may be purely symptomatic from the time the diagnosis is made, and may be directed merely toward the removal of certain symptoms and the prevention of certain complications. On the other hand, treatment may be radical and directed toward the complete removal of the obstruction.

Symptomatic treatment must be directed, in the first place, toward the relief of the symptoms that appear in the intervals between the various paroxysms of colic; in the second place, toward the relief of the

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[1] Lockwood, "Appendicitis, its Pathology and Surgery," *Medico-Chir. Trans.*, 1901, vol. lxxviii., p. i.

attacks of colic themselves, and, finally, against the symptoms due to occlusion of the bowel which may supervene at any time in chronic enterostenosis.

The therapeutic indications in the intervals between the attacks of colic are simple and clear. Everything must be avoided that may lead to overdistention of the intestine and in this way produce complete occlusion of the bowel. This postulate is fulfiled, as far as it is possible to fulfil it, by regulating the diet and carefully supervising the evacuation of the bowels.

The diet should be regulated with the most minute care. One of the fundamental rules is never to allow the patients to take too much food at one time; it is much better to let them eat small quantities at frequent intervals. The choice of the different articles of diet—that is, the quality of the food—is more important still than the quantity. In the first place, care must be taken that the patient really takes sufficient nourishment. At the same time, all unnecessary and injurious material should be avoided, and all articles of diet should be prohibited which might mechanically obstruct the stenosed area by forming an unyielding obturator in the narrowed portion of the bowel. Among such articles of diet I may mention large pieces of meat containing much fibrous and tendinous material, black bread, pieces of potato, hard-boiled rice, vegetables containing much cellulose, and fruit of all kind, especially the peel and the seeds. From this point of view the different varieties of food may be classified and the method of preparation carefully determined. It is best to avoid all articles of diet that are not pultaceous, finely divided, and finely chopped up. If the stenosis is far advanced, liquid or semiliquid food should only be given. Ultimately it may be necessary to give nothing by the mouth and to nourish the patient *per rectum.*

A careful examination of the motions is of use chiefly in stenosis of the large intestine, and is less important or of no value at all in stenosis of the small intestine. The reasons for this are apparent. Constipation should never be allowed to develop, and care should be taken that the patient has a daily evacuation of the bowels. If it is necessary to produce this result by artificial means, a rectal injection, preferably with simple water, or, if necessary, with water to which some irritating substance (salt, vinegar, soap, infusion of senna) has been added, may be employed. In some patients injections of large quantities of oil are more efficacious. Finally, some internal purgatives may be given, preferably one of the mineral waters or salts. If the stomach can tolerate such preparations as castor oil, cascara, or rhamnus frangula [buckthorn], or, in very obstinate cases, colocynth, these drugs may be administered.

When paroxysms of colic develop, all food should be stopped at once, as the quantity of material above the obstacle is merely increased by eating. It is not difficult to enforce this regulation, since, as a rule, the patients refuse all food during the paroxysms of colicky pain.

In stenosis of the large intestine large occluding masses are best

removed by rectal irrigation, as already described. In occlusion of the small intestine only, internal medication may be used, for which the saline and senna preparations are best adapted.

In all cases of temporary occlusion of the bowel, supervening in the course of stenosis of the intestine, which are characterized by the presence of very violent borborygmi or by visible peristaltic movements and visible tetanic stiffening of certain loops of intestine, rectal irrigation alone should be given and all purgatives carefully avoided, for under these conditions the signs developed show that intestinal contractions are being carried out to the utmost limit of the muscular power of the bowel-wall. In a case of this kind purgatives can be of no use whatever; on the contrary, their administration would produce overirritation of the intestinal muscular coat, and in this way lead to fatigue and paresis of these important muscle-fibers. Under the conditions outlined, therefore, opiates, and not purgatives, should be given in addition to rectal injections.

If the attack of colic is relieved by these measures, and if such attacks of colic are frequently repeated, it becomes the duty of the medical man to call the attention of the patient to the serious character of his ailment and to prepare him for the possible necessity of radical operative interference. The exact indications for operation in this disease will be dealt with below.

Complete occlusion of the bowel supervening on chronic progressive stenosis of the intestine may, as I have shown above, be due to a great variety of causes. In some instances the stenotic segment of the bowel is still fairly patent, and only becomes temporarily occluded by some compact mass contained in the bowel contents. In other cases the anatomic process producing the stenosis develops to such a degree that the condition created is practically the same as anatomic occlusion of the bowel; in other instances, again, the wall of that section of the intestine immediately above the stenosis becomes paretic, or, finally, the loops of intestine situated above the obstacle become so stuffed and distended and weighted with bowel contents that they drop down and lead to kinking of certain loops. In cases of stenosis of the sigmoid flexure overfilling and overloading of the intestine may lead to volvulus.

This enumeration of the different possibilities of occlusion alone shows that, with the exception of the last-named condition, in which symptoms of strangulation may be produced, the symptoms developed are usually due to simple uncomplicated occlusion of the intestinal lumen.

The course of the disease in these cases, therefore, is less rapid and less alarming. The indications for treatment in all these different cases also naturally vary greatly. If the syndrome of acute kinking or axial rotation of the bowel is pronounced, we should proceed according to the same fundamental principles that we have enunciated above when discussing the treatment of acute occlusion of the intestine in general. In a subsequent paragraph some of these methods will be considered more in detail. An operation can hardly ever be avoided, for there is very

little reason to hope that the intestine will spontaneously straighten out after it has once become kinked, or that it will rotate back into its normal position after it has once formed a volvulus. Occasionally, it is true, as we know from experience, the intestine does seem to become spontaneously patent again even after kinking has occurred.

If complete occlusion of the bowel is the result of the three other conditions that I have mentioned, bloodless measures should always be attempted at first. It is true that really good results can hardly be expected from this method of treatment in cases in which organic stenosis of the bowel has gradually progressed to mechanical occlusion. But in view of the fact that it is almost impossible to differentiate this form from the other possible forms of occlusion, and as no symptoms of strangulation ever develop, there is plenty of time ; from this point of view alone we should always attempt bloodless methods before proceeding to operative interference. Internal treatment of this condition consists in nothing more nor less than in instituting the different measures and administering those remedies which have already been mentioned in the account of the treatment of colic. In other words, various kinds of rectal enemata and purgatives should be given. In addition, the use of electricity and massage may be permitted under certain circumstances, especially when there is reason to suspect that there is insufficiency of the intestine, or to believe that mechanical propulsion onward of the bowel contents may lead to the desired result.

[According to Crämer,[1] massage should not be employed when there is any reason to suspect carcinomatous stricture, as it may favor the production of secondary growths.—Ed.]

Other and very important measures which may be employed are washing out the stomach and possibly direct puncture of the intestine.

All these bloodless measures ought, of course, only to be instituted when we have a clear conception of the anatomic lesions present and of the functional disturbances we are studying. Internal treatment, moreover, should be directed toward the relief of certain definite conditions and should be carried out with energy. The time, moreover, during which this treatment should be given is limited. It is not so limited, it is true, as in cases of acute strangulated occlusion of the bowel, but should, at the same time, never be continued beyond a certain time, for in this form of occlusion there is always danger of paralysis of the intestinal musculature from stasis of the contents and overexertion and fatigue. In addition, paralysis of the bowel may be brought about by meteorism from stasis, and, finally, the possibility of ulceration of the intestinal wall above the stenotic area and of perforation of the intestine must always be thought of. Under all circumstances operative interference should be instituted as soon as the symptoms of stasis in the intestine begin to assume a dangerous character or as soon as the action of the heart begins to grow weak. Generally speaking, it is always better to perform an operation before these complications appear.

The object of the operation is either to open the intestinal passage

[1] Crämer, *Münch. med. Wochenschr.*, June 17, 1902.

temporarily by means of an artificial anus, or radically to remove or circumvent the obstruction. It is a very difficult matter to determine which of these two operations should be performed in any given case, and it is impossible to formulate any general rules as to the advisability of the one or the other operation in any concrete case.

If the site of the obstruction is quite obscure, and if there is no evidence whatever on this point, it will probably be best in all cases merely to perform an enterostomy, the main object of this surgical measure being to remove the imminent danger and to save the life of the patient. In other instances the condition may be so clear and the diagnosis so positive that radical removal of the obstruction may be attempted at once—for instance, when a carcinoma in the cecum, forming a tumor that is distinctly palpable and circumscribed, is present. If such a swelling causes a constriction of the cecum that ultimately progresses to occlusion of the bowel lumen, the tumor may be removed by radical operation as soon as symptoms of complete occlusion develop. Occasionally, of course, it may happen that on opening the abdominal cavity certain unexpected conditions and complications are discovered (adhesions, metastases, etc.) which make it impossible to carry out the original plan of performing a radical operation and lead us to be content with an artificial anus.

Radical treatment of stenosis of the bowels can be performed only by surgical means. Occasionally, as, for instance, in fecal accumulation and in some rare instances of compression of the bowel from without, certain internal measures may be tried. The operation usually consists either in removal of the anatomic obstruction which produces the stenosis of the bowel lumen, or in performing an intestinal anastomosis around the obstructed area. No internal remedy and no other measure known at present can exert any effect upon a cicatricial stricture, constricting peritoneal adhesions, or carcinoma producing stricture of the bowel. It is perfectly futile to attempt removal of such primary causes of stenosis by internal and bloodless measures. The only efficient method of treatment is surgical.

In one of the preceding paragraphs attention was called to the fact that in chronic progressive stenosis of the intestine the conditions for operative interference, in a certain sense, are generally particularly favorable. It was shown that the medical man can usually select the exact time at which the operation should be performed, and is not hurried in his decision as he is in cases of acute occlusion. In other words, he can select a period in the disease in which the general health of the patient is good, and is also frequently able to advise operation between two attacks of occlusion—that is, at a time when the bowel lumen is not actually occluded. The most important question, therefore, to answer is, At what period of the disease, provided the diagnosis of stenosis of the intestine has been positively made, should a radical operation be undertaken? On the other hand, At what period of the disease, provided the diagnosis is not quite positive, should an exploratory laparotomy be advised? A great many different factors deter-

mine this decision. Some of them are purely medical, while others are surgical and technical, so that it is very difficult to formulate any general rules as regards this question. I must limit myself, therefore, to a brief sketch of some of the most important points that determine this decision.

When it is known that the case is one of carcinoma, the operation should be performed as early as possible. In this disease delay means aggravation, and the conditions for operation become less favorable as the disease progresses. The patient continuously loses strength, the neoplasm continues to increase in size, the necessary technical manipulations are rendered more difficult, and, finally, there is always danger of the development of metastatic growths in other organs. For all these reasons it is the medical man's duty in a case of carcinoma to call the patient's attention to the necessity of early surgical interference.

In cases of cicatricial stricture and constriction of the bowel due to adhesions the conditions are somewhat different. Many of the factors that are operative in carcinoma are not present in this form of constriction. In cicatricial stenosis and stricture of the intestine by adhesions the time for operation is determined chiefly by the degree of stenosis and the severity of the symptoms that are produced by the constriction of the bowel. If the symptoms are very severe, distressing, and dangerous, the patient should be advised to submit to operation as soon as possible, otherwise there is no immediate urgency for surgical interference. One of the chief differences between this form of stricture and carcinoma of the intestine is that in the latter disease there may sometimes be a recurrence of the disease, whereas in the former category this can hardly be anticipated; consequently the prognosis of operative interference in cicatricial stenosis and constriction of the intestine by adhesions is, as a rule, very good. At the same time I cannot refrain from emphasizing that I have occasionally witnessed cases in which the operation terminated fatally, even though all the chances of recovery were good and a good prognosis had been made. A large proportion of the cases, however, are operated upon with very good results. The few unfortunate results that I have seen, nevertheless, determine me to delay operative interference in cases of chronic stenosis of the intestine which are certainly not due to carcinoma. In the latter disease, as I have repeatedly said, early interference is always indicated. In the other forms, however, I delay operation until the condition of the patient positively forces me to advise surgical interference, or until dangerous or very distressing complications arise—as, for instance, unbearable pain and repeated attacks of occlusion of the bowel. My reason for taking this conservative view of the question is that I know from experience that the most conscientious, skilled, and experienced surgeon can never guarantee the results of this operation. The surgeon, of course, always knows where to begin the operation, but he never knows where he will end it. The surgeon may begin the operation expecting to find simple conditions, and on opening the abdomen may come upon a mass of adhesions; he may expect to find only one portion of the bowel

stenosed, and on examination find that it is narrowed in four to six different places. Further, the sequelæ of operation, such as paralysis of the intestine, etc., must all be included in the prognosis.

It is beyond the scope of this work to enter into a description of the operative technic that must be followed in performing the different operations that I have spoken of.

REVIEW OF THE TREATMENT IN THE DIFFERENT FORMS OF OCCLUSION AND STENOSIS OF THE INTESTINE.

In the preceding paragraphs I have described, in a general way, the treatment of all those cases in which no positive diagnosis as to the nature and the seat of the occlusion can be made, or in which, at best, only an uncertain and doubtful diagnosis can be arrived at. In this section I wish briefly to summarize the treatment in cases in which the diagnosis is certain, or at least highly probable.

It need hardly be specially insisted on that all the principles laid down and all the rules formulated in the general part of this section naturally also apply to all the special cases that may be met with. The diagnosis of internal strangulation of the bowel is probably never made with absolute certainty. The clinical picture of this condition may be produced by so many different anatomic conditions—many of which are impossible to diagnose during life—that the recognition of the true state of affairs is almost without exception impossible. I need only mention some of the forms of internal strangulation of the bowel and the anatomic causes of this lesion to show that it may be almost impossible to determine with any degree of certainty what the anatomic lesion is that produces the disease. What evidence, for instance, is there before an operation or before a postmortem examination that the bowel is strangulated by a Meckel's diverticulum, by an adherent vermiform appendix or an adherent Fallopian tube, by peritoneal cords or false ligaments, that it is compressed by the mesentery, or that certain portions of the bowel are kinked over peritoneal bands, or, finally, that the bowel is strangulated in abnormal fissures and holes in the abdominal cavity?

Occasionally, however, it is possible to diagnose the existence of internal herniaform occlusion of the bowel with a certain degree of probability; in fact, it may even be possible to determine that this internal herniaform strangulation of the intestine is due to some particular anatomic abnormality that we can recognize, as, for instance, when occlusion of the bowel occurs in cases of old external hernia or in patients with a history of old chronic peritonitis (pelvic peritonitis, perityphlitis, trauma, etc.). Again, in other cases a diagnosis may be made by excluding all other possible forms of acute occlusion, so that occasionally in this way the diagnosis of acute herniaform incarceration of the intestine may be arrived at.

In such cases immediate operation is imperatively demanded, and it is only when external conditions render prompt surgical action impossible, that gastric irrigation and opium are indicated. Operation should take the form of a laparotomy. An enterostomy should be advised

only as a last resort in those cases in which the patient is so much exhausted that he cannot stand a laparotomy. Enterostomy may also become necessary when an exploratory laparotomy fails to find the obstruction. Laparotomy is indicated both on theoretic grounds and on the basis of practical experience. R. von Oettingen has collected a number of cases of enterostomy performed for herniaform internal strangulation of the bowel. In his statistics there is not a single case in which enterostomy acted as a palliative, and only one case in which a curative effect is attributed to this operation. Even though a case occasionally occurs in which the obstruction spontaneously disappears after enterostomy, this does not modify the statement that I feel justified in making, from my own personal experience and from that of many other clinicians, that laparotomy followed by a search for the obstacle is by far the better operation of the two.

The results of laparotomy are relatively favorable when the seat of the obstruction is known and an operation with the view of removing it can be carried out. Naunyn has collected many such cases from the literature, and it is interesting to note, from his statistics, that 72 per cent. of the cases of acute occlusion occurring in subjects with external hernias recovered after laparotomy (28 recoveries among 39 laparotomies). These statistics, of course, do not apply to patients in whom occlusion of the bowel occurred as the result of strangulation of the intestine in an external hernia, but to cases of internal strangulation, either under peritoneal cords or under adhesions that had formed as a direct result of the external hernia. In estimating the value of these statistics it must, of course, not be forgotten that in a large proportion of the cases reported as cured the patients were not really suffering from internal strangulation of the bowel, but merely from kinking of the intestine.

It appears that occlusion of the intestinal lumen due to kinking of the bowel may occasionally be relieved by internal measures—in cases, of course, in which the kinking is very severe and well-developed bloodless measures are probably futile. I believe that many cases of occlusion of the intestine that are reported cured without an operation were due to incipient or slight kinking of the bowel. The reason for this is probably that in twisting of the intestine the symptoms of occlusion of the intestinal lumen are more conspicuous than the symptoms of strangulation, consequently under these conditions the course of the disease is less violent and the good effects obtained from unloading the intestine, by siphonage of the stomach, for instance, become apparent at once. In view of the fact, however, that kinking of the bowel is usually due to the same processes and anatomic conditions which produce internal herniaform strangulation of the bowel, and as in many of these cases exactly the same history is obtained, the differential diagnosis between kinking of the bowel and internal herniaform strangulation is often very difficult or even impossible; consequently the treatment of kinking of the bowel should in general be carried out according to the same principles as the treatment of internal herniaform strangulation, possibly with this difference, that in cases of sus-

pected kinking of the bowel in which the symptoms of strangulation are either absent or slightly marked bloodless measures may be attempted for a somewhat longer period than in internal strangulation of the bowel. One argument in favor of operative interference, particularly in kinking of the bowel, is the fact that by surgical means the primary cause of the kinking (a peritoneal cord or the like) can readily be removed, and thus any further recurrence of the occlusion of the bowel is permanently prevented.

The statistics of von Oettingen show that in twisting of the bowel the results of enterostomy are approximately the same as the results of laparotomy, whereas, as we have seen, this is not the case in internal strangulation nor in axial rotation of the intestine. It will probably always be impossible to decide on the exact method of operation until the abdomen is opened and the true condition of affairs is recognized.

The views as regards the treatment of volvulus vary greatly. Some observers, particularly, of recent years, Naunyn, believe that the physician and surgeon should consider every case of volvulus on its merits and that the operation should not be hurried unless pronounced symptoms of strangulation urgently call for operative interference. Those who adhere to this view claim that in many cases of axial rotation recovery is possible without operative interference; on the other hand, they call attention to the fact that the results of operative interference in volvulus are quite unfavorable (of 30 patients who were operated for volvulus, 19 died). Another argument against early operation is the frequent complication of volvulus of the sigmoid flexure with neoplasms, or, as we may say, the frequent production of volvulus by neoplastic growths. The best plan, therefore, according to these authors, is to proceed with caution and to delay operation in cases that run a mild course. On the other hand, they advise immediate operation as soon as violent symptoms of strangulation appear.

These considerations are not without value, and the point of view adopted by some of these authors is not irrational. We must never forget, however, that they base their opinions in part on cases of volvulus which were not clearly diagnosed during life, and in which the diagnosis was merely corroborated by the subsequent course of the disease. I maintain that in all cases in which the diagnosis is clear, in which the presence of acute volvulus can be positively demonstrated,—and these are precisely the cases we are discussing,—an operation is always indicated. I feel justified in enunciating this view on the basis of my own experience and on that of the majority of writers on the subject, and I maintain that an operation should be performed not only when the course of the disease is violent and rapid, but even when the symptoms are moderate in severity. Volvulus of the intestine is a surgical disease, and when it has been definitely diagnosed, the case should be handed over to the surgeon. Unfortunately, even with surgical treatment, volvulus shows most unfavorable results.

The statistics as to enterostomy in cases of this character are so absolutely unfavorable that laparotomy is probably the only operation

which should be considered where there is time to plan the operation. In sudden emergencies, and as a last resort when it is quite impossible to reduce the rotated segments of the intestine, enterostomy may be permissible.

In cases of intussusception of the bowel, provided the diagnosis is positively established, all the bloodless measures detailed above should be attempted in rapid succession. At first opiates should be administered, for in this way the peristaltic movements of the bowel can be arrested and the further development of the invagination be prevented. Water should be injected into the rectum, and the bowel should be inflated with gas. Both these procedures are said to aid in reducing the intussusception. Clysters of salt water, of the strength of 5 to 8 per cent., may also be injected for the same purpose. In very recent cases an attempt to reduce the invaginated bowel has several times been made by performing massage of the intestine through the abdominal walls, while a few clinicians have also attempted faradization through the abdominal wall. In cases in which the intussusception advances as far as the rectum, an attempt to replace the bowel has been made by introducing the finger into the rectum or by using sponge-sounds. When the symptoms of stasis of bowel contents are very pronounced, siphonage of the stomach should never be omitted.

In a large proportion of cases, particularly in adults (in invagination of the bowel occurring in early childhood the results are much more unfavorable), the internal measures that I have described, either singly or together, have been known to produce good results. In a much larger proportion of the cases, however, they were quite valueless.

In determining the exact period of the disease in which these different methods of internal treatment should be applied in invagination of the bowel, other points of view and other considerations are often adopted than in either form of acute occlusion of the bowel. In the first place, many writers always bear in mind that the occlusion of the bowel is not necessarily complete, and that the symptoms of occlusion, apart from the pain, are usually mild and benign to begin with, and that there is always the possibility of spontaneous cure of the intussusception. In fact, as gangrenous sloughing of the intussusceptum, followed by recovery, is comparatively frequent, this possibility must also be considered. Finally, it is well known that invagination of the bowel frequently becomes chronic. All these considerations, however, are based on fundamental fallacies and false reasonings, as will be seen from a perusal of the paragraphs on the course of the invagination of the bowel in another section of this work, which need not be repeated here.

In cases of acute invagination of the bowel, provided internal therapy and all the internal bloodless measures fail to produce the desired result within a short time, operative interference should be undertaken as soon as possible. This is the consensus of opinion of those who have had most experience in the treatment of the disease in the last decade and a half, since operation, even in earliest childhood, has become more and more frequent. Even when the symptoms of

occlusion are comparatively mild, it is best not to wait too long, for each day that is allowed to elapse without an operation renders the possibility of manual reduction of the intussusception more difficult. This method of manual disinvagination should be attempted immediately after the laparotomy is performed. According to Rydygier, this treatment should be preferred to all other measures. A *sine qua non* for success, however, is the absence of peritoneal adhesions. In acute invagination of the bowel peritoneal adhesions occur in the great majority of cases, particularly when the disease is allowed to run its course without active interference for several days. In some instances no peritoneal adhesions will be found in the early stages, and quite frequently a considerable period of time may elapse before they begin to form. If reduction of the invagination is not feasible, resection must be performed.

In chronic invagination of the bowel it is permissible to wait somewhat longer before undertaking an operation, but even in this condition the employment of internal measures and of bloodless manipulations should not be persisted in for too long a time, for we must never forget that even these manipulative methods of treatment, as well as irrigation of the rectum or the stomach, etc., possess elements of danger in themselves in all those cases, particularly when there is ulceration near the neck of the invagination. In cases of the latter kind we can readily imagine how the insufflation of air under pressure or large rectal enemata may produce rupture of the intestine.

When gall-stones become impacted in the intestine and occlude the bowel, the prospects of spontaneous recovery are by no means unfavorable. As the symptoms are not particularly violent in this form of occlusion, we often feel induced to use bloodless measures and internal treatment for some time. The chief method to be employed, and the one which produces the best results, is the administration of preparations of opium, which in recent years has become a rival of the treatment with atropin. In very many cases the most serious features of the clinical picture are the violent pain, due to reflex spastic contractions of the intestine and general reflex collapse, which can be relieved by opium. Purgatives are not indicated as much as is opium, while rectal injections of water are, of course, useless when the stone is impacted above the ileocecal valve. An attempt to push the calculus forward in the intestine through the abdominal wall is to be deprecated, because the stone may very readily produce inflammatory and ulcerative changes in the bowel-wall and rupture of the intestine as soon as it is forcibly moved about.

Laparotomy should be decided upon only in cases of gall-stone occlusion when symptoms of such severity develop that the life of the patient is endangered. A number of factors should determine us in being very conservative in advising laparotomy in this disease; in the first place, the diagnosis is very difficult, particularly when the stone remains impacted high up in the small intestine, and it may easily be confused with a severe but simple attack of gall-stone colic. In addition, the intensity of the process frequently varies in this disease, so that it is difficult to arrive at a positive decision in regard to the best

time for operation. Finally, the results of operative interference in this disease are not particularly encouraging, for, according to Naunyn's statistics, 16 out of 23 cases of gall-stone occlusion operated upon died, or a mortality of 70 per cent.

[Jonathan Hutchinson, Sr.,[1] has advocated a policy of non-interference by surgical means and has urged the use of anesthetics, opium, and rectal injections with air or fluid to diminish spasm and assist in the passage of the calculus. In an early stage, Mayo Robson[2] advises morphin to relieve the pain, and extract of belladonna, $\frac{1}{4}$ grain every four hours, and chloroform anesthesia, which, by enabling a thorough examination of the abdomen, may clear up the diagnosis. If no relief is obtained, operation should be undertaken, and, as the disease is in an early stage, the prospect is favorable. In acute obstruction by gall-stones, Treves considers that laparotomy should be performed as soon as the symptoms of obstruction are pronounced.—ED.]

The same essentially applies to other foreign bodies of large size which are round, and, being impacted in the intestine, produce occlusion. In the case of foreign bodies, of course, the evidence derived from the history of the case usually makes the diagnosis much easier. In the treatment of occlusion of the bowel produced by the impaction of sharp, angular, and pointed objects, certain important principles of treatment have been evolved from experience. Objects of this character may pass through the intestinal canal without producing any damage, provided they are inclosed in large masses of fecal matter. On the other hand, when peristaltic movements of the bowels become very violent, so that these bodies lose their protective covering of fecal matter and are rapidly propelled onward in the intestine, they may become very dangerous. The administration of purgatives is, therefore, distinctly contraindicated in these cases. Patients who have swallowed some sharp or pointed object, and who have developed symptoms of occlusion of the bowel, should be fed with large quantities of soft food, like potatoes, rice, oatmeal, etc. When the foreign body reaches the rectum, its extraction should be carried out on surgical lines.

It is unnecessary to enter again into a detailed discussion of the treatment of occlusion of the bowel following internal cicatricial strictures, compression, or constriction of the bowel from without, for all the important points have already been dealt with in previous paragraphs, and there is nothing further to add except that strictures of the rectum require special methods of surgical treatment. The technic may vary: the condition may be relieved either by slow, mechanical dilatation of the constricted rectum or by some cutting operation.

A few remarks may be added as regards the treatment of occlusion of the bowel due to carcinoma.

There is no known internal method of treatment which is of any use in the treatment of carcinoma of the intestine. All that can be done by medical treatment is to relieve some of the symptomatic mani-

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[1] J. Hutchinson, Sr., *Archives of Surgery*, April, 1896.
[2] A. W. Mayo Robson, *Diseases of the Gall-bladder and Bile-ducts*, 1897, p. 88.

festations of the disease, such as constipation, intestinal hemorrhage, pain and tenesmus, peritonitis, etc. The details of the treatment of these conditions have been described in the sections devoted to them. In suspected stenosis of the bowel certain dietetic regulations should be carefully observed that are indicated in the treatment of this condition. (For the details I refer to preceding paragraphs.)

The treatment of this disease is chiefly surgical. As early as 1854, —that is, fifty years ago,—Virchow opposed the prejudice that existed at that time against operative interference in carcinoma. In 1888 he repeated what he said in 1854, as follows : "If it is true that carcinoma is at first, and frequently for a considerable time, a purely local disease, it should be possible to cure the condition during this time by local treatment." It is impossible to say whether a definite cure is ever attained in carcinoma of the intestine when it is treated by extirpation early in the disease. We do not even know what percentage of such cases recover or for how long a time recurrences may be postponed by such local treatment, for there are no exhaustive statistics on the subject ; the only way to formulate a definite opinion on this point is to collect such statistics and to analyze and study them carefully. Theoretically, extirpation of localized carcinoma should certainly be advocated in as early a stage as possible, for we know that all internal treatment directed against this lesion is utterly useless. To a certain extent practical experience bears out this view, particularly in the case of carcinoma of the rectum. Carcinoma situated in this portion of the bowel is readily accessible to manual palpation, and can, therefore, be diagnosed very early. In addition, the operative technic of extirpation of a carcinoma in this position is comparatively simple, and has, therefore, been most frequently performed in cases of rectal carcinoma. The results have been exceedingly favorable, for in many of the patients treated in this way no recurrences of the disease occurred, and in another large proportion there was no recurrence for several years. In carcinoma of the colon the chances of recovery after radical operation seem to improve from year to year as the surgical technic is perfected, and different surgeons gain more personal experience in this particular field. So far as I can judge from personal experience in the treatment of this disease, I should certainly advocate early treatment of carcinoma of the colon by operative measures.

The physician's chief object, which is to make a diagnosis of the disease as early as possible, so that the surgeons can operate on these cases at a very early period in the development of the disease, is, however, one of enormous difficulty. From a consideration of the fact that it may be exceedingly difficult to make a positive diagnosis of carcinoma of the intestine, even when the whole symptom-complex is well marked, it can easily be understood that this diagnosis may be quite impossible when the initial symptoms of a carcinoma of the colon, the ileum, or the duodenum are quite indefinite and ambiguous. Any physician who has had much experience in this field of diagnosis will agree with this statement. The only way in which to arrive at a positive diagnosis in

cases of this character would again be surgical interference—that is, an exploratory laparotomy. It is impossible to formulate any general guiding rules as to the advisability of performing such an exploratory incision in any given case; the only rules to be followed are those given in the general description above.

It is much easier to arrive at definite conclusions as to the advisability of surgical interference in cases where the patients are suffering from unbearable pain and other distressing symptoms, or in which the main object of treatment is to remove some condition which immediately threatens the life of the patient. Provided the consent of the patient can be obtained, operation should be advised in these cases without hesitation. This applies chiefly to cases in which the symptoms of complete occlusion of the bowel are present. In cases of this character startling results are occasionally obtained from operative interference. By way of illustration the following case under my own care may be quoted :

> The patient was a man of about fifty, who, for several days, had had all the symptoms of complete occlusion of the bowel. On examination a distinctly palpable carcinoma of the cecum was found. The patient refused to allow any other surgeon but Billroth to perform the operation. Owing to external circumstances, the patient, actually suffering from intestinal obstruction, had to travel for twenty-six hours in an express train to Vienna. The carcinoma was extirpated, and the colon joined to the ileum. The patient was seen a year after the operation, was enjoying excellent health, and was capable of attending to all the duties of his calling. Later there was a recurrence, but the patient survived three years after the first operation.

It is not necessary to describe the operative technic of the surgical treatment of carcinoma of the bowel, especially as I have had no personal experience in this field. It is quite impossible to decide in advance whether total extirpation of the carcinoma should be undertaken or what other course should be pursued in operating, or, for instance, whether, instead of extirpating the tumor, an artificial anus should be made. All these matters can be decided only during the course of the operation, for in each case the conditions met with vary greatly. No general rules can be laid down excepting those that are gleaned from the personal experience of different surgeons. The chief task of the physician should be to throw light on the following points and to answer the following questions—viz., in the first place, Is the patient suffering from a carcinoma of the intestine ? Is this diagnosis positive ? Is it probable or is it merely possible ? In what portion of the intestine is the carcinoma ? Is it or is it not probably of considerable size ? Are there grounds for assuming that it is adherent to other organs, or can these adhesions be definitely shown to exist ? With what organs is it adherent, and to what extent have metastases formed in other organs ? With a satisfactory answer to these questions the physician's responsibility ends and that of the surgeon begins, provided, of course, operative interference is at all feasible and the case is not so far advanced or in so unfortunate a condition that morphin, the great *solamen ægrorum et etiam medicorum,* alone can bring relief.

Obturation of the Bowel by Fecal Matter.—The removal of masses of fecal matter which block the intestinal lumen is often a matter of considerable difficulty, but can, as a rule, be brought about to a sufficient extent by bloodless measures. As soon as the diagnosis is positively established, it is well to proceed energetically with large rectal injections of water. Sometimes hard masses of fecal matter fill up the ampulla of the rectum and constitute an obstacle to the entrance of water into higher portions of the bowel. Under these conditions the water injected will immediately return, and the feces must first be removed with the fingers or with some blunt instrument formed like a spatula. After this is done, large rectal injections should be frequently repeated, liter after liter being given. The best solution to dissolve fecal matter is soap water (Penzoldt), and the best method of carrying out these irrigations is to employ an irrigator, as in this way no harm can be done. Occasionally we are obliged to use a long rectal tube.

[When the removal of long-retained fecal masses is being carried out, symptoms of constitutional disturbance from toxic absorption, such as fever, headache, malaise, and even urticaria, may be noticed. Ten-grain doses of salol night and morning appear to have a good effect in counteracting these results (Treves). While the irrigation treatment is being carried out general abdominal massage may be employed, and small hypodermic injections of strychnin ($\frac{1}{60}$ grain) three times a day are specially recommended by Treves.—ED.]

In addition to the irrigations, purgatives should be administered internally, provided they can be retained and are not immediately vomited. Castor oil, salts, and mineral waters are the most suitable remedies ; in more severe cases preparations of senna or of colocynth may be used ; in very obstinate instances recourse may be had to croton oil. So far as can be judged from present insufficient experience, it appears that among the cases reported as "cures of ileus by atropin" there are some of fecal occlusion.

All these measures should be supplemented by methodic massage of the abdomen, care being exercised that the manipulations are not too violent nor too prolonged. The possibility of stercoral ulceration of the intestine and of friability of the intestinal wall should never be forgotten. If there is reason to assume that the musculature of the intestinal wall is insufficient, this may be considered a correct indication for the employment of electricity, particularly of the faradic current, in addition to massage.

It is probable that the employment of metallic mercury owes its reputation chiefly to the effects produced in cases of occlusion of the bowel that were due to obturation of the lumen by fecal tumors.

In conclusion, a few words may be permitted on the treatment of **paralysis of the intestine.** In the account of the treatment of obturation of the bowel by fecal material in the preceding paragraph insufficiency of the bowel was mentioned ; this condition can be overcome as soon as the evacuation of the bowel contents has been brought

about in the manner outlined. The muscular coats of the intestine should be stimulated to greater energy by persistent methodic treatment, chiefly massage, abdominal gymnastics, electricity, and hydrotherapy.

In contradistinction to this form of temporary insufficiency, pronounced paralysis of the intestinal wall is one of the most difficult and one of the most discouraging conditions to treat, particularly when it occurs in the course of peritonitis and after abdominal operations and traumas. The former type cannot be cured at all by direct means, but usually constitutes the beginning of the end in cases of peritonitis; all the other types, as a rule, yield only with difficulty to any method of direct treatment. Stimulation of the intestine by irritating clysters or by purgatives is usually ineffectual; the same applies to faradization and kneading of the abdomen. Frequently the only thing to be done is to relieve meteorism by symptomatic treatment—that is, by puncture of the abdomen. This procedure naturally merely postpones for a short time the sad termination of the case. Occasionally, however, spontaneous recovery of intestinal power occurs, especially when the paralysis of the intestinal musculature was not too advanced and did not persist for too long a time. It yet remains to be ascertained whether success will follow treatment with physostigmin (*cf.* p. 146). The most favorable cases for treatment are those in which the paralysis is due to reflex irritation, and in which the primary cause can be removed.

RUPTURE AND PERFORATION OF THE INTESTINE

(Solutions of Continuity of the Intestine; Perforatio et Ruptura Intestini).

RUPTURE of the intestine can occur without any preceding disease of the intestinal wall, and is then, almost without exception, due to some form of mechanical injury. Again, rupture of the intestine may be due to some pathologic process in the intestinal wall which undergoes progressive destruction. Cases of the latter kind are of special interest to the physician, while rupture and perforation of the intestine as a result of injuries usually belong exclusively to the surgeon, and will, therefore, be touched upon only in a cursory manner in this section.

ETIOLOGY.

The most important etiologic factor producing perforation of the intestine is ulceration proper. (See p. 238.) The different forms of intestinal ulceration, however, do not all lead to perforation of the intestine with the same degree of frequency, and, in particular, there is no necessary correspondence between the absolute frequency of the different forms of ulceration and of perforation.

The exact figures based on statistics would be of very little practical value; such statements would, moreover, be exceedingly difficult to make, as the statistical material at present available is hardly suitable for this purpose; the available statistics bearing on the different kinds of ulcer of the intestine and their secondary complications chiefly

refer to the occurrence of general peritonitis after perforation, but do not deal with the relationship between the different forms of ulcer and the incidence of perforation itself. Only a few general remarks, therefore, will be made on this subject.

A summary of our knowledge as regards perforation of the intestine, including not only perforation into the general peritoneal cavity, but also perforation into encysted spaces which were in existence before the perforation occurred and are distinctly cut off by the products of peritonitis, shows that perforation of the bowel undoubtedly occurs most frequently in the vermiform appendix (compare the section on Appendicitis and Perityphlitis). As the primary cause of the perforation of this part of the bowel is in an overwhelming proportion of cases decubital and catarrhal ulcer, this form of ulcer must be considered the commonest cause of intestinal perforation. It is true that catarrhal decubital ulcers, while they frequently produce perforation in the vermiform appendix, rarely do so in other parts of the intestine, with the exception that they relatively commonly produce perforation of the intestinal wall immediately above chronic strictures. Most authors, in giving the order of frequency with which perforation is produced by intestinal ulcers, put typhoid ulcers in the first place. This holds good only for perforation into the free peritoneal cavity, followed by general peritonitis. Grawitz, for instance, in his statistics on perforative peritonitis, records 32 cases of intestinal perforation due to typhoid ulcers, and only 24 due to perforation of the vermiform process. These figures, however, prove nothing whatever as to the absolute incidence of the latter form of perforation, which, as I have shown, is, absolutely speaking, the most frequent one. His statistics being derived from autopsy material, do not include many cases of perforation of the vermiform appendix which recovered, and so lead to erroneous conclusions, since perforation due to typhoid ulceration almost always terminates fatally, whereas many cases of perforation due to ulceration of the vermiform appendix recover, and do not reach the postmortem table. (For the clinical details of perforation of the bowel in typhoid ulceration, the reader should refer to Curschmann and Osler's "Typhoid Fever.") After typhoid, the most frequent form of ulceration of the bowel leading to perforation is the tuberculous. The reason why perforation of the bowel is, absolutely speaking, so rare in this form of ulceration is that tuberculous ulcers are very chronic, and commonly lead to the formation of peritonitic exudates on the external surface of the ulcer, which exert a protective influence, and in many instances prevent perforation of the ulcer. The comparative rarity of perforation of the bowel in dysentery is astonishing in view of the enormous destruction of tissue usually present in dysenteric ulcers. In the colossal statistics of Woodward the number of cases of perforation of the bowel following dysenteric ulcers is quite small. It can readily be understood why perforation is rare in chronic dysentery, since here also peritoneal adhesions are numerous and prevent perforation. In the acute form, on the other hand, it is very difficult

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to say why perforation is not more frequent. Perforation of the bowel is, comparatively speaking, common in round ulcer of the duodenum, especially when the low absolute incidence of this form of ulceration is taken into account. In exceptional cases perforation of the bowel may also be produced by other rare forms of intestinal ulcer, as embolic, syphilitic, and duodenal ulcers following cutaneous burns. [Perforation may also follow uremic ulceration.—ED.]

After genuine ulcers of the stomach, carcinomatous destruction of the bowel-wall is the most prolific cause of perforation. Such carcinomatous ulcers are, however, comparatively rare, owing to the fact that carcinomata of the intestine are usually hard and show little inclination to break down. In many of the cases of perforation of the bowel in the course of carcinoma of the intestine careful examination shows that perforation did not occur in the cancer itself, but above it, and that it was not due to breaking down of the neoplasm, but to decubital ulceration of the bowel-wall above the carcinomatous stricture. Interference with the circulation of the blood through the intestinal wall may lead to gangrenous disintegration of the intestine and perforation—for example, in internal and external hernia, volvulus, and in intussusception. Foreign bodies in the intestine may also produce ulceration of the bowel; this is seen most frequently in the rectum, where a perforation is occasionally produced directly by rough manipulation of the nozzle of the syringe.

In all these conditions perforation of the bowel-wall occurs from within outward—that is, from the mucous lining to the serosa. In another group of cases, however, perforation occurs in the opposite direction. In considering the etiology of this form of perforation a great many anatomic lesions that may be the starting-point of the perforation must be considered. The nature of the process, however, is always the same, for in all the cases perforation is due to accumulation of pus outside of the intestine and in its immediate vicinity, the pus later burrowing its way through the intestinal wall into the lumen of the bowel. [Adami[1] has recently investigated this condition and has employed the term exogenous perforative ulceration to describe the process.—ED.] Such accumulations of pus are usually situated in the peritoneal cavity, but they occasionally arise in other organs which have subsequently become adherent to the intestine by the peritoneal adhesions. I will enumerate a number of possible forms of circumscribed suppuration, without, however, arranging them strictly in the order of their frequency. Suppuration in the vermiform appendix again occupies the first place, for perityphlitic abscesses due to the appendix very frequently perforate into the intestine. Next in importance, intraperitoneal abscesses due to other factors—for instance, purulent tuberculous peritonitis; psoas abscesses, in connection with the spinal column, and suppuration of mesenteric and retroperitoneal glands —must be taken into account. Pus may also form in the spleen, the liver, the gall-bladder, the kidneys, and the female genital organs, lead-

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[1] J. G. Adami, *Montreal Med. Jour.*, June, 1903, vol. xxxii., p. 401.

ing to peritonitis with exudation and the formation of adhesions which
ultimately anchor the different organs to the intestine; gradually the
wall of the intestine also becomes involved in the suppurative process,
undergoes purulent infiltration, and allows the entrance of pus from
the primary abscess cavity into the intestinal lumen. Finally, carci-
nomatous degeneration and perforation of the intestine must be men-
tioned; carcinoma may start from any of the organs enumerated above,
and, in addition, from the stomach, the bladder, and the vagina.

[Carcinoma of the bowel may open into another part of the intestine
and give rise to spontaneous anastomosis; Pollosson [1] has recorded 2
cases in which spontaneous anastomosis between the large and the small
intestine occurred and prevented obstruction from malignant disease.
In a case under the care of Mr. Turner and the editor, [2] a retroperitoneal
sarcoma growing near the right kidney underwent pseudocystic change
and opened into the second part of the duodenum.—ED.]

In perforation of the bowel from within outward, particularly when
due to ulceration of the intestinal wall, the process is a gradual one.
The various layers of the intestinal wall are gradually involved in the
process of ulceration and are progressively destroyed until, eventually,
the whole thickness of the intestinal wall is degenerated and perfora-
tion occurs. Occasionally, however, the process is more sudden; thus
in some cases the wall of the intestine is partially already destroyed,
when suddenly the remainder of the wall forming the base of the ulcer
ruptures. This termination is occasionally due to external traumatism,
but may occur spontaneously—for instance, when a large amount of gas
accumulates in the intestine, causing either violent contractions of the
intestinal wall or great distention of the bowel. Solutions of continuity
occurring in an intestine which was perfectly normal up to the time of
the accident are, without exception, produced by external violence. To
my mind it is more than doubtful whether a perfectly healthy intestine
can ever become ruptured as the result of excessive distention from
within,—that is, by meteoristic accumulation of gas,—and I do not
know of any well-established example of this accident in the litera-
ture. Lower reports the case of a workman sixty-five years old who
ruptured his intestine in lifting a heavy weight. Death occurred in
thirty-seven hours. Section: Perforation one-quarter inch in length
six feet above ileocecal valve. No ulcer existed. The intestine is fre-
quently perforated by a stab or a gunshot wound. Rupture may also
occur in other accidents which open the abdominal cavity—for instance,
when patients are impaled by spikes or poles or are gored by a bull or a
deer, etc. Another form of rupture of the bowel due to traumatism
is specially important, as the accident is not immediately discovered
and may remain undetected for some time—namely, rupture of the
bowel after injury inflicted on the abdomen by some blunt means which
produced no visible contusion or solution of continuity of the skin.
A great many cases of rupture of the intestine as the result of crush-

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[1] Pollosson, *Lyon Medical*, April 16, 1899, p. 567.
[2] Turner and Rolleston, *Lancet*, 1901, vol. i., p. 1273.

ing, falls, blows, etc., are reported in the literature on accidents. No useful end would be served by giving the details of individual cases. We know from experience that the solid organs of the abdominal cavity, particularly the liver and the spleen, are more apt to rupture under these conditions than the intestine, which, being elastic, is less liable to rupture, provided it is perfectly normal, than the more solid, unyielding abdominal organs.

ANATOMY.

In perforation of the bowel due to pathologic changes in the intestinal wall there is, as a rule, only a single perforation. This refers to those forms of destruction of the bowel-wall that proceed from within outward. Occasionally, however, particularly in dysentery and in perforation of the vermiform appendix, there are two or more perforations. In cases in which perforation of the bowel occurs from without inward, especially when perforation is due to the action of an intra-abdominal abscess, the intestinal wall is quite frequently perforated in several places. As a rule, inspection of the perforation at once shows in which direction the destruction of the bowel-wall and the terminal perforation occurred. The circumference of the opening is usually smallest in that layer of the intestinal wall that was involved last in the process. For instance, in a case of perforation of the bowel due to a typhoid ulcer, the mucous and submucous layer of the bowel may be destroyed over a considerable area, whereas the serosa is practically intact except for a comparatively small opening leading into the peritoneal cavity. Occasionally, it is true, this difference in the amount of destruction in the different layers of the intestinal wall is insignificant or absent, particularly in cases of gangrenous destruction of the intestinal wall. Detailed descriptions of the various anatomic conditions found in these various forms of destruction of the bowel-wall will not be given here, as these descriptions properly belong to the sections in which the different primary disease forms are described.

In those forms of perforation of the intestine due to some pathologic process in the intestinal wall the seat of the perforation depends on the nature of the primary disease. In typhoid fever the most frequent seat of perforation is the ileum ; in perityphlitis, the appendix vermiformis ; in dysentery, the large intestine ; in round ulcer of the duodenum, naturally, the duodenum. In tuberculous ulcers the seat of the perforation varies greatly, and still more, of course, in intraperitoneal abscesses which discharge into the intestine. The size of the perforative opening also varies greatly : in some instances it is hardly visible and has a lumen that is no larger than a pin-head ; in other cases it may be a hole through which the little finger can be inserted.

The anatomic changes that follow perforation of the bowel vary. When the perforation occurs into the general peritoneal cavity, the appearances are different from those when perforation occurs into an encysted, circumscribed space formed by firm peritoneal adhesions. In the first instance, those forms of peritonitis develop which are described

in the section on Peritonitis from Perforation ; in the latter case, inflammatory and suppurative processes develop in the immediate vicinity, of which perityphlitic inflammation may be considered the prototype. (In order to avoid repetition, the reader should refer to the section in which perityphlitis is described for a detailed description of .these sequelæ of perforation of the bowel.)

A great deal has been written on traumatic injuries of the bowel, and the surgical literature is full of case reports of this kind (Poland, von Beck, Sieur, and others). The works of these authors and other clinical accounts should be consulted for detailed descriptions of this accident, for it is impossible to enter into an exhaustive account of this subject, which is essentially surgical, here. A brief review of the most important features of traumatic perforation of the bowel may, however, be given here.

A gunshot wound or a stab wound may, of course, produce perforation of any portion of the intestinal canal ; in rupture of the bowel following some injury with a blunt instrument or following severe concussion the jejunum or the ileum is the most common portion of the bowel involved. The duodenum and the colon are much less frequently injured under these circumstances.

[From a consideration of cases of traumatic rupture of the intestine in civil practice Makins[1] comes to the conclusion that the factors which actually determine what part of the intestine is likely to be injured are that the violence should be exerted on that part of the abdomen supported by a bony wall posteriorly, and hence that the bowel to be injured should either be fixed in the lower half of the abdomen or possess a sufficiently long mesentery to lie in that region. Fixation of the bowel is only of secondary importance. In 21 cases the small intestine was ruptured in 16 and the large in 5. In 116 cases collected by Curtis the small intestine was involved in no less than 112.—ED.]

The tears in the jejunum are usually found near the junction of the jejunum with the duodenum. In some cases the intestine may be ruptured in two places. As a rule, the tear in the intestine runs in a transverse direction, usually on that side of the intestine which is situated opposite to the attachment of the mesentery to the intestine. Sometimes, however, the transverse rupture may be found near the attachment of the mesentery.

[Speaking of bullet wounds of the intestine as seen in the South African War, 1899–1900, Makins says that while rupture of the intestine in civil practice is, almost without exception, at the free border of the bowel, it is just as frequent at the mesenteric margin in bullet wounds, a point of considerable importance, since wounds near the mesentery are much more likely to be accompanied by hemorrhage.—ED.]

The direction of the tear in the great majority of cases is transverse to the longitudinal axis of the bowel ; less frequently, parallel to this axis. In the large intestine the rupture may be found in the haustra

[1] Makins, *Surgical Experiences in South Africa*, 1899–1900; "Traumatic Rupture of Intestine," *Annals of Surgery*, August, 1899.

or in the tænia of the colon ; as a rule, however, both these parts of the colon are ruptured at the same time. The size of the opening varies considerably ; it may be exceedingly small or may occasionally involve the whole circumference of the bowel. It is an interesting fact that in nearly all the cases the serous lining of the bowel seems to tear first, for we find that the serosa is torn to a greater extent than the mucosa. In some cases the serosa and muscular coats have been found to be ruptured while the mucosa remained intact. Occasionally the mucosa has been found prolapsed through the wound in the serosa, and, in a few instances in which the opening in the serosa was very small, the prolapsed mucosa formed a sort of a tampon that constituted an effective protection against the extravasation of bowel contents into the peritoneal cavity. The anatomic sequelæ of perforation of the bowel following trauma are the same as those of acute spontaneous perforation.

[Makins, in the South African War, found that the openings in the intestinal wall varied in character according to the mode of impact. The gut might be merely contused by the lateral contact of a passing bullet and show elongated ecchymoses parallel, oblique, or transverse to the long axis, or the peritoneum only might be split. The small intestine is easily displaced, so that a bullet may pass through the abdomen and yet not perforate the intestine. Bullets passing from one flank to another, which would tend to traverse fixed portions of the colon, or anteroposterior wounds in the small intestine area, were more dangerous than bullets taking an oblique or a vertical course through the abdomen, where lateral displacement of the bowel by the bullet is possible. Circular perforations of the bowel may show protrusion of the mucous membrane both at the aperture of entrance and of exit ; the apertures are surrounded by localized ecchymoses which are quite characteristic of this form of injury and are of value as a guide in finding the openings during operation. The perforations are often small and regular, but may be in the form of long slits or gutters. Extraperitoneal wounds of the colon are more dangerous than intraperitoneal ones.

Makins has also drawn attention to the fact that in traumatic rupture of the intestine as seen in civil practice the actual amount of fecal matter which is extravasated into the cavity of the peritoneum is small, and that peritoneal infection would describe the result better than fecal extravasation. This result contrasts with the free escape of the intestinal contents into the peritoneum in cases of perforation of an ulcer.[1] By microscopic examination G. L. Cheatle[2] found that extrusion or prolapse of the mucous membrane through a bullet wound of the intestine could play no part at all in preventing extravasation of the bowel contents. The valvular character of the opening made by the bullet prevented extravasation. Treves considers it probable that the shock of a bullet wound through the abdomen stops peristalsis.—Ed.]

[1] *Annals of Surgery*, August, 1899.   [2] Cheatle, *Lancet*, 1893, vol. i., p. 101.

CLINICAL FEATURES.

In this section no description will be attempted of the phenomena which precede perforation of the bowel, for they depend entirely on the character of the primary morbid process responsible for the perforation, and have nothing to do directly with the perforation, and, in fact, may occasionally be entirely absent in cases of perforation—as, for instance, in traumatic rupture of the bowel. No account will be given here of the symptoms of peritonitis due to perforation of the bowel, for these will be described separately in later sections. I shall, therefore, limit myself here to describing and discussing those symptoms which are directly due to the solution of continuity of the bowel-wall. It is true that occasionally perforation of the bowel cannot be diagnosed, and that under certain circumstances this accident may produce no direct symptoms. Frequently, however, a pronounced and distinctive clinical picture is produced which is particularly characteristic when large quantities of air enter the peritoneal cavity.

The solution of continuity or rupture of the bowel *per se* occasionally produces one direct symptom at the instant at which it occurs, a symptom, moreover, that is sometimes quite characteristic. I refer to violent, occasionally overwhelming, *pain*. Special emphasis, however, must be laid on the fact that this pain, occurring during the act of perforation, must be carefully differentiated and distinguished from the pain resulting from the peritonitis, which develops soon after the perforation of the bowel. Educated and intelligent patients usually distinguish the character of this pain from all other kinds of pain that they have ever experienced. Many of them describe a sensation as though something had burst or given way in the abdomen. As this pain is experienced not only in traumatic forms of rupture, but also in perforation by ulcers in which the greater portion of the intestinal wall was already destroyed, and as this pain occurs at the precise moment when the peritoneum ruptures, we must assume that it is due to irritation of the nerves in the latter tissue. The intensity of the pain varies; these differences in the intensity of the pain are not so much dependent on the individual sensitiveness of the patient as on the nature of the pathologic process responsible for the perforation. Generally speaking, however, the pain is very severe, so that the patients often faint and nearly always present marked symptoms of collapse. The latter undoubtedly originate in a manner that is analogous to the symptoms produced by Goltz's tapping experiment. The facial expression is one of suffering; the patients are in a state of collapse; there is general weakness; the pulse is small, thin, and often rapid; nausea and vomiting often appear. This picture is essentially the same as that witnessed in sudden occlusion of the bowel, where it is produced by the shock of the occlusion itself. (I have already analyzed this form of collapse on p. 394, to which the reader should refer for the details.)

In some cases the pain produced by the perforation of the bowel is not so violent; the symptoms of collapse may be completely absent,

and cases are actually on record in which the patient was able to be about for some time after his intestine had become ruptured by some acute trauma ; it even seems to happen occasionally that the act of perforation occurs without any pain whatever.   This is especially the case when general peritonitis already exists or when the perforation occurs after firm adhesions have formed all around the injured portion of the bowel.   Finally, perforation may occur without causing pain in patients who are suffering from some severe general disease in which there is stupor and in which the sensorium is benumbed, as in typhoid fever.   The reverse, however, may be the case under these conditions, for occasionally patients who are in a state of deep stupor are suddenly aroused by the violent pain produced by the perforation of the bowel.

Sometimes it happens—and I have personally seen cases of this kind—that the pain ceases spontaneously after a short time and does not return up to the death of the patient.   In one case that I observed the rupture of the bowel occurred in the night of the fourth to the fifth of November ; the violent pain lasted about two hours ; on the morning of the fifth the patient was found in a state of severe collapse, but pain on pressure and spontaneous pain were entirely absent.   The absence of pain persisted ; the patient even recovered so far that he could perform the act of defecation.   Shortly afterward the condition of collapse grew worse, while pain was still entirely absent.   The patient died on the forenoon of the sixth.   On autopsy pneumoperitonitis was found. It is true that in this case the perforation was not in the intestine, but in a large gastric ulcer in the pyloric region.

In the great majority of cases of perforation of the bowel occurring in the course of febrile diseases (typhoid) the temperature suddenly drops, while at the same time the patient develops symptoms of collapse and the violent pain that I have described.   Gesselewitsch and Wanach, as well as a number of other observers, have noticed the opposite in some cases—namely, a chill and a rise of temperature up to 106° F., together with the pronounced symptoms of collapse.   It is important to remember this fact, because it is opposed to the generally accepted view ; in any concrete case of suspected perforation a rise of temperature should never lead us to exclude the diagnosis of perforation.

Another symptom that is directly due to perforation of the bowel is the accumulation of gas in the peritoneal cavity.   The gas may appear either in the peritoneal cavity itself (tympanites or pneumatosis peritonei) or in some sacculated abscess cavity in the peritoneum.   Occasionally intestinal gases or other intestinal contents may enter the bladder or the stomach when perforation of the bowel occurs into these organs.

It is frequently a difficult matter to recognize the accumulation of gas in the peritoneum ; in some instances it may even be impossible to determine the presence of gas in this cavity.   Much ingenuity and acumen have been expended by various observers in gaining reliable indications for the diagnosis of the occurrence of this event.

Experience and deduction from theoretic considerations show that it is quite possible for free gas to be present in the peritoneal cavity without producing recognizable distention of the abdomen; it is only when large quantities of gas are present in the peritoneal cavity that distention of the abdomen can be expected. It does not necessarily appear when there are only small quantities of gas in the peritoneal cavity. The following signs may be employed in making the diagnosis of free gas in the peritoneum:

The abdomen is uniformly distended. On percussion, a loud and deep note is obtained all over the abdomen. The percussion-note may be either tympanitic or non-tympanitic, according to the quantity of gas present in the peritoneal cavity and on the tension of the abdominal walls. When the note is not tympanitic, simultaneous auscultation and percussion with the pleximeter may bring out metallic sounds. The latter sign is not necessarily present, even though pneumo-peritoneum exists, for the percussion-sound may show certain differences, especially when old peritoneal adhesions prevent the free distribution of the gas through the peritoneal cavity. Conversely, the note may be uniform over all the abdomen in cases where there is no perforation, but where there is a marked degree of intestinal meteorism.

Some writers believe that dulness soon appears in the dependent portions of the abdomen from the development of peritonitis. This symptom, however, is not by any means so important as some authors seem to think; for, in the first place, the dulness may be completely absent, either because no exudation is being formed or because the fluid exudate, if it is formed, accumulates in portions of the abdomen that are situated so deep down that the presence of this fluid cannot be detected by percussion. In the second place, the same phenomenon may occur in cases of peritonitis without perforation, or even in cases without peritonitis and without perforation in which axial rotation and internal incarceration of the bowel are present, together with enormous degrees of local meteorism. In this condition, as I have shown in a previous paragraph, the portion of the bowel in which local meteorism develops may fill up the whole abdominal cavity and at the same time lead to a copious hemorrhagic extravasation; the latter may accumulate in the lowest portion of the peritoneal sac, and give rise to dulness on percussion there. We see, therefore, that dulness in the dependent portions of the abdomen is by no means a characteristic symptom of intestinal perforation.

Many clinicians are inclined to attach more importance to the disappearance of the liver dulness on the anterior surface of the abdomen; but this sign, again, is ambiguous and deceptive, for disappearance of the liver dulness is also found in intestinal meteorism, particularly if the liver becomes displaced by the pressure exerted from below. Under these conditions the most careful percussion may fail to reveal any liver dulness whatever, and the percussion-note obtained over the lungs may merge directly into the meteoristic note heard over the intestines. Another possibility must always be taken into consideration—namely,

that some loop of intestine may pass in front of the liver and in this way cause disappearance of the liver dulness.

[This occurs in acute atrophy of the liver. A distinction has been drawn between alteration of the liver dulness in the mammary and anterior axillary lines, where it is usually estimated, and in the mid-axillary and posterior axillary lines. Thus Thornton [1] finds that while the hepatic dulness may be absent in the mammary and anterior axillary lines, it never disappears in the midaxillary and posterior axillary lines in mere tympanites. He, therefore, regards absence of liver dulness in the midaxillary line as almost conclusive proof of the presence of free gas in the peritoneal cavity. In 10 cases of perforation of the stomach or duodenum in which special attention was paid to this point, English [2] found that the absence or diminution of hepatic dulness extended to the midaxillary line in 4 cases. On the other hand, a similar condition was found in 2 cases in which the abdomen was opened but no perforation was found.—ED.]

Conversely, this sign may be absent even though there is pneumo-peritoneum—namely, when the liver is fixed in position by adhesions. Disappearance of the liver dulness is a characteristic and typical sign only in one condition—namely, when a small quantity of air escapes from the intestine and accumulates between the liver and the anterior abdominal wall. If, in a case of this kind, the patient was examined shortly before the occurrence of the perforation, and, at that time, the liver dulness was found to be normal, the subsequent disappearance of the liver dulness may be considered pathognomonic of perforation.

This is explained by the fact that the gas escaping from the per-forative opening in the bowel naturally rises to the highest portion of the peritoneal cavity. If the patient is lying in the dorsal position, this point is situated between the liver and the diaphragm and behind the epigastric portion of the peritoneum. In cases of this kind a dis-tinctly circumscribed area of gray discoloration will be found on the surface of the liver on either side of the suspensory ligament (Traube). I will not enter into a discussion of the disappearance of the splenic dulness that is also always mentioned, as this sign is of no real impor-tance. When the numerous fallacies in connection with percussion of the spleen, even in subjects who can be moved about without difficulty, are taken into account, it will readily be understood that the deter-mination of the splenic dulness by percussion is quite impractical in individuals who can hardly be touched on account of the pain.

Traube also mentions a peculiar feel of the epigastrium on palpation corresponding to the area occupied by the circumscribed accumulation of air. He claims that the epigastrium under these circumstances feels doughy, and can be more readily indented on palpation than other portions of the abdominal surface.

In recent and uncomplicated cases of perforation of the bowel the col-lection of gas is, moreover, freely movable. This is due to the natural

[1] G. Thornton, *Lancet*, 1902, vol. i., p. 442.
[2] T. C. English, *Medico-Chir. Trans.*, vol. lxxxvii.

tendency of the gas present in the peritoneal cavity always to occupy the highest point; consequently it readily changes its position when the patient moves about. Even when there are large quantities of gas in the abdominal cavity, accompanied by exudation of fluid, the change of position of the former can readily be determined, provided the pain is not so severe that the patients are obliged to lie perfectly still. This latter factor—namely, the impossibility, on humanitarian grounds, of carefully examining the patient in this direction by causing him to move about—greatly impairs the value and the utility of this sign in making the diagnosis.

E. Wagner mentions another sign of peritoneal meteorism that he considers positive—namely, that, in spite of the presence of other symptoms of acute peritonitis (as tension and distention of the abdominal walls, elevation of the diaphragm, pain, tenderness, etc.), no movements of the intestine can be seen, felt, nor heard. This sign is, however, by no means pathognomonic, for in cases of intestinal meteorism, especially when complicated by general peritonitis, all visible and audible peristalsis of the bowels may be absent.

In addition to these signs, a few auscultatory phenomena must be mentioned. Some authors, for instance, state that the aortic sound acquires a metallic ring; this sign, however, may also occasionally be observed when the stomach is greatly distended with gas. Others report a percussion-note with a metallic ring appearing when the patients are shaken, provided fluid exudate is also present. But how can we dare to shake a patient who is tortured with pain? The sign, moreover, is without diagnostic value, for the same metallic splash can be heard whenever gas and fluid material are present in the stomach. Tschudnowsky has described amphoric breath-sounds over the abdomen said to be heard synchronously with the inspiration. He claims that these sounds are produced by the passage to and fro of air through the perforative opening. The sound, he says, is heard only in cases of perforation of the bowel in which the opening in the intestine is large and is not occluded by any of the neighboring parts. The absence of this sound, therefore, would in no way militate against the diagnosis of perforation. As a matter of practical experience, this amphoric sound is absent in the great majority of cases.

From the preceding discussion it will be seen that very few of the physical signs that we might expect would reveal the presence of free gas in the peritoneal cavity are reliable, and that the value of physical examination on this question is very slight. Nevertheless, one or the other of these signs, or several of them together, may aid us in establishing a fairly positive diagnosis. We know that all these symptoms are naturally absent when perforation of the bowel occurs into some encysted portion of the peritoneum that is completely closed by peritoneal adhesions. Under these circumstances the recognition of perforation of the bowel will be possible only under specially favorable conditions. In the section on Carcinoma of the Intestine the possibility of diagnosing perforation into the bladder, the stomach, and other por-

tions of the bowel was referred to. The reader should refer to this section, where this matter was dealt with in some detail, because perforation of the bowel into these organs occurs principally in intestinal carcinoma. (See pp. 428, 429, 430.)

It would be quite erroneous to assume that in perforation of the bowels the abdomen must always necessarily become distended ; on the contrary, we occasionally find the abdomen in a condition of boat-shaped retraction, with the abdominal muscles tense and hard as a board ; this is seen particularly in cases where perforation of the bowels occurs suddenly and in which the opening in the intestine is very large. This peculiar phenomenon, if it appears at all, is usually witnessed quite soon after the perforation occurs. We must assume that the sudden entrance of intestinal or gastric contents into the peritoneal cavity is a very powerful irritant which stimulates the abdominal musculature to contraction. It is impossible to determine, from a study of the clinical features in the published cases, what special factors are required to produce and to prevent this effect. I have seen it most frequently in perforation of gastric and duodenal ulcers, but it is also known to have occurred in perforating typhoid ulcers.

Lastly, another phenomenon may be indirectly utilized in making the diagnosis of perforation—namely, the appearance of a peculiar fruitlike, sweetish, aromatic odor, that does not emanate only from the mouth of the patient, but can be perceived all around him. This characteristic odor has been noticed a number of times in my clinic, but, unfortunately, not only in perforative peritonitis, but also in other forms of purulent inflammation of the peritoneum. It seems, therefore, to be characteristic of purulent peritonitis. The genesis of this odor is not clear. It may be expressly mentioned that it was noted in cases where there was very little acetone in the urine.

[In peritonitis there is usually a well-marked polymorphonuclear leukocytosis ; this is not, however, an absolute rule, as in very severe cases of perforative peritonitis leukocytosis may be absent. Leukocytosis, though of the greatest value as showing the existence of peritonitis or of intraperitoneal suppuration, especially in appendicitis, is not an index of perforation apart from peritonitis. In distinguishing perforation from peritonismus or hysterical peritonitis leukocytosis is a valuable indication of real inflammation. In typhoid fever, where there is usually a diminished leukocyte count, or leukopenia, the presence of leukocytosis in a case where perforation is suspected points to perforation or to peritonitis. Thayer's[1] observations on this point are to the following effect: Perforation in typhoid fever is usually followed in a few hours by a leukocytosis which may be considerable (above 15,000) or slight (under 10,000), and appreciable only on comparison with previous counts. In some instances a slight leukocytosis following perforation may tend to diminish and disappear, with the aggravation of the symptoms. Not infrequently after perforation there is a complete absence of leukocytosis or even leukopenia ; the absence or disappearance of leukocytosis

[1] W. S. Thayer, *Johns Hopkins Hosp. Reps.*, vol. viii.

after perforation is an indication of the malignancy of the infection or of the prostration of the patient; the prospect of success by surgical interference is, therefore, best in cases showing leukocytosis. A preperforative leukocytosis due to local peritonitis about deep ulcers may occur.—ED.]

An entirely different congeries of symptoms occurs when perforation of the intestinal wall occurs from without inward, due to the rupture of a collection of pus into the intestine. Here the violent initial pain, as well as signs of accumulation of gas in the peritoneal cavity, collapse, great prostration, and the other general symptoms are usually absent. We may say, *per contra*, that the occurrence of this form of perforation may occasionally lead to a decided improvement in the subjective feelings of the patient; fever present before the perforation may disappear, together with other objective symptoms of intraperitoneal or extraperitoneal suppuration. The most positive proof of the occurrence of this form of perforation of the intestinal wall is the appearance of pus in the motions. It is usually easy to detect the presence of pus in the stools if sufficient attention is given to this point, for, as a rule, there are considerable quantities of pus in the dejecta after perforation of an abscess into the intestine.

The symptoms described above—namely, violent pain and pneumatosis of the peritoneum—appear when perforation occurs into the free abdominal cavity. When firm peritoneal adhesions exist between different loops of intestine or between the intestine and some other solid organ, the clinical picture presented when perforation occurs is very different. In cases of this kind the pain is frequently very slight and cannot be compared to the pain experienced in perforation into the general abdominal cavity. No gas, of course, escapes into the peritoneal cavity. The necessary and constant result, however, of perforation of the bowel under these conditions is the formation of an inflammatory and suppurating focus, as is well seen in perityphlitis after perforation of the vermiform appendix.

Subcutaneous injury of the intestine from blunt objects often presents serious difficulties to diagnosis. Initial symptoms are frequently not at all proportional to the severity of the lesion. Even in very serious rupture initial shock may be absent (Schmitt). Angerer and Kirstein attribute great diagnostic import to marked rigidity of the abdominal muscles.

COURSE AND TERMINATION.

In cases of perforation into the general peritoneal cavity due to intestinal ulceration death may occur from collapse before peritonitis has time to develop. As I have remarked above, the pulse is usually greatly accelerated; occasionally, however, the pulse-rate is slow. Gluzinski reports a case in which the physician, who happened to be present at the moment when perforation occurred (in a case of acute appendicitis), noticed that the pulse-rate became very rapid. When he examined the patient on the following day, he found that the pulse-rate

was only 70, and that at the same time considerable improvement in the patient's general condition seemed to have occurred. For these reasons he decided to give up the operation which he had planned. A few hours later fulminating peritonitis developed and rapidly led to the death of the patient. Gluzinski then attempted to determine, by experimental means, what was the cause of this secondary slowing of the pulse. He argued correctly that it could not possibly be due to the acute primary irritation of the nerves. He made out, however, that it was due to the absorption of putrefactive gases which had escaped into the peritoneum from perforation of the bowel. He showed that these gases are very rapidly absorbed and that they contain substances capable of stimulating the inhibitory mechanism of the heart.

As a rule, peritonitis develops in the natural course of the disease, usually setting in within a few hours after the perforation. A special section will subsequently be devoted to the consideration of this very dangerous condition.

The course of the disease in cases of rupture of the intestine following severe crushing injuries of the abdomen without lesions of the cutaneous coverings, and in those forms of perforation due to stab or gunshot wounds of the intestine, is exactly the same as in perforation of intestinal ulcers. In the traumatic forms, however, the prognosis is usually somewhat more favorable because surgical measures offer better chances of success.

[With regard to bullet wounds, this statement probably requires some qualification in the light of experience gained in the war in South Africa. According to Makins, perforating wounds of the small intestine are extremely fatal; it is probable that in cases where it might be assumed from the course taken by the bullet that the small intestine had been perforated, and where recovery follows without local peritonitis or suppuration, that there has been no actual perforation. The prognosis of wounds of the large intestine is better than of the small intestine; the outlook is better in wounds of the cecum, ascending colon, and rectum from the greater fixidity of these parts and the lesser tendency to be covered by small intestine. An extraperitoneal wound of any of these portions of the bowel is more dangerous than an intraperitoneal, and more likely to give rise to septicemia (Makins). Further, experience showed that the cases of bullet wound of the intestines suitable for abdominal section on the field are exceedingly few (Treves [1]).—ED.]

A few cases are also on record which appear to show that occasionally a perforation of the bowel may heal spontaneously without the development of peritonitis. This spontaneous recovery is, as a rule, inaugurated by the approximation of a piece of the omentum to the wound; in this way the perforation is mechanically closed and the further extravasation of bowel contents into the free abdominal cavity prevented. Perforative injuries to the bowel that are only minimal in extent may, of course, close spontaneously, thanks to the physiologic tendency of the elastic and muscular elements of the intestinal wall to

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[1] Treves, *Medico-Chir. Trans.*, vol. lxxxiii., p. 294.

contract. That this is the case can readily be determined by studying the results of puncture of the intestine for the relief of meteorism.

As I have already said, perforation of the bowel where it is inclosed in firm peritoneal adhesions leads to the formation of an abscess. These abscesses may either be intraperitoneal or extraperitoneal, may involve a solid organ in the vicinity, or, lastly, may perforate into some other viscus—the bladder, the intestine, the vagina. These results will depend on the anatomic conditions present in each individual case. These sequelæ and the clinical pictures presented by these different lesions will be described elsewhere. It is very doubtful whether complete perforation of the bowel into the free peritoneal cavity can ever occur without producing grave results. I do not think that it has yet been shown that this can occur without any symptoms—in other words, that a patient in whom perforation of the bowel into the free peritoneal cavity occurs can recover from the accident without ever developing serious symptoms of the character I have described. It may, of course, happen that in cases of trauma only a certain portion of the thickness of the intestinal wall is ruptured, so that at first no symptoms appear. In the course of a few days, however, the solution of continuity becomes complete, and the typical syndrome of rupture of the bowels develops.

TREATMENT.

Perforation into the general abdominal cavity was formerly considered to be an almost absolutely fatal condition. It is true, a few cases are to be found in the older literature in which recovery from perforative peritonitis was recorded, but the validity of these reports was doubted at the time. In the account of perforative peritonitis it will be seen that, however serious this disease may be, it cannot be admitted that the disease is always necessarily fatal. However, I will speak of peritonitis later. At present we are concerned with the treatment of perforation and rupture of the bowel *per se*.

Examination of the surgical literature on intestinal perforation, particularly the publications of the last few years, shows that there are a large number of cases in which recovery has occurred even though the operation was performed under the most serious conditions. This applies more particularly to cases of traumatic rupture of the bowel, to gunshot and stab wounds of the intestine, and to other serious injuries. I need hardly emphasize the fact that in cases of this kind good results can be expected only when operative interference is undertaken as soon as possible after the accident, and, if possible, before the onset of peritonitis. A patient who is wounded in this way should immediately be prepared for an operation, a laparotomy performed, and the injury to the intestine repaired at once. Siegel's statistics show that the mortality of perforating abdominal wounds when subjected to operation in four hours is 15 per cent.; five to eight hours, 44 per cent.; nine to twelve hours, 63 per cent.; and later, 70 to 87 per cent. (For the technical details of the different methods of repairing intestinal injuries I must refer to surgical monographs on the subject.) In

order to illustrate how much can be accomplished by these means I will quote 2 cases:

A farm-hand fell from a considerable height and became impaled upon a pole. The point of this pole broke off and could not be found. The patient was examined a few hours after the accident, and was found to be in a state of profound collapse. In addition there were symptoms of peritonitis from perforation and of peritoneal rupture of the bladder. In the vicinity of the anus, to the left, a punctured wound was found which led into the rectum. When this wound was enlarged, a second lesion of the rectum was found above the prostate. The wound was thoroughly disinfected and the rectum tamponed with iodoform gauze; then a laparotomy was performed. On exploring the abdominal cavity two openings were found in the sigmoid flexure through which 8 cm. of the point of the pole were found inserted, the piece of wood being as thick as an adult finger; in addition, the posterior wall of the bladder was found to be injured. Particles of fecal matter were discovered in Douglas' pouch. The sigmoid flexure was resected, the wound in the bladder sutured, the peritoneum thoroughly cleansed, and an iodoform gauze packing introduced. The patient made an uneventful recovery (Borsak). A patient of Neugebauer's received 13 holes, 10 in the small intestine, as the result of a gunshot wound. Recovery occurred in ten days.

Modern surgeons have also attempted to treat cases of ulcerative perforation of the intestine and other forms of perforation in the same way as traumatic cases. Particular attention has been given to the treatment of perforation of the vermiform appendix. A special section will subsequently be devoted to the treatment of this condition. Other surgeons have also attempted to treat cases of perforation of round ulcer of the stomach, of typhoid, and of other ulcers of the intestine according to the same principles. This method of treatment will be considered in the section on Perforative Peritonitis. While the results of this surgical method of treatment are not particularly brilliant, the introduction of surgical methods into the treatment of this hopeless disease is a decided advance, for, as we have seen, internal treatment is very ineffectual. In cases where surgical interference is, for one reason or another, impossible, treatment is confined to absolute rest, to forbidding the administration of all food, to giving large doses of opium, to applying ice compresses, and, possibly, to undertaking the symptomatic treatment of collapse, according to well-established principles.

DISEASES OF THE PERITONEUM.

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DISEASES OF THE PERITONEUM.

PHYSIOLOGIC INTRODUCTION.

THE peritoneal cavity is a serous sac, the surface area of which, when spread out, is astonishing. Wegner has calculated that in a female subject of medium size it is approximately 17.182 square centimeters when the surface of all the organs and parietes covered by the peritoneum is taken into account. It will be seen, therefore, that the extent of the surface of the peritoneum is approximately the same as that of the external surface of the body, which, in a subject of the same size, comes to 17.502 square centimeters.

Formerly the peritoneal cavity was regarded as a perfectly closed sac. The epoch-making experiments and researches of von Recklinghausen, and those of Schweiger and Dogiel, Klein, Notkin, Beck, Muscatello, and others, have, however, shown that the endothelial lining of the peritoneum, particularly in the region of the diaphragm, is not continuous, but is interrupted by a large number of holes (stomata), the diameter of which has been found to be large enough to permit the passage of bodies twice the size of red blood-corpuscles. Incidentally, it may be mentioned that this view has been contested by a few observers (Kolossow). These stomata lead directly into the serous and subserous lymph-spaces and so establish a communication between the peritoneal cavity and the lymphatic system ; in this way, too, an almost direct communication is established between the peritoneal cavity and the vascular system, the peritoneal cavity being, in reality, separated from the vascular system (the innominate vein) only by the short thoracic duct.

[The question as to a free communication between the peritoneal cavity and the lymphatic system has recently been investigated by W. G. MacCallum,[1] who has shown that the large stomata described by von Recklinghausen do not exist as such ; that the endothelial lining of the peritoneum is complete ; and that there is no open communication between the peritoneal cavity and the lymphatic system. The lymphatic lacunæ are covered over by a roof of endothelial cells. Phagocytosis is extremely important in the mechanism of absorption of granular material from the cavity of the peritoneum ; phagocytes pick up granules and can be seen working their way through the endothelial cells forming the roof of the lacunæ. At the same time the pumping action of the diaphragm due to respiratory movements is

[1] W. G. MacCallum, *Johns Hopkins Hosp. Bull.*, May, 1903, vol. xiv., p. 105.

important and pigment-granules can be seen to pass between the adjacent margins of the endothelial cells.

The most important lymphatic trunks draining the peritoneal cavity are those which run up in the anterior mediastinum. In acute peritonitis the lymphatic glands in the first intercostal space are reddened, swollen, and contain micro-organisms. In fact, a diagnosis of peritonitis is sometimes possible from examination of these glands before examining the abdomen. Experimentally, bacteria can be found in these glands as early as six minutes after injection into the peritoneal cavity. These observations of H. E. Durham's [1] were made independently of Muscatello's [2] work on the importance of the lymphatic tracts of the anterior mediastinum.—ED.]

Wegner and some other authors also maintain that in the region of the mesorectum and of the stomach and in some other parts of the abdominal walls openings can be found in the endothelial covering which communicate directly with the lymphatic vessels of these parts. Oppel has suggested that the path taken by the lymph-stream from the peritoneal cavity is chiefly by the great omentum, and gives as his reason that in many cases where the peritoneal lymph was free from bacteria a large deposit of micro-organisms was to be found on the great omentum. Undoubtedly, different secondary paths exist whereby the lymph escapes from the peritoneal cavity, but totally new experiments by Clairmont and Haberer have again demonstrated that absorption takes place chiefly by means of that portion of the peritoneum covering the diaphragm.

The great importance of these relations is apparent, and will be frequently referred to in the course of the following account :

The important investigations of G. Wegner have also thrown a great deal of light on the vital processes going on in the peritoneum. Before the publication of his investigations, practically nothing was known about this subject ; a number of other researches supplementing Wegner's observations were subsequently published. All these studies have thrown much light on this obscure subject. As a knowledge of the functions of the peritoneum is essential in order to appreciate the significance of morbid processes involving it, a brief summary of our knowledge of the physiology of the peritoneum will be given here.

Normally, there is only a very small quantity of serous fluid in the peritoneal cavity, which is the product of a constant interchange of fluids, for between the two layers of the peritoneum there is practically only a single capillary space with a stream of fluid circulating to and fro between them, the circulation being maintained by a double process of transudation and absorption.

The absorptive powers of the peritoneum are of the greatest importance in certain pathologic conditions, and are truly remarkable both as to the rapidity with which absorption can occur and as to the quantities of fluid that can be absorbed. Wegner, in the course of a number of

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[1] H. E. Durham, *Medico-Chir. Trans.*, 1897, vol. lxxx., p. 191; *Jour. Path. and Bacteriol.*, vol. iv., p. 338.   [2] Muscatello, *Virchow's Arch.*, 1895, vol. cxliii.

experiments, found that in a dog weighing 13,400 gm., 170 c.c. of 870 c.c. of serum injected into the peritoneal cavity were absorbed within an hour, an amount of fluid corresponding to 1.3 per cent. of the animal's weight. In rabbits still higher figures were obtained, between 3.3 and 3.8 per cent. of the body-weight being absorbed within an hour.

The absorption is carried on partly by the lymphatic vessels and partly directly by the blood-stream. Not only are fluids and substances both soluble and dissolved, which may be present in the peritoneum in certain pathologic conditions or may have been introduced for experimental purposes, absorbed in this way, but also solid bodies— *e. g.*, colored particles, blood-cells, bacteria, etc.—in the fluid contained in the peritoneal cavity ; it was also found that colloid solutions containing salt were absorbed, though less rapidly than fluids and substances in true solution. The absorption of these materials is partly a vital, partly a physical, process, partly by means of the blood, partly by the lymph-stream. Corpuscular elements pass, as shown first by von Recklinghausen and later by Wegner, directly through the lymph-stomata, and so by the thoracic duct and vena innominata into the circulation. Wandering cells found in normal peritoneal fluid take up some of these corpuscular elements ; others are taken up by the endothelium itself. Muscatello and Salzer showed that absorption of insoluble bodies takes place entirely by means of the lymph-stream. Fluids and soluble substances, on the other hand, are absorbed largely by the blood ; in this view Starling and Tubby, Mendel, Orlow, Heidenhain, Hamburger, Klapp concur ; Meltzer and Adler alone insist on the absorption of the latter substances by the lymphatics.

[The omentum plays an extremely important part in absorption, from the peritoneal cavity, both of micro-organisms and of solid particles, such as carmin, ferrocyanid of potassium, etc. Durham[1] found that in an animal killed twenty minutes after intraperitoneal injection of micro-organisms the omentum shows enormous numbers of hyaline cells, micro-organisms, and phagocytes, while in animals which have died or have been killed twenty-four to forty-eight hours after an intraperitoneal injection of bacteria, which is efficient, but not too large, the omentum contains bacteria, but the peritoneal fluid is sterile on culture ; this throws very grave doubt on the chemic (aseptic) peritonitides of Taval and Lanz, in the absence of examination of the omentum and lymphatic glands in the anterior mediastinum. Adami[2] and Roger[3] have also drawn attention to the protective rôle of the omentum in peritoneal infection. In a fatal case of ruptured ectopic gestation Durham found the lymphatics of the anterior mediastinum injected with blood and the glands turgid with blood. Liquid is carried up into the anterior mediastinal glands more rapidly than solids ; thus a solution of carmin took three minutes, while India ink took eight minutes, to reach the anterior

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[1] Durham, *Jour. Path. and Bacteriol.*, vol. iv., p. 338.
[2] Adami, *Canadian Practitioner*, March, 1898.
[3] Roger, *Soc. de biolog.*, Paris, February 19, 1898.

mediastinal glands (Durham). The Fallopian tubes of sexually mature animals may pick up solid particles from the peritoneum, but the particles do not pass beyond the fimbriated extremities in immature animals (Lode).[1]—ED.]

Various factors influence the rapidity with which fluid is absorbed from the peritoneal cavity and the amount absorbed. It appears that under normal physiologic conditions the maximum is attained in both these respects, and that it is impossible to increase the rapidity or amount of absorption by the peritoneum. In the normal organism the rapidity of absorption is essentially regulated by two factors. The first of these is the pressure exerted by the abdominal muscles, and especially the rhythmic to-and-fro movements of the diaphragm; the other is the peristaltic movement of the intestines. The diaphragmatic movements act like a pressure- and suction-pump on the contents of the peritoneal sac ("the peritoneal cavity is an enormous lymph-space with the diaphragm-pump"); the latter—viz., the movements of the intestine—act in a different way—*i. e.*, they prevent the accumulation of intraperitoneal fluid in any one part of the peritoneal cavity, so that it cannot follow the laws of gravity and collect in the dependent portions; they carry the fluid over the different parts of the absorbing surface of the peritoneum, and in this way greatly enhance the absorptive powers of the peritoneum.

A number of factors may retard or even completely stop absorption of fluid from the peritoneal cavity; this point was originally investigated by Wegner and subsequently by several other authors, notably Schnitzler and Ewald, also Klapp, Clairmont, and Haberer. The factors capable of retarding absorption come into play chiefly, and perhaps only, under pathologic conditions; but this fact is of special interest and importance in this connection.

Reabsorption of intraperitoneal fluid is interfered with by venous engorgement, and when the tension and pressure in the veins become increased, their power of absorption of fluid becomes reduced. Under these conditions the intraperitoneal contents must increase, even though the rate of transudation remains perfectly normal. This result is seen in ascites due to venous engorgement.

It may also be considered established that a reduction in the energy of intestinal peristalsis, whatever its cause, diminishes, whereas an increase promotes normal absorption. The conclusion, on the other hand (Schnitzler and Ewald), that increased peristaltic movements of the bowels do not lead to increased absorption of intraperitoneal contents does not seem to me to be quite justified; at all events, it has not yet been proved beyond question; the chief argument of the adherents of this view is a certain experiment consisting in ligation of the transverse colon—this operation is expected to produce increased peristaltic movements of the bowel above the occluded portion; as a matter of fact, absorption from the peritoneal cavity does not seem to be increased when this is done; at the same time, I do not consider this evidence

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[1] Lode, *Arch. f. Gynäkol.*, vol. lxv.

conclusive for there is no positive proof that after ligation of the transverse colon the peristaltic action of the bowel above the obstruction is really increased. Other factors that can reduce the amount of absorption are diminished activity of the diaphragm and loss of tension of the abdominal muscles.

When the temperature of the abdomen is considerably reduced, the absorption of fluid from the peritoneal cavity is interfered with during the actual time that the temperature is depressed ; this is due to the direct effect of the intense cold, which naturally reduces the irritability of the nervous and muscular elements concerned in this process (Horvarth, Luederitz, and others). As a result of a low temperature, therefore, peristalsis is inhibited, and, as we have shown above, interference with this function can also interfere with absorption. Another factor that may be concerned in diminishing absorption after reducing the temperature of the abdomen is the contraction of the blood-vessels that naturally results.

Klapp especially made experiments showing the influence upon absorption from the peritoneal cavity of changes in the circulation of the cavity brought about by applications to the abdominal wall. He succeeded in markedly diminishing absorption by the external application of cold and likewise in stimulating it by such outward means—*e. g.*, warmth—as promote hyperemia.

Anemia, concentration, and dilution of the blood do not exercise any appreciable influence on the rate of absorption nor on the amount of fluid absorbed from the peritoneal cavity.

Changes in the peritoneum itself are of some importance in this connection. In view of the fact that in laparotomy the peritoneum is exposed and comes in contact with the air, several authors have studied the effect of drying on the peritoneum. Aside from the fact that drying the peritoneum is an important element in the genesis of peritonitic adhesions, the experiments of Schnitzler and Ewald have shown that drying, at the same time, causes retardation of the rate of absorption ; this becomes clearly manifest if the serous lining of a considerable portion of the bowel is dried, for then the absorption of fluid is unquestionably and quite considerably retarded. Klapp regards as the chief factor in this result the venous hyperemia which sets in. Clairmont and Haberer found experimentally that in the early stages of peritonitis absorption is hastened ; only in the later stages is it retarded.

It seems clear *a priori* that the transudation from the peritoneum must run parallel with its powers of absorption, and must, therefore, be very great ; as a matter of fact, the experiments of Wegner have demonstrated directly that this is actually the case.

Another property of the peritoneum which is quite remarkable and therefore calls for special mention is its great "plasticity." Experimental evidence, as well as clinical experience, shows that the peritoneum possesses in a very marked degree the power of forming plastic tissue as soon as it is subjected to the influence of certain irritants. When a solution of continuity occurs in the peritoneum, the severed parts soon be-

come agglutinated, and finally coalesce and grow together again. When some foreign body gains entrance into the peritoneal cavity, it is soon encapsulated, provided it is not too large and provided the irritation it exercises on the circumjacent tissues is not too severe, qualitatively speaking; the process of encapsulation of a foreign body proceeds in such a way that the extraneous material is rapidly surrounded by an accumulation of cells that later form a direct connection of an organic character with the serosa, and in this manner form so-called vascularized tissue; as soon as this new tissue is formed all around the foreign body, further irritation of the peritoneum is rendered impossible and the invading material, being completely encapsulated, is rendered perfectly innocuous. I shall not enter into a discussion of the origin of these cells here; it may suffice to call attention to the fact that they always surround any foreign body that enters the peritoneal cavity and that they also possess the function of surrounding any other irritant that may affect the inner surfaces of the peritoneum.

The question as to the causes of this high degree of "plasticity" that we have shown to be inherent in the peritoneum is important and requires explanation. Wegner attributes it directly to the normal transudation (irrigation) that occurs in the peritoneum; he says: "The vessels of the serosa continuously pour out a certain amount of fluid which contains all the nutritive elements of the blood; this fluid is capable of nourishing any cellular elements that may be floating free in the peritoneal cavity or that may be lying on the serosa, until such time as new blood-vessels can be formed and nutriment can be carried to the new formed tissue through this channel." During the last two decades many investigators have studied this question from both the experimental and the histologic points of view; from a practical standpoint, the formation of peritoneal adhesions and the participation of the endothelial lining of the serosa in the process are of special interest and importance. I am, however, obliged to omit any detailed description of these investigations here.

Further, Lennander has come to the conclusion that the bowel, when acutely inflamed, is, contrary to the generally accepted opinion, just as insensitive as the bowel in its normal state. He believes that diseases of the abdominal organs and bowels are accompanied by pain only when they give rise to mechanical or inflammatory (toxic or infective) irritation of the intercostal, lumbar, or sacral nerves underlying the parietal layer of the peritoneum. Colic, Lennander declares, is caused by sudden tension of the nerves in the parietal peritoneum in connection with acute distention of some part of the alimentary canal. "Directly those parts of the bowel (viz., cecum, ascending and descending colon, hepatic and splenic flexures of the colon) which have no mesentery or which are fixed to the parietal peritoneum by peritoneal folds or adhesions are more than usually distended, the parietal peritoneum is drawn toward them, and is, perhaps, in part stretched over them. No condition of the parietal peritoneum seems to cause so much pain as sudden tension."

The above remarks are worthy of note, and are given for what they are worth without any criticism.

ABDOMINAL DROPSY (Ascítes[1]).

PATHOGENESIS.

Ascites signifies the accumulation of fluid in the peritoneal cavity.

Under physiologic conditions the peritoneal cavity contains only a capillary layer of fluid—in other words, it is "empty." Departure from this normal condition can occur only when the equilibrium between transudation and absorption of fluid is disturbed; Wegner has shown definitely that under normal conditions this equilibrium is carefully adjusted, so that no more fluid transudes than is subsequently absorbed. In pathologic conditions the transudation of fluid may be normal while absorption is diminished, or, conversely, absorption may be normal while transudation is excessive, or, finally, transudation may be increased while absorption is diminished.

In the preceding section attention was called to the factors which influence the absorption of fluid from the peritoneal cavity, and the reader should refer for the details to the paragraphs devoted to the physiology of absorption; this section will be confined to a sketch of a few of the clinical conditions characterized by the accumulation of fluid in the peritoneal cavity. The accumulation of fluid in the peritoneal cavity occurs principally in the following conditions—the origin of the ascites in these states has already been discussed and is well understood:

(*a*) So-called ascites from stasis, where the pressure in the whole of the venous system is generally increased; this increase of pressure may be due either to certain diseases of the heart or the lungs, or, in particular, to local increase of pressure in the portal system; the latter is very apt to occur in obstruction in the trunk or in the small intrahepatic branches of the portal vein. The increase of pressure in the veins, in the first place, interferes with the absorption of fluid from the peritoneal cavity by the blood-vessels, and also directly by the lymphatic vessels, owing to the fact that the pressure is increased in the innominate veins, and, secondarily, in the thoracic duct; in the second place, increased blood-pressure in the veins and capillaries increases transudation of fluid into the peritoneal cavity. These two factors combined lead to the development of ascites in cases of general venous stasis.

(*b*) Another form of ascites is clinically as frequent as the foregoing one, namely, that due to acute or chronic peritonitis; in these inflammatory conditions exudation of blood-serum occurs from the affected

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[¹ Ascites = ἀσκίτης, from ἀσκός, a wine-skin. The termination -ιτης is, like the more common -ιτις, a feminine adjectival termination agreeing with νοσος, disease, understood, so that ascites is an abbreviation for ἡ ἀσκίτης νοσος, the "wine-skin disease," meaning thereby the disease in which the abdomen looks like a tight wine-skin. For this original and strict meaning of the termination -ιτις the reader should consult Dr. T. Buzzard's remarks apropos of neuritis in the *Transactions of the Pathological Society of London*, vol. xl., p. 347.—ED.]

surfaces of the peritoneum and accumulates in the cavity. (Space does not allow of a description here of the finer mechanism of this process, which, by the way, has been the subject of much controversy.) The question has been raised why, in inflammatory conditions of the peritoneum which are uncomplicated by stasis or by increased venous pressure, fluid should accumulate in the peritoneal cavity. It has been argued that the accumulation of fluid under these circumstances must be attributed to the closure of the lymph stomata in the peritoneum by inflammatory products. Von Recklinghausen, however, directs special attention to the fact that in some cases of inflammation and of carcinosis of the peritoneum he has repeatedly found the lymphatic vessels in the diaphragm and the main trunks of these channels on the thoracic side of the diaphragm to be completely occluded with pathologic material without any evidence of abnormal accumulation of fluid in the peritoneal sac. It is probable, therefore, that some other factors must be concerned in inflammatory ascites that favor the accumulation of fluid in the peritoneal cavity. It is possible that the absorptive powers of the blood-vessels in the inflamed area of the peritoneum are reduced, or that so large a quantity of fluid is poured out in inflammation of the peritoneum that the absorptive powers of the blood-vessels that are intended to remove this fluid are overtaxed, and ascites consequently develops.

(c) The pathogenesis of ascites in renal disease and in cachexia is difficult to interpret. Even if we assume that transudation is increased as a result of certain blood changes in these diseases, there is no satisfactory reason why this fluid is not absorbed, even though the amount poured out is increased, for we know that the absorptive powers of the peritoneum are truly enormous. In this, as well as in the forms of ascites due to stasis and to inflammation, we are forced to the conclusion that the absorptive powers of the peritoneum become insufficient to cope with the enormous amount of fluid poured out, and that, consequently, the accumulation of fluid in the peritoneal sac—the ascites—persists.

(d) Quincke has described a special and comparatively rare form of ascites in young girls (ascite des jeunes filles) which he explains in a special way; the development of this form of ascites is slow, painless, and hardly noticed by the patients, who are, as a rule, growing young girls; this form of ascites usually disappears with greater or less rapidity when the menses appear (compare also the etiology of peritonitis). Quincke's view is that with the growth and development of the ovaries a condition of hyperemia and of hypersecretion develops in the adjacent peritoneum, which, however, disappears as soon as the first follicle bursts; in other words, he maintains that this form of ascites is due to a nervous form of hypersecretion.

(e) Stilling describes a case of ascites which he calls " neuropathic " in a patient of forty years of age who suffered from marked ascites and enlargement of the liver, unaccompanied by any considerable edema of the lower extremities; in this case there were, for some unknown reason,

marked degenerative changes in the right splanchnic nerve; the changes in the left splanchnic nerve were unimportant. Taking into consideration well-known physiologic experiments, Stilling attributes the dropsy to the degeneration of the splanchnic nerve; he believes that the lesions of the heart and lungs discovered in addition at the autopsy do not explain the early and isolated appearance of marked ascites and enlargement of the liver. This supposition cannot be accepted offhand, since, to quote his own words, "the heart was enlarged, the right ventricle dilated, and its musculature pale and hypertrophied; the left ventricle dilated and its muscles weak"; again, "there was fairly marked emphysema of the lungs." Moreover, the first symptoms complained of by the patient nine months before her death were difficulty in breathing, cough, expectoration, and palpitation; after some length of time only there appeared the gradual increase in size of the abdomen. Such an extremely complicated and many-sided case can hardly serve as the basis of a pathologic conception which remains to be proved.

(*f*) Finally, as Klebs has shown, increased transudation of fluid into the peritoneal sac may occur as the result of changes in the peritoneal endothelium; Klebs has reported a case in which ascites developed as the result of such changes, which assumed the character of fatty degeneration of the endothelial layer of the peritoneum. The actual occurrence of ascites due to such degenerative changes in the endothelial cells must be regarded as a curiosity.

CONSTITUTION OF THE ASCITIC FLUID.

The ascitic fluid, in the overwhelming majority of cases, is derived from the blood-vessels; it is only in rare cases that there is evidence to show that the fluid is derived from the chylous vessels.

F. A. Hoffmann first noticed that ascitic fluid contains in solution practically all the soluble substances of the blood, which, as the former is usually derived from the latter, is not surprising. Of the blood proteids, albumin and globulin, but no albumose nor peptone (von Jaksch), are found; in addition, urea, xanthin and hypoxanthin, guanin, and uric acid are all present, though not constantly, and at most only in minimal quantities. There is some doubt as to the presence of sugar in pathologic fluids, particularly ascitic fluid, when derived from the blood and not from the chyle. The more recent investigations of such authors as von Jaksch, Pascheles, and Pickardt (see the literature on the subject in the paper by the latter author) all answer this question in the affirmative; Pickardt was able to demonstrate the presence of levulose in addition to dextrose in several instances.

The fluid in pure ascites from stasis is perfectly clear, yellowish or greenish-yellow, and alkaline in reaction. Quincke, who has carried out methodic microscopic examinations of the fluid exudates of the serous cavities, has demonstrated the presence of lymph-cells and of larger rounded or polygonal cells with fine granular protoplasm (with a nucleus, and occasionally the formation of vacuoles) in all transu-

dates, even in those that are perfectly clear and appear to contain minimal numbers of cellular elements; it is probable that these are the endothelial cells of the peritoneum. These clear fluids are specially frequent in ascites due to simple, venous stasis. Red blood-corpuscles, sometimes in small numbers, at other times in greater numbers, are also constantly found in these effusions.

In inflammatory forms of ascites the exudate, in addition to flakes of fibrin in more or less abundant quantities, and other large or small masses of coagulated material, always, of course, contains large numbers of pus-cells when the exudate becomes purulent; when hemorrhagic, there are large numbers of red blood-corpuscles; in carcinomatous affections of the peritoneum it occasionally contains cancer cells and "*Leydenia gemmipara.*" In addition, the different species of bacteria enumerated in the section on the Etiology of Peritonitis may be found in the ascitic fluid—for instance, tubercle bacilli, streptococci, staphylococci, diplococci, etc.

[*Inoscopy* is a new method of detecting micro-organisms when present in small numbers in serous effusions, and is specially applicable to the detection of tubercle bacilli. The clot which forms on standing acts as a kind of filter and entangles micro-organisms. When coagulation does not occur spontaneously, it can be induced by the addition of salted plasma. The clot is treated with gastric juice, which dissolves it, but does not affect the micro-organisms; the emulsion thus obtained is centrifugalized, and the deposit examined by ordinary staining methods (Jousset).[1] Maude Abbott,[2] working in Adami's laboratory, found the diplococcal form of the colon bacillus (described by Adami in connection with hepatic cirrhosis) in the ascitic fluid of cirrhosis; in one case of cirrhosis complicated by perihepatitis and perisplenitis it was present in the fluid withdrawn on three occasions during life.—ED.]

Finally, in peritonitis due to intestinal perforation, a great variety of substances derived from the intestinal contents may be found in the ascitic fluid.

From a practical point of view it is exceedingly important to decide whether transudates that are derived from simple venous stasis can be distinguished from exudates derived from inflammatory affections of the peritoneum; in order to be of practical value in the differential diagnosis it is essential that any given differences between the two varieties of ascitic fluid should be constant and quite characteristic of either variety; if this can be shown to be possible, a differential diagnosis between ascites from stasis and ascites from inflammation could be made from a simple examination of the fluid obtained from the peritoneal cavity by paracentesis. Numerous examinations on this point have been made by a number of different investigators; Ott has collected the literature on the subject up to 1896. As it is quite impossible to enter in full detail into all the controversies on this subject,

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[1] Jousset, *Arch. de Méd. Exper. et d' Anat. Path.*, 1903, vol. xv., p. 289.
[2] M. Abbott, *Jour. Path. and Bacteriol.*, vol. vi., p. 315.

a brief sketch only of the consensus of contemporary opinions will be given.

The chief point to be determined is whether the percentage amount of albumin present in the aspirated fluid combined with the specific gravity justifies an opinion as to the nature of the primary process in the peritoneum responsible for the transudation of the fluid we are analyzing. Runeberg, who has repeatedly studied this problem, is most emphatic, and without any hesitation answers this question in the affirmative, especially in his latest publication. He attaches most importance to the percentage of albumin. According to Runeberg, the three main pathogenetic groups of ascites—namely (1) the inflammatory form ; (2) that due to stasis ; (3) that due to nephritis—show marked differences in this respect, which, moreover, he regards as perfectly constant. An inflammatory exudate will be found to have from 4 to 6 per cent. of albumin ; transudate due to stasis, from 1 to 3 per cent.; and a purely hydremic transudate, at most 0.5 per cent. of albumin. Deviations from this standard occasionally occur ; in cases of ascites due to stasis especially the amount of albumin may be so increased that one might be tempted to diagnose an inflammatory exudate due to some form of peritonitis. Runeberg argues that deviations of this character must be attributed to a combination of one or more of the possible factors causing the outpouring of fluid into the peritoneal cavity. In long-continued venous engorgement, for instance, certain well-defined secondary changes seem to develop in the endothelium of the peritoneum, such as desquamation of the cells and thickening of the serous lining, both of these factors rendering the membrane more permeable for albumin. Many combinations of the different causes of ascites can be imagined, so that it is not surprising to find that the percentage of albumin may be more or less inconstant in such complicated cases ; for instance, in carcinoma of the portal area with ascites from stasis there may originally be the usual amount of albumin, amounting to about 1 to 2 per cent.; when, later, the disease becomes complicated by the secondary development of carcinosis of the peritoneum, the percentage of albumin in the ascitic fluid immediately rises to 4 to 5 per cent.; or, again, in a simple case of ascites due to renal disease the amount of albumin characteristic of hydremic ascites—viz., about 0.5 per cent.—is present, but when, later in the course of the disease, cardiac weakness comes on, the transudate changes its character and comes to resemble ascitic fluid due to venous engorgement, with a percentage of albumin increased to 2 per cent. Runeberg lays special stress on the fact that the percentage of albumin in ascitic fluid must be considered in the same manner as any other individual sign, and that all the concomitant factors and features of the case must be taken into consideration before basing any diagnostic conclusions on this single sign. In addition to the percentage of albumin, the specific gravity of the ascitic fluid is of some diagnostic importance in arriving at an opinion as to its origin ; a low specific gravity—1015 and less—indicates ascites from hydremia or stasis ; a higher specific gravity, ascites from inflammation.

Runeberg's positive statements, which practically correspond with those previously made by Reuss, are opposed by a considerable number of observers, among whom F. A. Hoffmann, Citron, Ott, and Pickardt may be specially mentioned. These authors and some others, it is true, arrived at the general conclusion that the percentage of albumin found in hydremic transudates and in ascites due to stasis was lower, as a rule, than the percentage of albumin found in inflammatory ascitic fluids; they also determined that the specific gravity of the fluid was generally higher in the latter than in the former varieties; but they were unable to draw a hard-and-fast line between the two kinds of ascitic fluid, either in the percentage of albumin or in the specific gravity. All the different observers quote figures to show that neither the percentage of albumin nor the specific weight of the ascitic fluids examined in different cases corresponded with the pathogenetic cause of the ascites, nor with the figures that would *a priori* have been expected from their pathogenesis. Ott agrees with other observers in the following conclusions: (*a*) No definite percentage of albumin corresponds to any one type of disease nor to any one form of exudate or transudate; (*b*) no definite conclusions as to the character or the origin of the ascitic fluid can be drawn from the percentage of albumin; further, as the specific gravity is quite inconstant, there are no grounds to justify a diagnosis of the character of the pathologic fluid present in the peritoneal cavity from these two factors, nor in drawing any conclusion as to the nature of the primary cause of the ascites.

Several observers (Moritz, Paijkull, Umber, Staebelin) have indicated the presence of special albuminous substances—*e. g.*, in the product of exudation one finds an albumin (mucin of the serosa, nucleo-albumin), precipitable by acetic acid, which is absent from the product of transudation.

Further, O. Rosenbach stated that even small quantities of iodin or its salts or salicylic acid given by the mouth appear in transudations quickly and in large quantities, while they appear, on the other hand, in exudations as traces only. He recommends this simple fact as a means to diagnosis.

A further distinction between transudation and exudation is believed to exist in the morphologic characters of the fluids. Microscopically, according to Alfred Wolff, one can distinguish two great groups: (1) That containing polynuclear leukocytes only with a few isolated erythrocytes; (2) that containing lymphocytes and red blood-corpuscles (no polynuclear leukocytes). Occasionally one finds a transition between (1) and (2), with polynuclear leukocytes and lymphocytes both present. Group (1) is found in cases which may be clinically described as due to exudation, even when they present macroscopically a serous appearance; group (2) presents chiefly the characteristics of transudation.

[*Cytodiagnosis* of peritoneal effusions, or the diagnosis of the cause and nature of the fluid in the peritoneum from the character of the cells found in it, cannot be said to have advanced beyond the initial stage. In the case of the pleura, the general principle has been estab-

lished that lymphocytes in predominating numbers point to tuberculosis ; polymorphonuclear leukocytes, to an acute inflammatory process ; and endothelial cells, to a mechanical effusion.   But this generalization cannot be considered established as regards ascites, and the published results are somewhat scanty.   Tuffier and Milian [1] found that the fluid in tuberculous peritonitis contained 78 per cent. of mononuclear leukocytes and lymphocytes, and Dopter and Danton [2] confirmed this.   Achard and Loepers [3] found endothelial cells in chronic peritonitis due to various factors and in ascites due to mechanical causes.   In 2 cases of lactescent ascites occurring in common or portal cirrhosis of the liver the cells were chiefly mononuclear (Souques,[4] Achard and Laubray [5]), but in a case of ascites due to cirrhosis Dopter and Danton found a majority of polymorphonuclear leukocytes and a few endothelial cells.

Dock [6] found that in carcinomatous ascites there were more cells showing mitoses than in simple or in tuberculous effusions, and that in the former case the mitoses were often atypical.   In melanotic sarcoma of the liver the ascitic fluid, though clear, may contain cells with pigment-granules inside them (Hektoen and Herrick [7]).—Ed.]

Special attention has of late years been given to an interesting form of ascites which, though known for over two centuries, has only recently been thoroughly and exhaustively investigated.   I refer to—

ASCITES CHYLOSUS ET ADIPOSUS.

In this form of ascites the fluid consists of a milky, fatty, chylous material or of a liquid resembling chyle.   Poncy, in 1699, published the first case of chylous peritoneal exudate ; Busey has reviewed the whole literature up to 1889, and Bargebuhr up to 1892.   Within the last six years there have been a number of contributions to this subject, and the literature of chylous ascites is even more extensive than at the time of Bargebuhr's summary.   The total number of cases of ascites chylosus is, of course, swelled by those not officially reported, as every physician with large experience has unquestionably seen an occasional case of this condition ; personally I have notes of 5 cases.

[In 1900 H. Batty Shaw [8] collected 115 cases of milky fluid in the abdomen, 68 of which were chylous, 27 chyliform (fatty), 1 lactescent non-fatty, and in 19 the nature of the fluid was not established.—Ed.]

Poncy correctly declared the fluid he obtained from puncture of the abdomen to be chylous in character.   After the announcement of Poncy's discovery a number of theories and explanations were advanced as to the origin and the mode of formation of this fluid.   Quincke advanced our knowledge of this subject considerably by distinguishing two groups

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[1] Tuffier and Milian, *Compt. Rend. Soc. Biol.*, April 27, 1901.
[2] Dopter and Danton, *Bull. et Mem. Soc. Med. des Hôp.*, Paris, July 12, 1901.
[3] Achard and Loepers, *ibid.*, Paris, July 12, 1901, p. 295.
[4] Souques, *ibid.*, Paris, March 21, 1902.
[5] Achard and Laubray, *ibid.*, Paris, March 21, 1902, p. 295.
[6] Dock, *Amer. Jour. Med. Sci.*, June, 1897.
[7] Hektoen and Herrick, *ibid.*, September, 1898, vol. cxvi., p. 255.
[8] H. Batty Shaw, *Jour. Path. and Bacteriol.*, vol. vi., p. 339.

of milky, fatty transudates which are fundamentally different. In the first group, which he called chylous ascites, the fluid is rendered milky and fatty by the actual presence of chyle in the peritoneal cavity; in the second group, which he termed ascites adiposus (chyliform or pseudochylous), the peculiar appearance of the fluid is not due to the presence of chyle, but to the admixture of fat, which appears to be derived from disintegrating cells.

The most characteristic feature of both these forms is the constitution of the ascitic fluid. This fluid is often white in color, occasionally with a tinge of yellowish-bluish or rose color; it is not transparent, and looks either like thick milk or occasionally like a sample of watered milk. If the aspirated fluid is allowed to stand, a thick sediment may settle at the bottom of the vessel, while a still thicker layer of creamy fatty material collects on the top of the liquid. If the fluid is shaken with potassium hydrate solution or with ether, it may clear up to a certain extent. The reaction of the fluid is alkaline. [In 1 case (Rotmann) it was acid.—ED.] Analysis reveals the presence of fat beyond doubt, the amount present being usually about 1 or 2 per cent., occasionally 4 per cent., and sometimes even as much as 5 per cent. In addition there are always some albuminous bodies in solution.

Senator, who indorses Quincke's classification, considers the following features as sufficiently characteristic for the differentiation of the chylous and the adipose forms of ascites. He says that in the former variety (chylous ascites) the microscope shows the presence of very fine droplets of fat scattered throughout the liquid, like particles of dust; apart from these there are no other fat-containing structures, or, at best, a very small number of cells in a state of fatty degeneration; in addition, chemic examination always shows the presence of sugar in this form. In ascites adiposus, on the other hand, according to Senator, no sugar can be found, while, in contrast to the other form of ascites, there are a large number of cells containing droplets of fat or showing signs of commencing fatty degeneration floating in the ascitic fluid, besides the usual finely distributed fat-particles which are thus common to both forms of ascites. The cells seen in the latter form are in part lymph-cells, in part carcinomatous, and in part endothelial cells derived from the peritoneum. The presence of degenerated cells in the one form and their absence in the other are unquestionably correctly observed by Senator, and this point may be made use of in distinguishing them.

[Renvers and Cervello[1] suggest that in malignant disease of the peritoneum toxic bodies manufactured by the cells of the growth produce degeneration in the cells in the ascites and so produce fatty ascites. The same hypothesis—of fatty degeneration due to a toxin—might be employed to explain fatty ascites in tuberculous peritonitis.—ED.]

The presence of sugar in the one form and its absence in the other are, however, denied by some investigators; as a matter of fact, a number of writers (Pascheles, von Jaksch, Pickardt, and others) have recently shown that sugar, and not merely reducing substances, is usually pres-

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[1] Renvers and Cervello, *Riforma Med.*, 1897, p. 278.

ent in all forms of exudates or transudates derived from the blood. [In S. Martin's case of true chylous ascites there was no sugar.

Milky Non-chylous (Non-fatty) Ascites.—This form has recently been distinguished from the true chylous and the chyliform (fatty) varieties. The first case was described by Lion[1] as lactescent, non-chylous ascites. The fluid is exactly like that in the other forms of milky ascites to the naked eye, but neither microscopically nor chemically does it contain fat. Further, it does not separate into layers on standing like chylous ascites, and retains its characters on filtering. The opalescence is not due to bacteria, for it may be quite sterile; it was thought by Lion to be due to a proteid allied to casein, and he refers to 6 cases of opalescent ascites from which Hammarsten obtained muco-albumin. Achard and Laubry and Souques have described a nucleo-albumin as the cause of the opalescence. In a case examined by R. Hutchison[2] the character of the lactescent ascites was due to the presence of some form of nuclein or paranuclein containing proteid, and not to lecithin or mucoid substances. Another explanation of the milky, non-fatty ascites is that it depends on a lactescent, limpid, non-fatty condition of the blood-serum, which is different from the fatty blood-serum seen in the lipemia of diabetes, and may be physiologic in some individuals after a hearty meal; it may also be pathologic, and, according to Castaigne,[3] is specially related to epithelial change, such as tubal nephritis, in the kidney.—ED.]

The pathogenesis of fatty ascites varies in different cases and differs from the causes of chylous ascites.

The presence of fat in adipose ascites is not due to the entrance of chylous fluid into the peritoneal cavity; the fat, moreover, is derived from other sources—in the great majority of cases, from cells that are in a condition of fatty degeneration. The origin of these cells may vary. In the majority of cases the cells are in a state of fatty degeneration, owing to the existence of tuberculous peritonitis; sarcoma and carcinoma more frequently cause fatty degeneration of cells (Quincke, Brieger). Ascites adiposus has, however, also been observed as the result of fatty degeneration of the endothelial cells of the peritoneum in ascites due to stasis, in cirrhosis of the liver, and in heart diseases. It is conceivable that in some instances fatty degeneration may occur in lymph- or pus-cells which happen to be present in the peritoneal cavity. Micheli and Mattirolo, and later Gross, have succeeded in proving lecithin or lecithin-like bodies the cause of the turbidity in pseudochylous effusions. The last-named author thinks that it is perhaps more often present than has been hitherto suspected. The origin of the lecithin must be sought in the disintegration of cells, whether of leukocytes in ascitic fluid or of carcinomatous cells. It is not yet decided whether the accumulation of an abnormal amount of fat in the blood (lipemia) is a cause of ascites adiposus. The only case of

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[1] Lion, *Arch. de Méd. Exper. et d'Anat. Path.*, vol. v., p. 826.
[2] R. Hutchison, *Trans. Path. Soc.*, vol. liii., p. 274.
[3] Castaigne, *Arch. Gén. de Méd.*, June, 1897, p. 666.

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this kind on record (Popham) does not prove this point with any certainty, since, in addition to lipemia, chronic peritonitis was present; it is clear that the latter condition could account for the presence of fat in the ascitic fluid, for it has been shown that occasionally the endothelial cells of the peritoneum may undergo fatty degeneration and pour the product of their disassimilation into the ascitic fluid present in the peritoneal cavity. [Osler accepts lipemia as a causal factor, especially in patients on a milk diet.—ED.]

In genuine chylous ascites the fat found in the peritoneal cavity is really derived from chylous fluid. For a long time this was doubted, particularly by French writers on the subject. Of recent years, however, there has been direct experimental proof of the suggestion that chyle may actually be poured into the peritoneal cavity, and in this way give rise to genuine chylous ascites; thus, a subject was fed exclusively on milk and butter and the fat-content of the ascitic fluid determined; it was found that the quantity of fat present in the ascitic fluid corresponded exactly to the butter ingested (Straus). In other cases the presence of erucic acid $(C_{22}H_{42}O_2)$ could be detected in the ascitic fluid after it had been given to patients with chylous ascites; the latter experiment is particularly conclusive, because erucic acid is not a normal constituent of the human body (Minkowski). The microscopic appearance, moreover, of the fat-droplets in the chylous ascitic fluid corresponds exactly with the microscopic appearance of the fat-droplets seen in the chyle.

In a certain proportion of the cases the fat from the chyle gains direct entrance into the peritoneal cavity through tears in the lymph- or chyle-vessels. Cases have been recorded in which the thoracic duct was found ruptured above the receptaculum chyli; in other instances some of the lacteals were torn, while in other cases a chylous cyst or occasionally a lymph-gland was found ruptured. In a certain number of instances where the solution of continuity in the walls of the chyle-carrying vessels was so small that it could be detected only by microscopic examination the lesion was due to entozoa. In another group of cases there is no rupture of the vessel-walls, but merely stasis of chyle or some impediment to its flow; here, as in the case of the blood-vessels, a true transudation of chylous fluid into the peritoneal cavity must be assumed, the chyle passing through the uninjured walls of the chylous vessels. In cases belonging to the latter category an examination of the peritoneum, particularly of the small intestine and of the mesentery, shows the presence of an arborescent network formed of white injected vessels which are occasionally dilated and are consequently clearly visible postmortem.

The rupture of larger chylous vessels is often brought about directly by some external or internal trauma (lifting heavy weights, being run over, whooping-cough, miscarriage). More frequently the rupture is the direct result of the stasis of lymph brought about by the presence of some obstruction to the flow of lymph through the lymphatic vessels; blocking anywhere in the lymphatic system of the abdomen may pro-

duce this result. It need hardly be mentioned that obstruction to the flow of lymph may produce the transudation of chylous fluid into the peritoneal cavity without rupture.

Obstruction to the flow of lymph has been found in the following locations : in the left innominate vein (Nothnagel), in the left subclavian vein, in the thoracic duct, in the receptaculum chyli, in the lymph-glands, and in the smaller lymphatic vessels.

The obstruction to the flow of lymph consists in an obturation of the lymph-vessels in the affected district ; the lumen of the lymph-channel itself may be occluded, or the lymph-channel may be compressed from without, chiefly by tumors, particularly lymphatic glands infiltrated with carcinoma ; the latter condition may lead to compression of the subclavian or the left innominate vein, of the thoracic duct, or of smaller lymphatic vessels on the peripheral side of the receptaculum chyli. Carcinomatous proliferation may also be found directly in the smaller lymph-channels or may originate directly from these vessels—this explains the relative frequency of chylous ascites in carcinosis of the peritoneum. It is self-evident that numerous other factors, provided they exert the same effect, namely, compression of the lymph-vessels, may also lead directly to the development of chylous ascites ; among these agencies may be mentioned some forms of chronic peritonitis other than carcinosis, which can easily lead to mechanical compression of the lymphatic vessels. In some cases it has been found that the flow of lymphatic obstruction was due to endophlebitis obliterans in the subclavian vein. As a curiosity, a published case, in which a concretion in the cistern of Pecquet obstructed the flow of lymph, may be mentioned.

I wish to emphasize the fact, however, that obstruction of the thoracic duct does not necessarily or constantly produce chylous ascites. Cases have been recorded of obliteration of the thoracic duct in which there was no chylous ascites. This fact is explained by the investigations of Monroe, Magendie, and Dupuytren, who showed that the flow of lymph can be carried on through collateral channels when the thoracic duct is occluded. In fact, von Recklinghausen has shown that stasis of lymph occurs much more readily when a large number of the lymph-channels are occluded in a circumscribed area than when the main lymph-duct is obliterated ; this probably explains the great frequency of chylous ascites in carcinosis of the peritoneum.

[Hemorrhagic ascites may occur in tuberculosis, malignant disease of the peritoneum, and in cirrhosis of the liver. In some instances of ascites in cirrhosis the hemorrhagic condition occurs only on the second tapping, and is not due to concomitant tuberculosis, but to traumatism and hemorrhage from vascular adhesions at the previous paracentesis (Barjon and Henry[1]). Blood-stained fluid, usually small in amount, may be found in the peritoneum in various conditions, such as strangulation of the intestine, acute pancreatitis, twisted ovarian cysts, etc.

Hemoperitoneum or extravasations of blood into the peritoneum

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[1] Barjon and Henry, *Lyon Médical,* June 19, 1898.

occur from traumatism and rupture of viscera, such as the liver, spleen, mesentery, etc., in extra-uterine gestations, ruptured aneurysms, etc. In rare instances no cause is forthcoming; in a case recorded by Cheeseman and Ely [1] recurrent hemorrhagic effusions occurred in both pleuræ and in the peritoneum. The abdomen was tapped 43 times in five years, and then underwent spontaneous cure.

Effusion of bile into the peritoneum, if aseptic, does not set up inflammation. Dévé [2] has collected 15 cases of choleperitoneum due to rupture of a hydatid cyst, which is in communication with a bile-duct, in the peritoneum. The abdomen swells, usually slowly, but sometimes rapidly, and the effusion has a great tendency to reaccumulate, and requires frequent tappings. In spite of the great powers of absorption of the peritoneum no jaundice occurs.

In rare instances ascitic fluid is brown, from the presence of melanin; this has been noted in some cases of melanotic sarcoma of the liver (W. Legg,[3] Senator [4]).—Ed.]

CLINICAL PICTURE OF ASCITES.

The accumulation of ascitic fluid in the peritoneal cavity may give rise to certain functional disturbances, and when the amount of fluid is considerable, its detection is, therefore, not difficult. [When there is considerable edema of the abdominal wall, the presence of ascitic fluid may be very difficult to detect.—Ed.] Small quantities of ascitic fluid, on the other hand, are often difficult to detect, and, moreover, produce no functional disturbances or discomfort. In addition to the quantity of ascitic fluid, its distribution in the peritoneal sac is of importance; the clinical picture will necessarily vary accordingly; in other words, it will be different when the ascites is localized or encysted than when it is free in the general cavity of the peritoneum.

When the amount of ascitic effusion is very large, the abdomen is usually correspondingly distended, and, occasionally, the abdominal distention may be enormous; a characteristic feature of abdominal distention from ascites is that the greatest degree of distention is always seen in the flanks when the patient is lying on his back; this is quite apparent even when the whole of the abdomen is enormously distended, and can be explained by the tendency of the accumulated fluid to follow the laws of gravity and to sink to the most dependent portions of the peritoneal cavity. This sign is of value in the differential diagnosis between ascites and tympanites, for, in the latter condition, the middle of the abdomen is, as a rule, the most prominent and distended part. In many cases, of course, it is quite impossible to determine, from the shape of the abdomen alone, whether the distention is due to the accumulation of fluid or of gas. The skin over the abdomen is tense and

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[1] Cheeseman and Ely, *Amer. Jour. Med. Sci.*, August, 1899, p. 166.
[2] Dévé, *Rev. de Chir.*, July 10, 1902, p. 67.
[3] W. Legg, *Trans. Path. Soc.*, vol. xxix., p. 225.
[4] Senator, *Charité Annalen*, 1890, vol. xv., p. 261.

tightly stretched, shiny, and occasionally marked with striæ ; the umbilicus, even in the absence of subcutaneous edema, is usually obliterated ; sometimes it protrudes above the level of the abdomen and looks like a prominent vesicle. In long-standing cases the subcutaneous veins are generally distended and dilated, and are plainly visible on the abdominal surface, and this occurs even when the portal circulation through the liver is unobstructed. On palpating the abdomen in the ordinary way a most marked sensation of fluctuation is felt by the hand ; and if the abdominal walls are not too fat, a distinct wave may be seen to pass from one side of the abdomen to the other. The percussion-note is very dull, and in the dependent portions may be similar to the note obtained by percussing the thigh ; in the upper and median portions of the abdomen the percussion-note may be loud and may have a tympanitic ring. When the patient changes his position from one side to the other, the note on the side opposite to the one occupied by the subject becomes clearer and louder.

Considerable difficulty may occasionally be experienced in determining whether the distention of the abdomen with fluid is due to ascites or to the presence of an enormous cyst of the ovary, liver, or mesentery.

[Powell[1] and Hatch[2] have referred to cases where a large hepatic abscess was thought to be ascites. In Hatch's case the patient was a boy aged nine years.—ED.]

Some recorded cases show that a huge soft peritoneal lipoma has also occasionally been mistaken for ascites.

[Adami[3] has collected 42 cases of retroperitoneal lipomata. A third of the cases grew from the perirenal fat, but lipomata may arise in the mesentery or elsewhere in the abdomen. Females are more often affected ; it usually comes on in middle or later life, and progresses slowly.—ED.]

In the case of a lipoma, however, the sensation of fluctuation is different, and, in addition, the percussion-note elicited when the patient changes his position is not so typical in its changes as in the case of free ascites ; the same applies to the percussion-note over a large abdominal cyst. In ascites, as we have seen, the percussion-note is specially dull in the dependent parts of the abdomen—*i. e.*, the flanks when the patient is lying on his back ; in the case of a cyst, we are very apt to find that in one or the other side of the abdomen or in one of the other flanks, when the patient is in the dorsal position, there is a resonant tympanitic note ; sometimes a resonant tympanitic note may be obtained on both sides ; this is, of course, due to the presence of intestine in these portions of the abdomen which are not occupied by the cyst. Special attention, however, must be called to the fact that in ascites the same resonant percussion-note may occasionally be present in the dependent portions of the abdomen from the presence there of coils of intestine distended with gas. The shape of the abdomen may also be somewhat irregular in encysted ascites, and the abdomen may be distended more in one direction than in another. Eichhorst calls attention

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[1] Powell, *Indian Med. Gaz.*, February, 1898.   [2] Hatch, *ibid.*, August, 1898.
[3] J. G. Adami, *Montreal Med. Jour.*, January–February, 1897.

to the fact that in ascites fluctuation can usually be elicited even above the level of the fluid in the abdomen, whereas this is not so in ovarian cysts; in other words, in abdominal ascites fluctuation can be obtained not only in the dull area, but also in areas where the note is tympanitic, whereas in ovarian cysts the fluctuation is strictly limited to the area of absolute dulness. In many cases it is quite impossible to arrive at a positive differential diagnosis between the two conditions until an exploratory laparotomy is made.

Enormous accumulations of fluid cause trouble and distress *per se.* They may even cause pain from resulting pressure and tension of the skin of the abdominal walls; in addition, there is always a feeling of weight and fulness in the abdomen; the intestine is compressed by the large amount of fluid, and, in addition, the diaphragm is forced upward so that the thoracic space becomes limited, and even dangerous degrees of dyspnea and cyanosis may be produced by compression of the lungs.

Quite frequently, in addition to ascites, even if it is inflammatory or hydropic, the lower extremities are swollen and edematous; as a rule, the swelling of the limbs is less than the ascites, although occasionally the amount of edema may be very considerable. This edema of the legs is usually regarded as evidence of interference with the return of blood through the inferior vena cava, owing to the fact that this vessel is compressed within the abdomen by the accumulated ascitic fluid. This explanation holds good within certain limits; it is supported by the presence of anastomotic venous channels on the surface of the abdomen (epigastric, mammary). Giovanni has advanced an entirely different explanation, however, which seems to be true for many cases of edema of the legs; he assumes that in many cases of ascites there is also cardiac insufficiency, and that this is often directly responsible for the swelling of the limbs; in addition, of course, this tendency is enforced by the pressure exerted on the intra-abdominal veins by the ascitic fluid in the peritoneal cavity; in some of the cases the cardiac insufficiency that Giovanni holds responsible for the edema is the result of the ascites or is a concomitant symptom in the general morbid condition of which ascites is only one of the signs.

[In cirrhosis of the liver edema of the feet often appears when there is no ascites. It may be due to several factors, such as cardiac weakness, or possibly, in some instances, alcoholic neuritis, but it is probable that it is often toxic and due to the action of a poison having a lymphagogue action (Hale White [1]).—ED.]

In moderate degrees of ascites the well-known percutory signs— *i. e.,* dulness in the dependent portions of the abdomen, especially in the flanks—can be obtained; as soon as the patient changes his position, the dulness also shifts, so that it disappears from one place, only to appear in some other dependent part of the abdomen; at the same time the flanks of the patient are seen to bulge slightly, and an indefinite sense of fluctuation may be obtained on palpation; occasionally, however, the latter sign is quite absent. Moderate degrees of ascites rarely

[1] Hale White, *Clinical Jour.,* April 25, 1898.

produce symptoms that can be attributed to the abnormal accumulation of fluid in the abdomen alone; so long as the accumulation of fluid remains within certain bounds, the compression of the intestine is so slight that no disturbance of intestinal function results; this is due to the power possessed by the intestine of counteracting the effect of pressure from without, provided it is not extreme, by an increase of its own functional powers.

Very small accumulations of fluid inside the abdomen cannot be diagnosed, as the fluid is distributed along the dorsal aspects of the peritoneal cavity and in the pelvis, and consequently evades detection by physical examination. When it is very important to detect small quantities of ascitic fluid, the patient should be examined in the knee-elbow position; a small amount of fluid, if present, will sink down and accumulate around the umbilical region, so that a certain amount of dulness should be elicited in this portion of the abdomen.

[In such cases, where only small quantities of ascitic fluid are present, Garciadiego [1] recommends that rectal or vaginal examination should be made when the patient is in such a position that intra-abdominal fluid gravitates into Douglas' pouch, where it gives the finger the feeling of fluctuation.—Ed.]

Fr. Müller performed a number of comparative tests in dead bodies, and found that in adults 1000 c.c. of fluid injected into the abdominal cavity produced no dulness whatever; that 1500 c.c. produced a slight degree of dulness, only barely perceptible in the most dependent portions of the abdomen; and that 2000 c.c. of fluid gave a distinct and absolute dulness, which altered with changes in the position of the body. (In the bodies of children of three months the same relative results were obtained with 100, 150, 200 c.c. of fluid.) In the living subject, however, as Müller has shown, the conditions are not quite the same as in a corpse, for the fluid does not distribute itself between the different loops of intestine as in the corpse, but shows more of a tendency to accumulate in the most dependent parts of the abdominal cavity. It is possible, therefore, to recognize even smaller quantities—*i. e.*, less than two liters—in a living subject.

Von Criegern recently asserts that even small quantities of free fluid can be appreciated by introducing, while seated, the forefinger into the inguinal canal, the patient being in the upright position. By tapping the abdomen, a thrill will be detected by the forefinger. In women, according to Landau, the uterus appears, in commencing dropsy, to lie, as it were, on a water-pillow.

When there is only a small amount of fluid in the abdomen, it is often difficult to decide whether the dulness on percussion in the flanks is really due to the accumulation of this small amount of fluid in the most dependent portions of the abdomen, or whether this dulness is due to some other cause; careful examination of the patient and consideration of all the features of the case will, however, usually decide this point. The dulness may also be due to the presence in the flanks of

[1] Garciadiego, *Semaine Médicale*, 1894, p. 96.

loops of intestine that are contracted, and consequently contain no air, or are filled with solid or even with much liquid material. In the former case there will be no modification of the percussion-note when the patient changes his position ; in the second case there will, at best, be only a very slight change in the note, and frequently, as in the former eventuality, none at all ; finally, when the loops of intestine in the flanks are filled with liquid material, they are, as a rule, more freely movable, and consequently considerable variations in the percussion-note are more prone to occur when the patient moves from one side to the other. In order to arrive at a differential diagnosis under these circumstances it is well to look for signs of fluctuation, for in cases in which loops of intestine filled with liquid bowel-contents are lying in the flanks and are producing the dulness on percussion, a peculiar splashing sensation will be detected by one hand when the flanks are percussed with the other hand, whereas in case of ascites, genuine fluctuation, such as we have described above, will be felt.

In cases of encysted inflammatory exudates the differential diagnosis between this condition and ascites may be very difficult, and very striking errors may be made. The diagnosis of purulent exudates is, comparatively speaking, easy, for there are the pain, the fever, certain definite etiologic factors, etc., which all point to the diagnosis of purulent peritonitis and naturally suggest the possibility of an encysted form of this affection. In cases, on the other hand, that run their course without fever and almost without pain it is very difficult occasionally to decide whether we are really dealing with a case of encysted ascites, or, for instance, with an ovarian cyst.

The treatment of ascites is the same as that of the primary cause responsible for its development. When the primary cause cannot be removed, as, for instance, in cirrhosis of the liver, a number of different drugs may be given in order to produce diuresis ; among them are digitalis, diuretin, urea, calomel, etc.

[Apocynum has a vigorous diuretic action, but has the disadvantage of irritating the stomach, and should, therefore, be combined with a small quantity of cannabis indica. Copaiba resin, in 10- to 15-grain doses, has also been recommended, as have asparagus (Hare),[1] urea, and liver extract (Mouras) in cirrhosis.—ED.]

As a rule, however, paracentesis is eventually required in order to relieve the pressure and the feeling of fulness and of dyspnea that these patients complain of. Occasionally diuresis is reëstablished after the ascitic fluid is once removed by paracentesis abdominis. In other cases —and everything will depend on the character of the primary disease— paracentesis must be frequently repeated in order to make the patient's existence bearable ; it is astonishing to note what enormous quantities of fluid may occasionally be withdrawn in the course of time by this method.

[The dry method or restriction of fluids has been employed as an adjuvant to paracentesis in ascites of cirrhosis by W. H. Dickinson.[2]

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[1] H. A. Hare, *Therap. Gaz.*, October, 1899.
[2] W. H. Dickinson, *Allbutt's System of Medicine*, vol. v., p. 891.

The operative treatment of ascites in cirrhosis of the liver consists in multiplying the vascular adhesions around the liver in the peritoneal cavity by opening the abdomen and rubbing the peritoneum, so as to set up a slight degree of peritonitis. The great omentum is also sometimes stitched to the abdominal wall or fixed between the diaphragm and the convexity of the liver. This operation was conceived independently by Talma and by Drummond and Morison,[1] and is often spoken of as the Talma-Morison operation. In 1902 Greenough [2] collected 122 cases in which this operation had been performed. In 102 cases of cirrhosis thus treated, 60, or 59 per cent., were in no way benefited, and some died sooner than they otherwise would; 42, or 41 per cent., were improved, and of these, 9 were living and in improved health two years after the operation.—Ed.]

INFLAMMATION OF THE PERITONEUM (PERITONITIS) —ITS PATHOGENESIS AND ETIOLOGY.

GENERAL PATHOGENESIS.

The development of the etiology and pathology of peritonitis during the last twenty years is a remarkable piece of work, and has been brought about by the individual efforts of a number of investigators, particularly bacteriologists. The subject has also been advanced in a very considerable degree by the results of modern operative surgery. Formerly it was but a vague chapter—nothing more than a recapitulation of facts—which described in other words the clinical condition, while it is now one of the most lucid and brilliantly worked-out subjects.

Formerly attention was chiefly directed to finding out the organ or organs from which the inflammatory process started or to make out whether, in addition to such a " secondary " peritonitis, there might not also be a " primary " peritonitis; further, some subdivisions of the subject on clinical and anatomic grounds observed by individual observers were attempted, hence the older literature contains reference to puerperal, traumatic, perforative, tuberculous, and carcinomatous forms of peritonitis; meanwhile the primary cause of the inflammation of the peritoneum was apparently overlooked. This omission was not corrected by Wegner's experiments, although this author, while unfamiliar with modern ideas of bacteriology, dealt with important points in the true etiology of peritonitis. Soon after Wegner's researches, however, a number of papers by Leyden, Albert Fraenkel, and other writers appeared, which dealt with the causes of peritonitis from the modern point of view. The investigations of Nepveu and of Garré on the bacterial flora of the fluid found in hernial sacs were for a long time practically unnoticed. It remained for Grawitz to give a stimulus to active research in this direction by showing experimentally that the introduction of pyogenic bacteria into the peritoneal cavity did

[1] Drummond and Morison, *Brit. Med. Jour.*, 1896, vol. ii., p. 728.
[2] Greenough, *Amer. Jour. Med. Sci.*, December, 1902, vol. cxxiv., p. 979.

not constantly and necessarily set up peritonitis. Thus peritonitis did not occur under the two following conditions—(1) when the number of pyogenic micro-organisms was not so large as to overtax the absorptive powers of the peritoneum; (2) when the pyogenic micro-organisms were not suspended in irritating media; he showed that when the micro-organisms injected into the peritoneal cavity could be absorbed, or when they were suspended in an indifferent menstruum, they were as innocuous as non-pyogenic, harmless bacteria.

After these researches a large amount of work, partly clinical, partly of an experimental bacteriologic character, appeared, all bearing on the elucidation of the etiology of peritonitis, by the following authors: Pawlowski, Reichel, Waterhouse, Boennecken, Orth, Bumm, Kraft, Laruelle, Weichselbaum, Alexander Fraenkel, Eugen Fraenkel, Predöhl, Malvoz, Walthard, Burginsky, Barbacci, Muscatello, Ziegler, Tavel and Lanz, Arnd, Silberschmidt, Wieland, Klecki, Harbitz, Achard and Broca, Deaver, Veillon and Zuber, Krogius, Friedrich, Oppel, and others [Flexner, Durham.—ED.]. Papers continue to pour in regularly, and with increasingly accurate bacteriologic methods the results continue to multiply and also to become more reliable.

In addition, a number of investigations into the causes of perityphlitis belong to the same category and throw much light on the etiology of peritonitis. Many of these researches were of a purely experimental character, and with due care may be made use of in studying the causes of peritonitis in human subjects. There is still much controversy on this subject, and different authors advocate different views as to this or that point; this, of course, is not the place to reproduce all the various debatable points, nor is it possible to review them critically. In accordance with the general plan of this text-book my remarks will be confined to bringing out the most essential points contained in these contributions, and to laying particular stress on undoubted facts which seem specially likely to throw light upon the pathogenesis of peritonitis in man. The following remarks apply to the conditions found in human subjects; where this is not the case, special attention will be called to the fact. Investigations of a purely experimental character and all considerations solely based on experimental data will be mentioned only where it seems desirable to do so, and will be referred to as such.

Generally speaking, three forms of peritonitis can be distinguished— viz.: (*a*) Bacterial; (*b*) chemic; (*c*) mechanical.

Bacteria play the most important rôle in the etiology of peritonitis, especially of the acute form, and it may be said that in the great majority of cases acute peritonitis is of bacterial origin.

[The practical outcome of the bacterial origin of peritonitis is shown by the mental attitude, which is gradually abolishing the term "acute peritonitis" in favor of the more rational one of "acute peritoneal infection" (Mayo Robson[1]).—ED.]

Acute peritonitis exclusively due to chemic irritants is rare in human pathology.

[1] Mayo Robson, *Brit. Med. Jour.*, 1903, vol. ii., p. 242.

Both forms of irritants may produce chronic inflammation of the peritoneum.

It is not possible to prove that every case of peritonitis is due to a chemic or a bacterial irritant, nor can it always be shown with a reasonable degree of certainty that in every given case the peritonitis is due to either of these two etiologic factors. Sometimes, especially in chronic peritonitis, the action of some mechanical factor must be admitted. It is still necessary, therefore, to adhere to the old-established anatomico-clinical subdivision of peritonitis, and to distinguish a chemic, a bacterial, and a mechanical form, assuming in the latter case that the mechanical factor is the primary cause of the peritonitis.

BACTERIAL PERITONITIS.

After it had once been proved that in the great majority of cases of peritonitis, particularly in all the acute and diffuse forms, bacteria are the primary factors of the inflammation, it remained to be shown, in the first place, what species of bacteria give rise to peritonitis, and, in the second place, whether a single species of bacteria always produced the same kind of peritonitis or whether, in the various known forms of peritonitis, different species of bacteria are operative at the same time. A number of experiments on animals have been undertaken in order to solve this problem. In view of the fact, however, that the results obtained from these experiments cannot always be applied directly to human subjects, since different animal species react differently to different forms of bacterial infection, and some of the results upon animals are in direct contradiction to observations upon man, it is essential to decide these questions on the data obtained from direct clinical observation in man.

The following bacterial species, which are enumerated promiscuously, have so far been found in human peritonitis : Streptococcus pyogenes, Bacillus coli communis, pneumococcus (Diplococcus pneumoniæ), Staphylococcus pyogenes aureus, Bacterium lactis. In addition, the following species are described by Tavel and Lanz, who have investigated this subject more thoroughly than any other writers up to date : Diplococcus intestinalis major and minor, Coccus conglomeratus, bacillus of glanders, of tetanus, a bacillus resembling the Bacillus diphtheriæ, Bacillus pyocyaneus, and Bacillus pyogenes fœtidus, Proteus vulgaris, and several others. It is clear that in peritonitis due to or associated with perforation of the intestine almost any of the forms of bacteria met with in the bowel may be found in the peritoneal cavity ; it is, of course, true that all forms of bacteria do not develop with the same rapidity and facility in the peritoneal cavity. Finally, the tubercle bacillus, the gonococcus, and actinomycosis must be mentioned. Incidentally, the syphilitic form of peritonitis, which is very rare, may also be mentioned here.

A very important addition to our knowledge was made by Veillon and Zuber, who directed the attention of observers to the action of

anaërobic bacteria in appendicitis. This had been hitherto entirely neglected in research upon the etiology of peritonitis. They found several anaërobic forms of bacilli (Bacillus fragilis, ramosus, perfringens, fusiformis, furcosus) and a single coccus, the Staphylococcus parvulus. Krogius and Friedrich also made some experiments which led them to consider the anaërobic bacteria as important in every respect as the aërobic in the production of peritonitis. The former give rise to the foul odor of the feces and to the formation of gas; their toxins cause the symptoms of extremely acute intoxication. The apparent oversight of the anaërobic bacteria, despite every care in investigation, is explicable from the fact that with the method of culture most in vogue the aërobic bacteria overgrow and displace the anaërobic forms.

On autopsy either one or a number of species of bacteria may be found—simple or mixed infections. The statistical material at hand so far does not justify any definite conclusions as to the relative frequency of these two forms, but mixed infections seem to predominate; it may, therefore, be stated that in the majority of cases of bacterial peritonitis several different species of bacteria are found in the peritoneal fluid. Tavel and Lanz and Eugen Fraenkel [The reader should here refer to Flexner's observations, mentioned on p. 718.—ED.] chiefly found mixed infections in their cases; Predöhl's observations are to the same effect—out of 14 cases examined, 10 were cases of mixed infection. Alexander Fraenkel, on the other hand, found that simple infection predominated in his cases, being present in 20 out of 31 cases. To quote the most recent observer, Krogius found in 40 cases of perityphlitic abscess or general peritonitis originating in the appendix a pure culture in 3 cases only, and mixed infection in 35 cases; in 2 cases— one of effusion (hydrops) and one of appendiceal abscess—he could apparently discover no living bacteria in the contents of the diseased appendix. In peritonitis due to perforation mixed infection is the rule; this can readily be understood in the light of what has been said in a preceding paragraph as to the entrance of intestinal bacteria into the peritoneal cavity in this accident; in the traumatic and the puerperal forms of peritonitis it is much more common, on the other hand, to find only a single species of bacteria.

Of all the different bacteria which may be found in the peritoneal cavity in cases of acute peritonitis, the Streptococcus pyogenes, the Diplococcus pneumoniæ, and the Bacillus coli communis are by far the most important ones, while in chronic peritonitis the tubercle bacillus is the most common. As tuberculous peritonitis will be described in another section, the tubercle bacillus will not be further considered here.

There can be no doubt as to the very important rôle played by the Streptococcus pyogenes in the pathogenesis of acute exudative and especially of purulent peritonitis; all the authors who have investigated this subject agree on this point; I need only mention, for instance, Albert Fraenkel, Eugen Fraenkel, Alexander Fraenkel, Bumm, Predöhl, Tavel and Lanz, Achard and Broca, and others. It is frequently present in pure culture in the peritoneal exudate, and is met with in a

variety of peritonitides which are etiologically different; in puerperal peritonitis it is particularly frequent and may be considered the chief cause of this form of the disease; the same applies to the great majority of cases of traumatic peritonitis. In remarkable contradistinction to this, Krogius found the Streptococcus pyogenes, in 40 cases of appendicitis, once only; on the other hand, he found a diplococcus resembling the Diplococcus pneumoniæ pretty often. He, therefore, suspects that formerly this diplococcus was often mistaken for the streptococcus.

[A. O. J. Kelly's[1] results support Krogius's; in 94 cases of acute appendicitis the streptococcus was found only once. Low,[2] however, found streptococci in 81 per cent. of cases of appendicitis under three days' duration, and Lartigau,[3] in 73 per cent. of cases of four days' duration. It appears that streptococcal infection is often the primary condition, and that, as time goes on, a secondary infection with Bacillus coli supervenes.—ED.]

After the Streptococcus pyogenes, the Bacillus coli communis plays the most important rôle; pure cultures of this germ, as Alexander Fraenkel has shown by his careful investigations, may occasionally be found in the peritoneal exudate. Some authors, such as Laruelle and Malvoz, are inclined to attribute the great majority of all cases of peritonitis of intestinal origin to infection of the peritoneum with this germ, whether the peritonitis be due to perforation or to some other primary disease of the bowel (ulcer, strangulation, volvulus, etc.). This point of view seems to be, generally speaking, correct; we are not justified in saying, however, that the coli bacillus is the exclusive cause of inflammation in peritonitis of intestinal origin, for in many cases which are unquestionably of intestinal origin (for instance, after gastric ulcer, tuberculous ulceration of the bowel, appendicitis, etc.), other forms of bacteria, such as the pneumococcus, the Streptococcus pyogenes, may be found in the peritoneal exudate. Another very important point must also be remembered in estimating the rôle of the Bacillus coli, namely, that this micro-organism may have displaced all the other bacteria present in the peritoneal fluid postmortem. Charrin and Veillon, for instance, report a case in which they found a pure culture of the pneumococcus of Fraenkel in pus aspirated from the peritoneal cavity one hour after the death of the subject; in pus from the same case examined again twenty-six hours after death the Bacillus coli communis had replaced the pneumococcus in the peritoneal pus even though the body was kept cold from the time of death up to the autopsy, it being winter at the time.

Krogius obtained pure cultures of the Bacillus coli communis from the pus of perityphlitic abscesses and general peritonitis originating in the appendix in 35 out of 38 cases, and attributes to this organism a very important, though not quite the first, place in the etiology of peritonitis taking its origin from the bowel.

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[1] A. O. J. Kelly, *Phila. Med. Jour.*, 1899, vol. iv., p. 1032.
[2] Low, *Med. and Surg. Reps.*, Boston City Hospital, 1900, p. 173.
[3] Lartigau, *Studies from Department of Pathology*, College Physicians and Surgeons, Columbia University, 1900–02.

The Diplococcus pneumoniæ was several times found to be the only micro-organism, especially in the acute peritonitis of infants (Weichselbaum, Alexander Fraenkel, Boulay and Courtois-Suffit, Charrin and Veillon, Netter, Debove, Barbacci, Stooss and Tavel, and others). Krogius, as had Barbacci, often found the diplococcus in peritonitis originating in the appendix.

[From its much higher incidence in young girls, and from the inflammation being frequently restricted to the lower part of the abdomen, so that it often imitates appendicitis, it has been thought that the Diplococcus pneumoniæ may gain entrance through the Fallopian tubes (Brun[1]). This, however, is not strongly supported by data obtained from autopsies or from clinical observation, vulvovaginitis not being common (Stooss).[2] Michaut[3] believed that the inlet was through the throat and the blood-stream. In 33 cases collected by Quéhart,[4] 27 were in girls and 6 in boys; it is rare in adults, but is less infrequent in men than in women (Cassaet). The pus found in the peritoneum is diluted and forms two layers—an upper, slightly opaque fluid, and a thick, yellowish-gray, opaque deposit. According to Dieulafoy,[5] the onset of pneumococcic peritonitis is sudden, without any prodromal symptoms; there are sudden pain, vomiting, and diarrhea, with subsequent abdominal distention. At different periods in the course of the disease it resembles, and must be diagnosed from, appendicitis, typhoid fever, and tuberculous peritonitis.—ED.]

No other bacteria are of any real importance in the causation of peritonitis; the only ones of real practical importance are the Bacillus coli communis, the Diplococcus pneumoniæ, the Streptococcus pyogenes, and the tubercle bacillus. It is, of course, true that some of the other forms mentioned above are occasionally present in mixed infections of the peritoneum, but it is a very difficult matter to arrive at any definite conclusions as regards their individual pathogenicity for the peritoneum, owing to the fact that they are never present alone in the peritoneal exudate, but only in combination with one of the three species of bacteria whose great pathogenicity is established beyond doubt. Some of the bacteria, however, deserve brief consideration, because they have in rare instances been found alone in the peritoneal fluid and because they may, therefore, be considered the chief etiologic factors in the production of inflammation of the peritoneum in these cases.

It is remarkable that the Staphylococcus pyogenes aureus, which is of the greatest importance in the production of experimental peritonitis, is so rare in the clinical forms observed in man; all observers report this micro-organism in only a few exceptional cases. In a few instances, however, this bacterium has been found in pure culture in peritoneal exudate, so that its actual pathogenicity for human subjects has been proved.

[Flexner[6] divides peritonitis into—(a) Primary peritonitis, where

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[1] Brun, *La Presse Médicale,* February 27, 1897.
[2] Stooss, *Jahresb. f. Kinderheilk.,* October 20, 1902.   [3] Michaut, *Thèse de Paris,* 1901.
[4] Quéhart, quoted by J. H. Bryant, *Brit. Med. Jour.,* 1901, vol. ii., p. 767.
[5] Dieulafoy, *Clinique Médicale de l'Hotel Dieu,* 1898, p. 396.
[6] Flexner, *Phila. Med. Jour.,* November 12, 1898.

infection is brought to the peritoneum by the blood- or lymph-stream and is not due to any lesion of its contained viscera or to any surgical operation; in 106 cases of peritonitis examined bacteriologically, 12 belonged to this group. (*b*) Secondary peritonitis, of which there are two forms—(1) Exogenous peritonitis, in which the micro-organisms have in great part come from without, from the wounds of abdominal operations; in this group there were 34 cases, the micro-organism most frequently found being the Staphylococcus aureus, which was present in 15 cases, in 12 of which it was in pure culture and in 3 combined; the Staphylococcus albus was present in 3 cases, in one of which it was combined. (2) Endogenous peritonitis, in which the micro-organisms come in part or wholly from the intestinal tract; this class comprised 60 cases—the infections are more often multiple, and streptococci are the prevailing micro-organisms; thus streptococci were found in 39 cases—in pure culture in 7 cases and combined in 32—staphylococci were infrequent, there being 4 cases with Staphylococcus albus and 3 with Staphylococcus aureus.—ED.]

A number of recorded clinical cases make it probable that the gonococcus may also occasionally set up peritonitis (Hunter, Ricord, Tarnowski, Horowitz, Dittel, Zeissl, Cushing, and others). Wertheim's work, in fact, positively establishes the gonococcus in the rôle of an occasional cause of peritonitis. Positive proof has so far not been adduced to show whether in these cases there is a single or a mixed infection; but the former view is the more probable.

[Hunner[1] has collected 31 cases of gonococcal peritonitis and regards it as differing from other forms of peritonitis, as the patients are extremely ill for some time and then suddenly take a turn for the better. When diagnosed, as it always should be, though it frequently imitates appendicitis, it should not be operated upon. Of his 31 cases, 17 were operated upon, 13 recovering and 4 dying; of 14 not operated upon, 8 recovered and 6 died: the latter were practically moribund on admission, and it was doubtful whether any treatment would have been of any use. Cushing,[2] who had previously recorded 2 cases of gonococcal peritonitis which were due to a single infection, pointed out the special features of this form of peritonitis—viz., its dry, fibrinous character, with practically no pus or serous exudate. Northrup[3] has described 2 cases of general gonococcal peritonitis in young girls under puberty, and Comby[4] has collected 8 other cases under thirteen years of age. Comby speaks of the peritoneum being grazed with a gonococcal infection which sets up peritonism rather than peritonitis, there being an explosive onset of symptoms which subside in twenty-four to forty-eight hours. The peritoneum offers considerable resistance to gonococcal infection, which is more prone to supervene when its resistance has been diminished by menstruation. In this connection it may

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[1] Hunner, *Johns Hopkins Hosp. Bull.*, October, 1902, vol. xiii., p. 247.
[2] Cushing, *ibid.*, May, 1899, vol. x.
[3] W. P. Northrup, *Arch. Pediatrics*, December, 1903, p. 910.
[4] Comby, *Arch. de Méd. des Enfants*, vol. iv., p. 513.

be interesting to notice that on the basis of 165 cases in which he determined the presence of pus in the pelvis, before operation, by blood counts, Dützmann[1] comes to the conclusion that a leukocytosis of from 11,000 to 13,000 pointed to gonococcal infection, while a leukocytosis of 30,000 indicated streptococcal infection.—ED.]

The significance of actinomycosis will be dealt with in the section on Perityphlitis.

Weichselbaum has reported a case of peritonitis following rupture of the spleen in typhoid fever, in which there was general peritonitis, with enormous numbers of typhoid bacilli in pure culture in the exudate. This and other recorded cases of a similar character must, of course, be regarded as mere curiosities. Once again, in the face of recent observations, particularly those of Veillon and Zuber, Krogius, and Friedrich, we must refer to the importance of the anaërobic forms, for, taking these into consideration, several points require renewed investigation before the bacteriologic etiology of peritonitis is definitely established.

How do the bacteria gain an entrance into the peritoneal cavity? In males the peritoneal sac is closed from the outside world on all sides, while in females it communicates with the exterior through the ostium abdominale of the Fallopian tubes.

Although this passage into the abdominal cavity through the ostium, Fallopian tubes, uterus, and vagina is lengthy, microbes do occasionally traverse it, reach the peritoneum, and set up inflammation. According to the facts brought to our knowledge by Wertheim and others, such as Zweifel, there is now no reason to doubt the possibility of gonococcal infection of the peritoneum as a result of gonorrheal salpingitis, and the clinical experience of a number of observers is in favor of this possibility. Menge and Bumm's observations would seem to militate against this view, but this negative evidence does not seem to me to be valid as against the mass of recorded positive evidence. Since the gonococcus cannot gain an entrance to the peritoneum by any other path in cases of this kind,—viz., of gonorrheal salpingitis,—it must be positively assumed that the infection of the peritoneum occurred in this way from without. It is probable that other bacterial species, such as the tubercle bacillus, may infect the peritoneum in the same way, but as other avenues of infection are open in the case of all these other possible germs, the proof is not so convincing as in the case of the gonococcus, that the germ actually reached the peritoneum *via* the Fallopian tubes.

In spite of what has been said, this mode of entrance must, at best, be exceptional, and is out of the question in male subjects. The general rule may, therefore, be formulated as regards the entrance of bacteria into the peritoneal cavity that infection of the peritoneum can occur only : (1) When there is a free pathologic communication between the peritoneal cavity and the outer surfaces of the body or between the peritoneum and some organ (or abscess) containing bacteria ; (2) when

[1] Dützmann, *Centralbl. f. Gynäk.*, 1903, No. 47.

bacteria succeed in penetrating the wall of some organ or structure which lies in contact with the peritoneum ; for we know that bacteria cannot penetrate through the normal skin (at least no proof of this contingency has ever been given) ; (3) when bacteria are carried to the peritoneum through the blood-current.

1. It is perfectly clear how micro-organisms get in when the peritoneal cavity is opened by injury. The bacteria, especially the streptococci, enter with the air or are carried into the peritoneal cavity with the instruments or other materials that are introduced into the abdominal cavity. [The reader should refer back to Flexner's observations on p. 718.—ED.]

A similar communication is opened up when one of the abdominal organs ruptures and its contents pass into the peritoneal sac. As perforation of the intestine is the most frequent accident of this kind, the entrance of the copious bacterial flora contained in the bowel into the peritoneal cavity is very common. The result of this accident is, in the first place, always a mixed infection ; but of the numerous species which thus enter the peritoneal cavity, only one, as a rule,—the Bacterium coli commune,—represents the true inflammatory agent (" phlogogene agens "). In rare instances other bacteria may assume this rôle, such as streptococci, staphylococci, Diplococcus pneumoniæ. No doubt exists about the method of microbic invasion in perforation of the stomach, of a liver abscess, of the spleen in typhoid fever, or of an extraperitoneal abscess or even of an intraperitoneal abscess, which, having become encapsulated and separated off from the general peritoneal cavity by firm adhesions and connective tissue, is in reality equivalent to an extraperitoneal abscess.

The entrance of bacteria into the peritoneal cavity in a number of other cases, which may also be ranked among the traumatic perforations, apparently also occurs by direct communication, although the path by which the bacteria work their way in is not so clear and apparent in this category. It may frequently happen that an attack of peritonitis develops after some injury from a blunt instrument without any lesion of the skin (a blow, a fall, or some similar accident). As the injury *per se* does not produce the anatomic changes in the peritoneum, and as, according to our present views, it must be assumed that bacteria gained an entrance in some way or other into the peritoneal cavity, we can only accept the view of König, Gerhardt, and others that in these cases there is some slight injury of the intestine which allows bacteria to pass out through the bowel-wall. After death it may be impossible to find any lesion of the intestine, because it may have healed long ago ; nevertheless, it may originally have been large enough to allow bacteria to pass into the peritoneal cavity ; possibly, too, slight hemorrhage occurring at the same time might favor the growth and development of the bacteria. In some cases we must assume, further, that the small tear involved only the mucosa and muscularis, and did not extend further than the subserosa. This is illustrated by a remarkable case recorded by Henoch, and frequently quoted in the literature, in which an appar-

ently idiopathic peritonitis developed in a child that had been ill treated.

2. When no pathologic communication, either small or large, exists, bacteria can, nevertheless, work their way into the peritoneal cavity directly through the tissues, provided cavities of the body situated in immediate proximity to the peritoneum contain them ("infection by continuity," Tavel and Lanz). In cases of this character the germs grow into the peritoneum from neighboring organs. Recorded cases show that infection of the peritoneum may occur from neighboring lymph-passages; in these cases the micro-organisms multiply and progress along the lymph-channels until they reach the peritoneum. This is well seen in the lymphangitic form of puerperal peritonitis in which streptococci travel from the numerous lymph-spaces of the mucosa of the vagina and the uterus into the lymph-sinuses of the uterine musculature, and from there into the subperitoneal lymph-channels, the broad ligaments, and the peritoneum covering the uterus. The same sequence of events is seen in peritonitis following infection of the pleura, for here the germs apparently travel into the peritoneum through the lymph-channels of the diaphragm. Lastly, this mode of infection has been directly demonstrated by Iversen (quoted by Lennander) in some cases of appendicitis (scolecoiditis). [Compare also Doyen's [1] remarks on lymphangitis in appendicitis.—Ed.]

The greatest interest, of course, attaches to the question how bacteria can pass through the intestinal wall and reach the peritoneum in the absence of any gross perforation of the intestinal wall. There is no doubt whatever that this is impossible in health, for we know that the common intestinal bacteria never penetrate through the healthy wall of the intestine. It has, it is true, been assumed that the bowel is pervious to micro-organisms in cases of slight obstruction merely from constipation, when macroscopically the mucous membrane is normal and microscopically the epithelium is practically retained (Posner and Lewin); this, however, has been disputed (Marcus, Austerlitz and Landsteiner, Opitz). [Adami,[2] Ford,[3] and Lartigau [4] have supported the view that under apparently normal conditions micro-organisms can penetrate into the deeper layers of the intestinal wall and be carried into the bloodstream, producing a condition of latent infection or subinfection.—Ed.] Some pathogenic bacteria, it is true, seem to possess the power of passing through the bowel-wall when they accumulate in the intestinal lumen, especially the tubercle bacillus, which, however, does not at once reach the serosa, but passes first into the lymphatic follicular apparatus of the bowel-wall, and from there into the lymph-glands. The localized peritonitis often seen over tuberculous ulcers of the intestine never appears until the underlying intestine is already diseased and abnormal and consequently permits the passage of bacteria.

On the other hand, it may be considered settled that the common

[1] Doyen, *La Méd. Mod.*, May 26, 1897.
[2] J. G. Adami, *Jour. Amer. Med. Assoc.*, December 23, 1899.
[3] W. W. Ford, *Trans. Assoc. Amer. Phys.*, 1900, vol. xv., p. 389.   [4] Lartigau, *loc. cit.*

intestinal bacteria can also travel through the intestinal wall, and even produce inflammation of the peritoneum under favorable conditions when the structure or the nutrition of the intestinal wall becomes altered. This point has been proved in strangulated hernia by the experimental and clinical investigations of numerous authors. Some writers, it is true, oppose this view,—for instance, Garré, Rovsing, Orth, Waterhouse, and, in some particulars, also Ziegler,—while other authors—Nepveu, Clado, Körte, Bönnecken, Arnd—who have investigated this point have obtained positive results. Bönnecken, in particular, found living bacteria which were capable of development in enormous numbers in the clear watery fluid of hernial sacs, as well as in the sanguineous, offensive exudates found in gangrenous loops of intestine. The microbes found by Bönnecken were identical with those in the lumen of the intestine, and he succeeded in discovering these bacteria not only in artificial, experimental hernias in animals, but also in the hernial fluid in human subjects. The bacteria were found in the hernial fluid of strangulated hernias in man within four hours after the incarceration, and at a time when the only discoverable lesions of the intestinal wall were a moderate degree of simple venous hyperemia and a somewhat advanced stage of serous infiltration of the tissues. Arnd, too, has demonstrated positively that the intestinal wall, even though it is only so slightly affected that it can subsequently regain its normal function and structure, may occasionally allow bacteria to pass through, for he found bacteria on the outer surface of such loops of intestine in several cases.

The investigations of Tavel and Lanz are extremely interesting and important; from examination of a large number of cases of strangulated hernia in human subjects they found that in the great majority of their cases bacteria were absent from the fluid contained in the hernial sac, while at the same time they failed to find any evidence of peritonitis. In only a small minority of the patients were bacteria found in the hernial fluid, while the clinical picture presented seemed nevertheless to be the same as in cases where no bacteria were present in the peritoneum. This observation is the more remarkable as many of the cases were complicated and severely strangulated hernias, in which the exudate in the hernial sac was markedly blood-tinged, and in one of the cases the irritability of the intestine was so completely lost that it had to be resected.

For the present it is impossible to reconcile these contradictory statements; it is possible that the intestinal wall is more permeable in one individual than in another, or that in some cases the contents of the intestinal lumen in general and of the lumen of the affected loop in particular vary. At all events, we know that a number of positive observations have been recorded in human patients, and, on general principles, it must be admitted that under certain pathologic conditions the wall of the bowel becomes permeable for bacteria.

The observations as to the passage of bacteria through the intestinal wall in cases of strangulated hernia also throw some light upon the

development of peritonitis in some other diseases of the intestine, such as internal strangulation of the bowel, volvulus, and intussusception. In all these diseases changes, such as hyperemia and serous infiltration, occur in the intestinal wall, which render the affected parts permeable for the bacteria contained in the intestinal lumen. It appears, too, that the bowel is certainly pervious in enteritis. I have published a case of diffuse suppurative peritonitis which arose thus : Suppurative streptococcal tonsillitis, followed first by acute gastric catarrh (the same streptococcus being found in the tissues of the stomach-wall), and secondly by peritonitis. Lennander holds the same opinion, and refers to a case of peritonitis following on enteritis by continuity.

[Tavel[1] believes that while the normal bowel does not allow microorganisms to pass through its walls, circulatory and nutritional changes make this possible ; the healthy peritoneum, however, may absorb the micro-organisms and so prevent peritonitis ; if the resistance of the peritoneum is reduced by the action of toxins, peritonitis will result. —Ed.]

Lastly, in this category we must include those cases of peritonitis of intestinal origin which develop as the result of some other affections of the intestine, such as ulceration of the bowel-wall, dysentery, typhoid, tuberculosis, syphilis, and influenza.

3. Finally, bacteria may reach the peritoneum through the blood. This hematogenous mode of infection is very rare and is far less frequent than any of the other forms of infection enumerated above. (See also the section on the Clinical Etiology of Peritonitis.) It is well known that in the great majority of infective diseases hematogenous peritonitis is a very rare event ; we may even go so far as to say that in many of these diseases it probably never occurs. This also applies to the influenza bacillus, an organism which, as we know, does damage to the greatest number of organs ; in influenza, for instance, pleurisy (also the hematogenous form, which appears independently of any affection of the lungs) often supervenes, whereas, according to Leichtenstern, there is no recorded case of primary hematogenous infection of the peritoneum. The published cases of influenzal peritonitis must be attributed to the severe grippal enteritis occasionally seen in this disease ; they, therefore, so far as their mode of origin is concerned, rank with the class of cases described under " 2." In the same way Lennander believes that cases of peritonitis, now called hematogenous, arise by a direct (contiguous) infection from the bowel, the microbes growing into the peritoneal cavity along the lymph-channels of the intestinal wall.

Some authors insist on the occurrence of hematogenous peritonitis in the course of acute rheumatism ; and some very competent observers, such as Eichhorst, have reported cases of this kind. At all events, the development of peritonitis in the course of polyarticular rheumatism must be a very rare event ; personally, I have only once seen such a case. Analysis of the brief reports published on this subject also shows that in many of the cases peritonitis did not appear alone, but that other

[1] Tavel, *Correspondenzbl. f. Schweiz. Aerzte*, October 15, 1901.

lesions complicated the clinical picture ; thus G. Singer, for instance, found peritonitis only twice in 64 cases of acute rheumatism, and both of his cases were, moreover, complicated with endocarditis and pericarditis and with double pleuropneumonia. How is it possible, in cases of this kind, to prove that the infection of the peritoneum occurred directly through the blood-stream ?

It seems probable that occasionally peritonitis may occur by way of the blood in cases of pneumococcus infection, and that in rare instances tuberculous peritonitis must be regarded as hematogenous in origin. The question whether or not primary independent streptococcal infection of the peritoneum ever occurs will be considered under the heading of So-called Primary Idiopathic Peritonitis.

The most common and satisfactorily established form of hematogenous peritonitis is found in septic processes. Here the bacteria migrate from some septic focus into the blood-stream, and are carried to the peritoneum by this channel ; it makes no difference whether the primary focus is situated in some infected organ or whether the bacteria are derived from an infected thrombus situated in a blood-vessel. This form of peritonitis is called " peritonitis of septicemia," and should not be confounded with so-called " septic peritonitis."

CHEMIC PERITONITIS.

The purely chemic (or toxic) form of peritonitis—*i. e.*, inflammation of the peritoneum without the intervention of bacteria—is rare as compared with the bacterial form just described, at any rate as regards acute peritonitis. In considering the mechanical form of peritonitis in one of the following paragraphs, I shall have occasion to return to this subject and to show to what extent this applies to the chronic form of peritonitis.

It may be considered settled at the present day that chemic substances can produce the tissue changes called inflammation, and even real suppuration in the clinical sense ; there has been much controversy on this subject, but, as I have said, the matter may now be considered definitely settled. A considerable number of chemic substances possess this property, but may all be left completely out of account. In human pathology they rarely or never, with the possible exception of corrosive sublimate, come in contact with the peritoneum. The excretions and secretions of the human organism itself, which in pathologic conditions come in contact with the peritoneum, are quite incapable of producing inflammation of the peritoneum, and are perfectly harmless in this respect, unless they contain bacteria.

Naunyn, Gilbert and Girode found that the bile was always sterile so long as the condition of the bile-passages was normal ; Létienne, Tavel and Lanz found the bile sterile in a certain proportion of the cases they studied, but also found that in a number of instances the bile contained a variety of microbes, most commonly the Bacterium coli commune and the staphylococcus. In the experimental introduction of bile into the peritoneal cavity of animals a number of observers failed

to find evidence of inflammatory changes in the peritoneum. Similar observations are recorded in human subjects by McSwiney, Tavel and Lanz, Lähr, Thiersch; all these writers found that the peritoneum remained free from inflammation even after rupture of healthy bile-passages.

[Dévé [1] collected 15 cases of choleperitoneum due to rupture of a hydatid cyst, in communication with a bile-duct, into the peritoneum. —Ed.]

Of course, when, from rupture of an inflamed or suppurating gall-bladder, the bile passes into the peritoneal cavity, peritonitis necessarily develops; this is not surprising, for it is easy to understand how the inflammation of the peritoneum is brought about by the bacteria contained in the diseased and ruptured bile-passages. Alexander Fraenkel reports hemorrhagic forms of peritonitis with severe general symptoms produced in animals by the injection of bile containing no bacteria directly into the peritoneal cavity.

Normal urine, when it enters the peritoneal cavity from a healthy urinary bladder, is also incapable of setting up inflammation unless it contains bacteria. As a rule, of course, in those conditions where urine usually passes into the peritoneal cavity, it contains microbes, and the resulting inflammation of the peritoneum cannot be considered as chemic in character.

The pure secretions of the stomach and the intestine and the gastric and intestinal ferments do not properly belong to this class of agents, as they never enter the peritoneal cavity in human subjects except when mixed with other constituents of the gastro-intestinal tract that are capable of producing peritonitis (bacteria, etc.). It is worth mentioning, however, that, experimentally, Pawlowsky found that trypsin had a marked inflammatory action, and can induce serous and hemorrhagic, but not purulent, inflammation. Other authors who have repeated some of his work have failed to corroborate it.

The gases of the intestine *per se* are, according to the researches of Nowak, incapable of producing inflammation of the peritoneum.

The atmospheric air is also incapable of producing inflammation of the peritoneum, provided it is aseptic and does not produce mechanical lesions of the peritoneum by its presence in the abdomen.

It appears, therefore, that all the substances enumerated are chemically without direct significance in producing inflammation of the peritoneum, and can act only indirectly and under definite conditions, which will be referred to later. In the case of the bacterial toxins—*i. e.*, the products of bacterial life that act chemically—the conditions, however, are entirely different. These poisons directly produce inflammation of the peritoneum and can act alone and without the intervention of any other factors; these poisons are the ones, then, that in human subjects produce the chemic form of peritonitis.

The fact that bacterial poisons can set up inflammation has been fully established by the investigations of Brieger, Leber, and others,

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[1] Dévé, *Rev. de Chir.*, July 10, 1902, p. 67.

but it is impossible to give an exhaustive account of this subject here. While it may be considered positively settled that these various bacterial poisons can produce peritonitis in animals, it still remains to be proved whether they can actually be considered clinically to be the cause of some forms of peritonitis in man.

That this is actually the case has been shown more especially by the valuable observations published by Tavel and Lanz. In a number of cases of peritonitis due to different causes, such as internal strangulation of the bowel, volvulus, perityphlitic abscess, and cholecystitis, these writers prove beyond any doubt that the peritoneal exudate contained no bacteria. In view of the fact that in none of these cases was there any perforation of the bowel, they felt justified in concluding that the inflammatory process in these instances was actually due to the action of the bacterial poisons manufactured in the affected organs (intestine, gall-bladder, and abscess cavity), which subsequently found their way into the peritoneal cavity. In these cases, therefore, the peritonitis was genuinely chemic in origin.

[Durham's [1] observations, that after the experimental injection of bacteria into the peritoneum the peritoneal fluid may be sterile twenty-four to forty-eight hours later, while the omentum contains many micro-organisms, have already been referred to. He has noticed the same in cases of peritonitis in men; thus in a fatal case of strangulated hernia no cultures could be obtained from the peritoneal fluid, but the omentum and the anterior mediastinal glands gave an abundant pure growth of Staphylococcus aureus. These facts make one hesitate before accepting as chemic a case of peritonitis in which these structures have not been examined.—ED.]

It is a remarkable fact that in such cases suppuration is absent. The exudate is usually either simply fibrinous or consists of a mixture of fibrinous and hemorrhagic material.

To the class of chemic peritonitis some other forms must manifestly be added, such as peritonitis following torsion of an ovarian stump after an ovariotomy (Schroeder), peritonitis after inflammation occurring in the interior of a cyst or after diphtheric cystitis (Alex. Fraenkel)—in all these cases no bacteria can be found in the exudate.

There is another group of chemic peritonitis that occurs in the course of some other diseases, such as nephritis. It is a well-known fact that peritonitis is by no means rare in this latter affection. Thus, to quote statistics, Becquerel observed peritonitis 12 times among 129 cases of nephritis; Rosenstein, 10 times among 114 cases. Frerichs, among 292 fatal cases of Bright's disease, saw peritonitis 33 times. It is true that in these cases the primary chemic cause of the peritonitis is unknown, but it is generally regarded as due to waste-products which are not eliminated properly in nephritis and consequently accumulate in the blood.

[The frequency with which bacterial infection causes peritonitis in chronic renal disease is well shown in Flexner's [2] statistical study of

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[1] H. E. Durham, *Jour. Path. and Bacteriol.*, vol. iv., p. 357.
[2] Flexner, *Jour. Exper. Med.*, 1896, vol. i., No. 3.

terminal infections; even in cases where no micro-organisms are found in the peritoneal exudate, it is possible that they were present at an earlier stage, and it cannot be regarded as absolutely certain that the case is one of chemic peritonitis.—ED.]

MECHANICAL PERITONITIS.

It must be admitted that mechanical irritation may occasionally produce peritonitis. Fifty years ago Virchow assigned much more significance to this factor than can be justifiably attributed to it now. Virchow's views were based on the current knowledge of the time, and naturally the recent great development of our knowledge of the etiologic factors of peritonitis has led more modern writers to modify the views enunciated by the older ones. Many of the cases of peritonitis regarded by Virchow as mechanical can now be shown to be chemic or bacterial in origin. Nevertheless, a certain group of cases remains which must be regarded as solely due to mechanical irritation of the peritoneum. The chief mechanical factors responsible for peritonitis are pressure and increased friction.

Some authors are inclined to object to the data obtained from clinical evidence and from anatomic investigation, but their objections cannot apply to the results obtained from animal experiments, for the results thus obtained are definite and in no way ambiguous. The first experiments of this kind were carried out by Wegner, who injected air into the peritoneal cavity of animals. Even when this inflation with air was frequently repeated and continued for months, and the peritoneal cavity inflated to such a degree that the intra-abdominal tension was greatly raised, no functional disturbances whatever were noticed. When these animals were killed after several months, marked changes were found on the surface of the liver and the spleen, and to some extent on the anterior surface of the stomach. There were cicatricial thickening of the peritoneum covering these organs, and alterations in the shape, especially of the sharp margins of these organs. The serous lining of the diaphragm, and more particularly of the mesenteries, were considerably thickened and in parts presented a cicatricial appearance. Histologically there were fatty degeneration and desquamation of the endothelium and chronic thickening and induration of the peritoneum. The fatty degeneration of the endothelium was probably a result of the interference with the nutrition due to excessive pressure exerted on the surfaces of the peritoneum when the peritoneal cavity was inflated with air. After the desquamation of the endothelial layer has occurred, the underlying connective tissue soon undergoes inflammation, owing to the irritation to which it is exposed; thickening and induration are thus rapidly brought about. Wegner attempted to show that the excessive pressure exerted by the air in the peritoneal cavity, and not the reduction of the temperature or the presence of an abnormal mixture of gases, or, lastly, the entrance into the peritoneum of "dust-like" particles, was responsible for these inflammatory and irritative changes.

From the modern bacteriologic point of view some objections can, of course, be brought against these experiments, but they are not valid against similar experiments carried out with all possible precautions by Wieland. His results are specially interesting, as they prove beyond a doubt that peritonitis can be produced by purely mechanical means. Wieland introduced absolutely sterile fish-bladders, filled with ordinary sterile bouillon, into the peritoneal cavity of rabbits with all the necessary antiseptic precautions, and after a certain length of time found that aseptic inflammation of the peritoneum had occurred in the immediate neighborhood of the bladders, consisting in the formation of abundant highly vascularized adhesions—in other words, chronic adhesive peritonitis. There was no sign of suppuration either macroscopically or microscopically. Here the irritation of the peritoneum was due to a mechanical factor—viz., the pressure exerted by the introduction of the distended fish-bladders.

[From experiments on rabbits, Vogel[1] concludes that local peritonitis and adhesions may be due to effused blood, mechanical injuries, slight burns, chemic irritation, and foreign bodies. In some instances after rupture of a hydatid cyst into the peritoneal cavity a remarkable condition—peritoneal pseudotuberculosis of hydatid origin—results (Dévé[2]), which has been regarded as a defensive process on the part of the organism. The peritoneum has a number of small granulations the size of a pin's head or of a millet-seed on its surface, which histologically resemble tuberculosis, but contain débris of cyst-wall or hooklets undergoing absorption instead of tubercle bacilli.—Ed.]

Factors such as pressure and friction unquestionably play a part in a considerable number of the cases of peritonitis seen in practice, and some forms of chronic adhesive peritonitis and of chronic indurative peritonitis must be attributed to pressure and friction alone, such as the chronic thickening of the peritoneum in the furrow of a "corset liver," the peritoneal thickenings around the hepatic, splenic, and sigmoid flexures of the colon in cases of fecal accumulation in these parts of the bowel, in the vicinity of old hernial openings, and, lastly, those thickenings seen in cases of large intra-abdominal tumors; in the latter the conditions are usually quite analogous to those produced experimentally by Wieland.

[B. Robinson[3] has suggested that muscular action, such as vigorous contraction of the psoas muscles, may determine local peritonitis and adhesions, but he does not argue for the process being exclusively mechanical. His view is that if a piece of intestine lies within the range of a powerful muscle and is temporarily damaged, micro-organisms may pass through the walls of the bowel and set up local peritonitis.

The marked thickening of the peritoneum over hydatid cysts in the peritoneal cavity, especially when they are in positions such as the recto-vesical pouch, where more or less constant movement will give rise to

[1] Vogel, *Deutsch. Zeitschr. f. Chir.*, vol. lxiii.
[2] Dévé, *Rev. de Chir.*, July, 1902, p. 79.
[3] B. Robinson, *Medical Record*, August 29, 1903, p. 324.

friction, illustrates this point.   Shattock [1] has described a large corneal fibroma in the subperitoneal tissue of the rectum containing a small piece of steel, and draws attention to similar thickenings on the peritoneal surfaces of large ovarian cysts.—ED.]

It need, of course, hardly be mentioned that many other forms of partial adhesive and indurative peritonitis are due to bacterial and chemic irritants.   It is evident, therefore, that chemic, bacterial, and mechanical factors are all capable of causing peritonitis; experimental investigations and postmortem observations further show that the nature of the irritant determines the anatomic form of the inflammation, and that, in man at least, definite relations exist between the character of the primary irritant and the nature of the resulting peritoneal inflammation.

Purulent peritonitis is always of bacterial origin; in other words, without bacteria there can be no purulent inflammation of the peritoneum.   The converse is not, however, true, for bacterial peritonitis is not necessarily purulent, or possibly, more correctly, need not have reached the stage of pus-formation; purely chemic forms of peritonitis are serous, serous-hemorrhagic, or fibrinous; purely mechanical forms of peritonitis are only adhesive.

[In acute and rapid cases of peritonitis the signs of inflammation in the peritoneum may be slight, but the micro-organisms in the omentum and anterior mediastinal glands may be important aids in recognizing the condition (Durham [2]); some of these very acute cases are better described as acute peritoneal infection than as peritonitis.—ED.]

After this review of the general factors capable of producing the different forms of peritonitis (serous, purulent, hemorrhagic, fibrinous, adhesive), it is necessary to consider the special conditions which must actually be present before inflammation of the peritoneum can develop.   The numerous investigations of recent years into the affections of the peritoneum have shown that inflammation does not always necessarily occur when any one of the various irritants enumerated above exerts its action on the peritoneum; it has been shown, moreover, that definite conditions must exist in order that such an inflammatory reaction may occur.   In many of the clinical accidents which are followed by peritonitis several of the different factors capable of producing peritonitis are active at the same time, so that it is often difficult to decide which one of them is principally or exclusively responsible; for instance, in perforation of the bowel bacterial, chemic, and mechanical irritants all affect the peritoneum at the same time.

A large number of investigators have occupied themselves with this question, especially Tavel and his pupils; although here and there contradictory statements and differences of opinion crop up, the general consensus of opinion is more or less uniform, and there is considerable agreement among the various writers on this subject.   As it would be

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[1] S. G. Shattock, *Trans. Path. Soc.*, vol. xliv., p. 151.
[2] H. E. Durham, *Jour. Path. and Bacteriol.*, vol. iv., p. 338.

outside the scope of this clinical hand-book to enter into a detailed discussion of this subject, however fascinating it might be, I must confine my remarks to a brief summary of the main results obtained on this point. It must be specially insisted on, however, that there are some discrepancies between clinical observations and the results of experimental research which it remains for future investigation to clear up.

To understand properly the pathology of peritonitis following upon bacterial infection, it must be looked at from several different points of view. To a certain extent the peritoneum itself renders the bacteria harmless : again, phagocytosis takes place either by means of leukocytes or perhaps by means of the endothelial cells ; further, the peritoneal fluid has had a bactericidal power attributed to it, which it is able to exert when the bacteria are only of moderate virulence. Further, it is most important to bear in mind that the peritoneum has great powers of absorption, which are responsible for the disappearance of various substances which may happen to enter the peritoneal cavity, whether they are innocuous or toxic ; from the peritoneal cavity these foreign bodies enter the general circulation and may exert their deleterious effects on the organism. Fifteen minutes after injecting streptococci into the peritoneal cavity of a rabbit they are found in the heart, in the marrow of the bones, in the liver, and in the kidneys. Hence invasion of the peritoneal cavity by foreign bodies is followed by two results— viz., (1) Noxious substances,—for instance, bacteria,—even when they enter the peritoneal cavity in large numbers, rapidly disappear again before they can exert any bad effects ; (2) certain poisonous substances, which enter the peritoneal cavity or are manufactured there (bacterial toxins), are absorbed into the circulation with such rapidity and in such large quantities that serious constitutional effects are produced.

The conclusions to be deduced from experimental research in connection with this subject are that no one of the various agents (bacteria, bacterial products, intestinal ferments, solid particles) which enter the peritoneal cavity after perforation of the bowel are singly able to set up diffuse purulent peritonitis. It is only when several of these agents act together—*i. e.*, when one factor reinforces and supports the other in its harmful effects—that purulent inflammation develops. The mechanism of this complicated process is as follows : The chemic irritants (bacterial products and possibly also bile and intestinal ferments, as well as various chemic substances employed in some of the experiments) in all probability produce conditions favorable to bacterial activity—in other words, they prepare a suitable nidus for the growth of micro-organisms and for their toxic effects ; this depends on damage of the serosa, which impairs its functional powers ; absorption from the peritoneal cavity is thus diminished, and the bacterial products not being removed, consequently accumulate and exert their baneful effects.

According to Walthard's experiments, however, functional paresis of the serosa is not enough to cause or to favor the production of purulent peritonitis by bacteria ; the additional factor necessary is preliminary peritonitis due to chemic irritants. The latter form of inflammation is

characterized by hyperemia, swelling, and proliferation of endothelial and connective-tissue cells, leukocytosis, and exudate of a serous, fibrinous, or serofibrinous nature from the surfaces of the peritoneum. It is only when this preliminary condition is present that the bacteria present in the peritoneal sac can exert their effect and produce suppurative inflammation. Tavel and Lanz go so far as to say : "There is no primary bacterial peritonitis, since, under normal conditions, the peritoneum either absorbs any bacteria which are introduced into the abdominal cavity or is entirely unaffected by their presence."

Solid particles (mucus, blood, feces, etc.) alone are also incapable of producing purulent peritonitis, and when introduced aseptically, give rise to chronic adhesive peritonitis ; this effect, as Wieland has shown, is purely mechanical, and is the direct result of mechanical irritation and of pressure on the surface of the peritoneum. When, however, introduced together with bacteria, they play an important part in the pathogenesis of peritonitis, for they protect the bacteria and make absorption of micro-organisms difficult or quite impossible. Bacteria can readily develop in this protective wall, and are consequently able to manufacture their toxins, which, as a rule, first produce a chemic form of peritonitis, followed, as soon as the vicious circle is completed, by purulent bacterial peritonitis.

Some experiments show that the introduction of large numbers of extremely virulent bacteria may be followed by so rapid an absorption of toxins that general intoxication and death rapidly follow. In cases of this character there is no time for the development of purulent peritonitis. [The term " acute peritoneal infection " is much more applicable to these cases than " acute peritonitis " (Mayo Robson [1]).—Ed.] These are the most fulminating forms of so-called septic peritonitis in which no exudate and no pus are found and in which the only visible change in the peritoneum is a diffuse hyperemia, which is the expression of the initial stages of a chemic form of peritonitis ; the patients, however, die of general toxemia before the local process in the peritoneum has had time to develop. [In these cases bacteria are found in the omentum and changes may be found in the anterior mediastinal glands (Durham [2]). —Ed.]

I have already referred to the rôle of solid particles in the production of partial adhesive peritonitis, and have mentioned that it has been shown experimentally that aseptic solid bodies produce this form of peritonitis by the pressure and damage they exert on the endothelial layer of the peritoneum. With this, fibrin is separated from the blood or produced by changes in the peritoneal tissues themselves, and the fibrin thus formed covers the surface of the peritoneum. The fibrinous layer rapidly becomes vascularized and converted into fibrous adhesions. Clinically, a similar condition is seen in the liver of tight lacing, where its peritoneum is subjected to external pressure ; in chronic enlargement of the liver and the spleen the same conditions obtain, for here the

[1] Mayo Robson, *Brit. Med. Jour.*, 1903, vol. i., p. 242.
[2] Durham, *Jour. Path. and Bacteriol.*, vol. iv., p. 338.

peritoneum is compressed by the enlarged organs (provided the changes in the peritoneum in this condition are not due to the action of chemic bodies formed in the interior of the enlarged organs), and, finally, in chronic fecal accumulation the serosa is pressed upon and partial adhesive peritonitis results from distention of the intestine by the accumulated mass of fecal material in the bowel.

Peritonitis after laparotomy must be explained by the entrance of air into the peritoneal cavity, followed by drying and later by necrosis of the superficial endothelial cells of the peritoneum ; the effect can, of course, be produced only when the air is present in the peritoneal cavity for some time (Walthard, Delbet). When the endothelial layer of the peritoneum becomes necrotic in this way, its power of absorption also becomes impaired, and peritonitis results, for the bacteria and their products which enter the peritoneal cavity at the same time as the air can then exert their effects and produce purulent peritonitis. When no bacteria enter the peritoneum with the air, aseptic dry peritonitis with the production of adhesions results (Wegner).

SPECIAL CLINICAL ETIOLOGY.

The overwhelming majority of cases of peritonitis are secondary to disease of the abdominal organs covered by peritoneum or in close proximity to the peritoneal cavity, and must be considered as "secondary peritonitis." The great teachers of the older French school made this observation long ago, and all subsequent writers have confirmed this.

The three etiologic factors (bacterial, chemic, mechanical) enumerated above may all be at work in cases of peritonitis that originate from abdominal organs. It is true that accurate anatomic analyses of these cases are not always available, and that the exact point of origin cannot always be determined, but it may reasonably be assumed that one of these three factors is always responsible for the development of peritonitis. Bearing in mind what has been said on the general etiology of peritonitis, there will be no difficulty in applying this information to the interpretation of individual cases. The pathologic anatomy of peritonitis, the clinical features of the various forms of the disease, and, lastly, the study of the morbid changes in different organs which predispose to the development of secondary forms of peritonitis, are factors of importance in this connection.

Diseases of the intestine and female genital organs are the most prolific causes of secondary peritonitis and should always at once be suspected when any question arises as to the cause of peritonitis.

The Intestine.—**Diseases of the vermiform appendix** occupy the first place, and may lead to the development of numerous varieties of peritonitis, from mild circumscribed adhesive forms to very severe diffuse purulent and septic forms. A special section will be devoted to inflammations of the peritoneum in the right iliac fossa of intestinal origin, to which the reader should refer.

Ulcers of the intestine come next in importance to affections of the appendix. The various forms of intestinal ulceration (see p. 238) are

not, however, all of the same importance in the production of secondary peritonitis. Some acute ulcers of the bowel,—as, for instance, septic ulcers and ulcers following cutaneous burns, as well as some chronic ulcers,—*e. g.*, in leukemia, scurvy, uremia,—hardly ever lead to secondary peritonitis.

[In 22 cases of uremic ulceration of the intestines collected by W. H. Dickinson,[1] peritonitis was present in 7, and in 4 of these perforation had occurred.—ED.]

The importance, on the other hand, of typhoid, tuberculous, diphtheric, catarrhal, decubital, peptic, and syphilitic ulcers cannot be overestimated.

The chief importance of intestinal ulcers is their tendency to rupture into the general peritoneal cavity and set up perforative peritonitis, which, of course, is exceedingly dangerous. Typhoid ulcers perforate more rapidly than any other form, owing to the fact that they cause rapid tissue destruction and progress rapidly downward into the bowel-wall; there is, therefore, no time for the formation of protective adhesions. Next in importance are duodenal ulcers. The other forms of intestinal ulcer are less prone to perforate, and when perforation does occur, it is usually into a localized and shut-off part of the peritoneal cavity in which there is a collection of pus surrounded by adhesions.

Ulcers of the bowel are more apt to lead to the formation of localized adhesive peritonitis, which, of course, is not so dangerous as peritonitis from perforation. It begins when the destructive process in the bowel has extended down to the muscular or the subperitoneal coat. There may be either simple peritoneal thickening (in tuberculous ulcers this thickened tissue is, as a rule, infiltrated with tubercles) with the formation of adhesions to neighboring organs, or encysted and circumscribed collections of pus.

Carcinoma may lead to peritonitis in several ways. The most infrequent form is perforative peritonitis, for carcinoma of the intestine rarely goes on to perforation; when this does occur, general acute peritonitis usually follows. The commonest result is chronic adhesive peritonitis involving more or less tissue and starting from the serous covering of the affected portion of the bowel; as a rule, the process extends outward and produces considerable adhesions with neighboring organs. Occasionally, when perforation of the intestinal wall occurs after adhesions have already formed, an encysted peritoneal abscess with all the characteristic features of a fecal abscess (especially as carcinoma is usually situated in the large intestine) develops. Occasionally, too, the suppurative process extends in different directions and ultimately leads to the perforation of the abscesses into some adjacent organ—the intestine, the stomach, the bladder.

In ordinary intestinal **catarrh** the peritoneum is not involved; in some rare forms of severe infective or phlegmonous enteritis, however, the peritoneum covering the bowel is occasionally involved. As has

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[1] W. H. Dickinson, *Medico-Chir. Trans.*, 1894, vol. lxxvii., p. 116.

been shown above, catarrhal changes may allow micro-organisms to pass through the wall of the bowel.

Occlusion and stenosis of the bowel are of great importance in this respect. So far as their rôle in the genesis of peritonitis is concerned, a sharp distinction must be drawn between acute occlusion and the chronic forms of stenosis. In the latter, ulceration is chiefly responsible for inflammatory changes in the peritoneum; in addition, the nature of the stricture—for example, when due to carcinoma—exerts an influence in this direction. As a general rule, however, peritonitis is not frequent in chronic stenosis, and occupies a subordinate position in the general clinical picture.

In acute occlusion of the bowel (kinking, intussusception, and particularly in volvulus and strangulation) the conditions are different; the importance of secondary peritonitis in these conditions has recently been exhaustively studied not only clinically, but also from an experimental, and especially from a bacteriologic, point of view. It is well known that peritonitis does not always develop in the course of acute intestinal obstruction. No satisfactory explanation has so far been forthcoming why peritonitis occurs in one case and not in another where the structural changes and the clinical picture are essentially the same. Time is an important factor, for the longer the obstruction persists, the more marked are the changes in the wall of the bowel.

In exceptional cases peritonitis is due to chemic factors; in the great majority of cases, however, it is bacterial in origin. As a rule, peritonitis starts from the point of strangulation, this being the most altered portion of the diseased bowel, and from this point the process may extend over the whole peritoneum. Corresponding to the duration of acute intestinal obstruction, there is local or diffuse peritonitis. In acute gangrene with rupture of the bowel perforative peritonitis, of course, naturally results. When the process runs a slow course,—as, for instance, in intussusception,—local adhesive peritonitis, which ultimately leads to the formation of adhesions between two surfaces of the peritoneum, may occasionally occur.

That traumatic rupture of the bowel must lead to peritonitis is clear; some experiments on animals show that under specially favorable conditions peritonitis does not necessarily follow this form of intestinal perforation (for instance, when the perforation is closed by the omentum or when the perforated bowel is empty and becomes vigorously contracted); in clinical practice this accidental prevention of secondary peritonitis is so rare that it can hardly be taken into consideration.

Lastly, reference must be made to a form of partial chronic adhesive peritonitis which occasionally starts from the intestine in cases of fecal accumulation, even in the absence of any structural changes in the bowel; here the peritonitis is the direct result of the mechanical irritation of the peritoneum by the distended intestine.

The Stomach.—The stomach is occasionally the starting-point of peritonitis, though far less frequently than the intestine. Ulcer and

carcinoma are the two lesions most frequently responsible for this complication. In these gastric lesions several different forms of peritonitis may occur—viz., either perforation through a large opening in the stomach-wall before adhesions have formed, leading to general peritonitis, or merely to the formation of adhesions with some of the neighboring organs—colon, spleen, pancreas, liver; further, adhesions may first form and then perforation may occur into the colon, or, lastly, a local abscess may be set up.

Peritonitis is only rarely due to lesions of the stomach other than those just mentioned. A few exceptional cases are on record of perforative peritonitis following softening of the stomach-walls occurring shortly before death. Peritonitis after phlegmonous gastritis is also very rare. It is hardly necessary to mention peritonitis following traumatic perforation of the stomach. [I have seen extensive chronic peritonitis associated with advanced " cirrhosis of the stomach."—ED.]

The Liver and the Bile-passages.—In primary diseases of the liver and bile-passages local peritonitis limited in extent to the affected organ is more frequent than more general peritonitis.

Localized peritonitis in the so-called " corset liver " is the direct result of the mechanical pressure applied from without, and is merely localized perihepatitis.

Starting from the liver itself, the peritoneum becomes thickened over circumscribed areas of the liver surface corresponding to the position of abscesses or of syphilitic lesions in the organ, but is less frequent over hydatid cysts and nodules of new growth.

[There is a great difference between hydatid cysts imbedded in the substance of the liver and those which hang down from its under surface, the latter being exposed to more or less constant movement from the descent of the diaphragm in respiration and to friction, and show very considerable chronic peritoneal thickening.—ED.]

In interstitial hepatitis chronic thickening of the peritoneum extending over large areas of the surface of the liver is constant. [Considerable importance has been attached to local chronic peritonitis of the surface of the liver in cirrhosis, as it may account for ascites (Weber [1]); this " associated " ascites (Campbell Thomson [2]) is not of the same grave significance as ascites which develops as a terminal event in cirrhosis and seldom requires tapping more than once (Hale White [3]).—ED.]

In amyloid, fatty, and the nutmeg liver of chronic venous engorgement and in acute yellow atrophy the peritoneum covering the organ is unaffected.

General peritonitis of hepatic origin is rare, but it occurs in hepatic abscess when perforation occurs into the peritoneal cavity or when it leads to inflammation of the surface which extends to more distant parts of the peritoneum. A hydatid cyst may rupture either spontaneously, which is rare, or as the result of some external traumatism; in either

[1] F. Parkes Weber, *Edinburgh Med. Jour.*, April, 1903.
[2] H. Campbell Thomson, *Medico-Chir. Trans.*, vol. lxxxiv., p. 251.
[3] W. Hale White, *Guy's Hosp. Reps.*, 1892.

case diffuse peritonitis develops. In a few cases of multilocular echino-coccus, purulent peritonitis has been noted.

Of recent years a form of chronic adhesive peritonitis starting from the gall-bladder has deservedly attracted attention ; this form of peri-tonitis not only involves the gall-bladder, but leads to the formation of adhesions with the colon, the duodenum, and the stomach, and may consequently produce very serious disturbances.

[Mayo Robson,[1] Tuffier and Marchais,[2] Thomas,[3] F. Page,[4] and others have drawn attention to pyloric obstruction due to pericholecys-titic adhesions. The cases may closely resemble carcinoma of the pylorus. These adhesions may lead to kinking, strangulation, etc., and acute obstruction of the intestines. Niles has recently recorded a case of stenosis of the hepatic flexure of the colon due to this cause. Adhe-sions between the gall-bladder and vermiform appendix may possibly account for the simulation of appendicitis by gall-bladder cases.—ED.]

When gall-stones or definite chronic catarrhal cholecystitis are present, the genesis of this form of peritonitis is clear ; in the absence of any such conditions, however, there must either have been disease of the interior of the gall-bladder in the past, or this local adhesive peri-tonitis must belong to a category of cases which will be referred to later on.

Cholelithiasis occasionally leads to the formation of a pericholic abscess which subsequently perforates into some neighboring organ,— most frequently the duodenum,—but sometimes into the general peritoneal cavity. The gall-bladder, the cystic or the common duct may also per-forate into the general peritoneum ; however, when this occurs, diffuse peritonitis does not necessarily always develop, and in cases in which this accident does not occur, it must be assumed that the ruptured bile-passages were sterile. Perforation of the bile-passages may also, as is well known, occur from causes other than cholelithiasis—for instance, from ulceration of their walls in typhoid fever ; it has been stated that in very exceptional instances rupture of the bile-passages may be spon-taneous.

The Spleen.—Chronic inflammation of the peritoneum covering the spleen is incomparably more frequent than any other form of peri-tonitis. Chronic perisplenitis, usually leading to the formation of adhe-sions with the stomach, the intestine, the diaphragm, and the abdominal parietes, may either be universal or only partial. The former variety is seen in many forms of chronic splenic enlargement, such as occurs in leukemia, lymphadenoma, and malaria, whereas in more acute enlarge-ments of the spleen, as in relapsing fever, the development of universal inflammation of its peritoneal coat is not so common. The partial form of serositis is most common in association with infarcts and neoplasms of the organ.

[1] A. W. Mayo Robson, *Trans. Clin. Soc.*, 1893–94, vol. xxvii., p. 1.
[2] Tuffier and Marchais, *Rev. de Chir.*, 1897.
[3] Thomas, *Rev. Méd. de la suisse*, January, 1897.
[4] F. Page, *Brit. Med. Jour.*, 1897, vol. i., p. 205.

General peritonitis of splenic origin is rare. It may develop, for instance, when embolism of the splenic artery leads to the formation of a splenic abscess which ruptures into the general peritoneal cavity before there has been time for the formation of adhesions on the surface of the spleen. I have, however, also seen diffuse peritonitis of splenic origin set up in another manner—viz., in the course of typhoid fever an area affected with bacterial thrombosis on the surface of the spleen gradually led to the development of perisplenitis, and from this local lesion general peritonitis developed by direct extension of the inflammation to adjacent parts.

The Pancreas.—This organ does not play an important rôle in the genesis of peritonitis. In rare and exceptional instances acute pancreatitis (with destruction of tissue and abscess formation) has been known to lead to peritonitis as a direct result of rupture of the abscess in the pancreas into the general peritoneal cavity. [Acute pancreatitis, of course, leads to acute inflammation of the peritoneum in the lesser sac (bursa omentale) of the peritoneum.—ED.] Other forms of pancreatic disease—carcinoma, cysts, and pancreatic concretions—may all occasionally lead to local peritonitis.

The Kidneys and Bladder.—Purulent inflammation of the kidneys and purulent pyelitis may produce peritonitis; the purulent process first produces paranephritis, and later, by direct extension, peritonitis; occasionally the purulent process does not progress so gradually, but the abscess in the kidneys directly ruptures into the peritoneal cavity and this produces a diffuse form of purulent inflammation.

Hydronephrosis and renal cysts may be complicated by local forms of peritonitis; these possibly are produced by the mechanical pressure exerted on the peritoneum by the morbidly enlarged organ. According to Ebstein, hydronephrotic kidneys, when very large, are always adherent to the peritoneum, whereas adhesions inside the abdominal cavity are rare. When rupture of a hydronephrosis occurs, the contained fluid nearly always escapes into the peritoneum, but peritonitis does not necessarily follow, for, as is shown by recent investigations, normal urine is usually aseptic; rupture of a hydronephrotic kidney has even been known to occur several times in the same individual. Diffuse peritonitis results only when the urine contains pathogenic bacteria.

In the case of the urinary bladder, the more serious forms of ulceration of the mucosa and also the diphtheric forms of inflammation of this membrane, whatever their origin, may extend to the subperitoneal coat and eventually even to the peritoneum; this is followed either by localized suppuration (paracystic abscess) or by general peritonitis; occasionally, too, perforation of the bladder may occur under these conditions and is usually followed by general peritonitis.

The Genital Organs.—The female sexual organs are of greatest importance in the production of peritonitis, while the male sexual organs are comparatively insignificant. In male subjects the only cases in point are those in which the gonococcus enters the peritoneum from the seminal vesicles (Velpeau, Tillaux) or the vas deferens (Hunter), and are

seldom seen. It remains to be proved whether the gonococcus can ever find its way into the peritoneal cavity through the lymph or blood or directly through the tissues.

In female subjects, on the other hand, the sexual organs are absolutely the most frequent cause of peritonitis. This is due to the following factors:

In the first place, parturition, by injuring the parts, opens a ready channel for the entrance of pathogenic micro-organisms into the peritoneum. Puerperal peritonitis is sufficiently important to be described in a special section.

Since the publications of Noeggerath it is generally recognized that an extraordinarily large number of cases of pelvic peritonitis are due to gonorrheal infection. Gonococcal endometritis tends to spread to the tubes and ovaries, and ultimately to involve the peritoneum of the uterus and appendages. The resulting peritonitis is either acute, very chronic, or chronic with frequent acute exacerbations. These different forms of peritonitis all lead to the development of a great variety of anatomic lesions of the genital organs and to adhesions with neighboring organs, especially the intestine. Occasionally, though not frequently, gonorrheal infection may lead, first, to peritonitis confined to the tubes and ovaries, and, subsequently, to diffuse peritonitis.

In addition to these forms of peritonitis due to infection from the female sexual organs peritonitis which is not infective occasionally depends on the female sexual organs; the peritonitis produced under these conditions may be either localized or general. Menstruation *per se* predisposes the sexual organs to disease; the same applies to a variety of catarrhal affections of the vagina, the uterus, the tubes, and the ovaries, for in catarrh of all these organs there may be symptoms of irritation of the peritoneum covering them. Inflammatory affections of the female sexual organs and tumors arising there which may all lead to secondary peritonitis are also very important. Finally, from a consideration of the numerous traumatic influences to which the female genitals are exposed, all of which are capable of setting up peritonitis, it is not surprising that peritonitis in women very frequently starts from the sexual organs. [Gynecologic operations may, of course, set up peritonitis. In some instances where, from intestinal obstruction, the abdomen was reopened and adhesive peritonitis found, cultures were sterile (Kelly[1]).—Ed.]

Notwithstanding the importance of this subject in the pathogenesis of peritonitis, I must confine my remarks here to the brief sketch given above.

The Blood-vessels of the Abdominal Cavity.—The importance of the blood-vessels in the genesis of peritonitis has already been referred to in the paragraphs on the general pathogenesis of the disease, where it was shown that pathogenic bacteria are carried to the peritoneum through this channel, and that infection is in this way transmitted from distant purulent and gangrenous foci (so-called metastatic peritonitis). Peritonitis may, however, also originate from the blood-

[1] Kelly, *Operative Gynecology*, vol. ii., p. 81.

vessels situated in the peritoneal cavity itself—for example, in embolism of the mesenteric artery, in phlebitis involving the portal system or other veins—*e. g.*, the spermatic or the umbilical veins. The importance of phlebitis in the latter instance must be speedily emphasized, inasmuch as it appears to be the most frequent cause of peritonitis in the new-born. Umbilical phlebitis naturally originates from infection of the umbilical wound.   .

In cases of the latter kind the infection extends from the interior of the blood-vessel to the perivascular connective tissues, and so eventually to the peritoneum.

Other Morbid Conditions which may be the Starting-point of Inflammatory Processes Ultimately Involving the Peritoneum.—Peritonitis comparatively frequently starts from mesenteric or retroperitoneal glands, which may have been primarily infected with the bacillus of tuberculosis, the streptococcus, etc. This method of production of peritonitis is important, as many cases of so-called idiopathic primary peritonitis may possibly be thus explained. This variety of peritonitis may appear in one of two forms—either as general, even purulent, peritonitis or as the chronic adhesive form ; the latter is more frequent and often produces serious secondary consequences, such as kinking of the bowel, strangulation by bands, or incarceration of the intestine.

Peritonitis may also follow inflammation or suppuration in the abdominal parietes, suppuration in the upper portion of the rectum, burrowing and psoas abscesses, caries of the ribs, vertebræ, and pelvic bones.

Infection of the peritoneum through the diaphragm is also an important mode of origin. Inflammatory agencies, either corpuscular or chemic in character, may readily pass through the diaphragm and involve the peritoneum. Peritonitis may, for instance, follow pleurisy or abscess of the lung or other forms of pulmonary or pleuritic disease. That purulent infection of the peritoneum can occur through the diaphragm has been known to clinicians for a long time, thanks largely to von Recklinghausen's researches.

This sketch touches on all the essential points of the clinical etiology of peritonitis. In all the cases mentioned some pathogenic agency enters the peritoneum from neighboring parts, or at least exerts an effect on the peritoneum from the neighborhood.

In another group of cases the infective agent enters the peritoneum through the blood ; here peritonitis is spoken of as a complication or a metastasis of some acute infective disease. The important point about this category is that the pathogenic micro-organism under certain circumstances and in concrete cases also infects the peritoneum. This event, as has already been shown, is very rare, peculiar as this may seem. Clinical observation has shown that peritonitis in the course of infectious diseases is usually secondary to infection of one of the abdominal organs or to some other focus of infection in the peritoneum, such

as those enumerated above ; in the great majority of cases the infection of the peritoneum does not occur through the blood-stream. Our former views on this subject must be revised, as modern observations contain very little evidence pointing to the occurrence of purely hematogenous forms of peritonitis in the course of the infectious diseases. My personal experience coincides with the following summary of these observations :

As regards the exanthemata, the most experienced observers agree that peritonitis either does not occur at all in the course of variola, scarlatina, or measles, or that it is "exceedingly rare" ; others state that peritonitis never complicates these diseases unless there is, in addition, some "local" cause present to account for its development. Taking into consideration the extreme prevalence of measles, scarlatina, even small-pox, and the rarity, comparatively speaking, of peritonitis as a complication, it will readily be admitted that in these rare cases no pathogenetic relation between the infectious disease and peritonitis can be postulated.

The same holds good in the case of typhoid and typhus ; genuine (hematogenous) peritonitis is a rare event if it ever occurs in these diseases. In typhoid fever, peritonitis, apart from the perforative form, is nearly always of intestinal origin. Liebermeister, in an analysis of 2000 cases of typhoid fever, found peritonitis only 16 times (apart from those due to perforation), all of which were of intestinal origin. In a recent case of Moser's the hematogenous origin of the serofibrinous peritonitis was not absolutely certain, since it could equally well have arisen from an infarction of the spleen. Moreover, a "sticky membrane" appeared to cover the spleen.

In erysipelas the peritonitis has been present in cases where the abdominal wall was also involved in the erysipelatoid process. A few exceptional cases are on record of peritonitis in facial erysipelas. In malarial fevers chronic perisplenitis is found, but not general peritonitis. In epidemic cerebrospinal meningitis and diphtheria peritonitis has not been observed. Influenza has already been referred to (p. 724).

In cholera peritonitis is absent. In dysentery peritonitis is unquestionably of intestinal origin.

In the case of plague, Albrecht and Ghon have never seen anatomically general peritonitis set up by the plague bacillus, nor has H. F. Müller seen it clinically.

In croupous pneumonia, meningitis, as we know, is not particularly rare, whereas peritonitis is almost unheard of unless there is, at the same time, some pleurisy and the pneumococci work their way through the diaphragm and enter the peritoneal cavity in this way. I have already pointed out above (p. 718) that pneumococci are occasionally found in the exudates in peritonitis, and that this micro-organism may, in fact, be the only bacterium present, so that in given cases it must be assumed that the pneumococcus is the cause of the peritonitis, but in these cases pneumonia is not present. For the present the path by

which the pneumococcus gains entrance to the peritoneal cavity in these cases remains uncertain.

There are, therefore, only two acute infective diseases left which may lead to the development of peritonitis through the blood, and they —acute articular rheumatism and septicemia—(see pp. 724, 725) only rarely produce this complication. In exceptional cases the occurrence of peritonitis in scurvy has been described.

When we consider how frequently the pericardium, pleura, and even the meninges are affected in the various acute infective diseases enumerated above, it is indeed astonishing how rarely the peritoneum is involved.

It seems hardly possible that we have to do with a diminished vulnerability of the peritoneum as compared with other serous membranes. Possibly better arrangements exist in the peritoneal cavity for removing infection than is the case with the pleura and meninges. Reference has already been made to three such factors—viz., phagocytosis, the bactericidal power of the peritoneal fluid, and the powerful absorptive capacity of the peritoneum. We find in the blood itself, with most of the named infective diseases, relatively few bacteria; probably, therefore, but few will get into the peritoneal cavity from the blood at any one time, and these few could be quickly removed or rendered harmless before they had time to exert their influence.

Tuberculous peritonitis will be considered in a special section.

Syphilis plays a comparatively subordinate part, for syphilitic peritonitis is rare, especially in adults. Localized adhesive peritonitis is the most frequent form; it appears as a partial inflammation of the peritoneum covering various abdominal organs, such as the liver and the intestine, when involved by syphilitic lesions; this syphilitic peritonitis usually remains strictly localized, and only exceptionally leads to the development of diffuse forms of the disease, with exudation of fluid into the peritoneal cavity.

[Universal chronic perihepatitis is occasionally associated with syphilis; in Hale White's [1] 22 cases syphilis was the apparent factor in 3. Cheadle [2] believed that perihepatitis is more marked and more frequent in association with syphilitic disease of the liver than in any other condition. In rare instances numerous minute gummata may involve the capsule so extensively as to set up universal chronic perihepatitis (Sharkey [3] and N. Moore [4]).—Ed.]

The diffuse peritonitis due to perforation of syphilitic ulcers of the intestine is rare; when perforation occurs in syphilis of the bowel, the resulting peritonitis presents the same features as "perforative peritonitis" due to any other form of intestinal perforation. In children and in the fetus syphilitic peritonitis is much more frequent than in adults; as a matter of fact, fetal peritonitis is, in the great majority of cases,

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[1] Hale White, *Allbutt's System of Medicine,* vol. iv., p. 121.
[2] W. B. Cheadle, *Some Cirrhoses of the Liver,* 1900, pp. 41, 43.
[3] S. J. Sharkey, *Trans. Path. Soc.,* vol. xxxiv., p. 118.
[4] Norman Moore, *ibid.,* p. 133.

syphilitic in origin. The morbid changes seen under these circumstances vary : the peritonitis may be serofibrinous, purulent, or adhesive, and occasionally the adhesions may lead to constriction of the bowel.

As the chief reasons which justify the acceptation of traumatic peritonitis as a special form have already been given, it will only be necessary here to refer to the subject.

So-called puerperal peritonitis in the fetus and in the new-born has been vested with the dignity of an independent and special form of peritonitis. Etiologically, these two forms of the disease are identical, for they are both due to infection of the peritoneum by septic organisms during the puerperium. The only difference lies in the method of infection. In puerperal peritonitis in the fetus the infective organisms enter the fetus from the mother and travel through the placental circulation to their destination in the peritoneum of the fetus. The evidence of infection is found not only in the peritoneum, which may contain hemorrhagic and serous exudate, but also in other serous cavities of the fetus, such as the pleura, pericardium, and meninges. In the new-born, infection of the peritoneum almost invariably occurs by way of the umbilicus, and, as a rule, through the lymphatic vessels (Buhl). Here septic lymphangitis is the starting-point of peritonitis ; therefore the latter form of infection corresponds to the septic traumatic form of peritoneal infection seen in adults.

Lastly, another group of etiologically related cases must be specially mentioned here, viz. :

PRIMARY IDIOPATHIC PERITONITIS.

Ever since peritonitis has been the subject of scientific discussion,— *i. e.*, during the whole of the last century and up to the present time,—the question has been debated whether a primary idiopathic form of peritonitis exists. While, on the one hand, competent observers believe that it occurs and some clinicians actually described epidemics of this disease, on the other hand, equally competent observers, such as Louis, called attention to its extreme rarity and went so far as to doubt or even completely to deny its existence. It is impossible to discuss this interesting question in the narrow limits of this work, and all historic details of the controversy must, therefore, be omitted ; all that can be said is that, while general practitioners usually believe in idiopathic peritonitis, its existence is not accepted by pathologic anatomists ; this is due to the fact that the latter are frequently able to discover starting-points for the inflammation of the peritoneum at the postmortem examination, whereas the practitioner may not have succeeded in finding these primary foci and consequently may have been led to assume the existence of an idiopathic form of the disease. The antagonism of pathologists to the conception of idiopathic peritonitis has of late years been supported by bacteriologic investigation of doubtful cases.

In order to arrive at a fairly accurate conception of this question, two things are necessary : In the first place, it must be clear what is to be understood by primary idiopathic acute or chronic peritonitis ; in

the second place, reliable criteria, to enable us to decide whether we are entitled to assume the existence of an idiopathic form of the disease, must be formulated and adhered to in every case.

Peritonitis, either acute or chronic, may be called primary and idiopathic when the disease of the peritoneum is the only pathologic-anatomic condition present, and when no other morbid condition can be discovered anywhere else in the body. All pathologic and clinical evidence of acute or chronic infective diseases must be absent, and there must be no anatomic evidence of any other disease whatsoever which could lead to the development of peritonitis either by infection or by intoxication through the blood.

It will be seen from this definition that the existence or the absence of primary idiopathic peritonitis can be established only by careful pathologic-anatomic examination. The clinical history alone, however accurate it may be, can never be sufficient to justify a diagnosis of primary peritonitis. It is only, therefore, when a clinical history and definite anatomic data are present that it is justifiable to raise the question whether a given case of peritonitis should be considered idiopathic or whether it should be regarded as one of the many possible secondary forms of peritonitis. Even under these circumstances, however, a definite decision can probably never be made *intra vitam*.

It is, of course, unnecessary to postulate that in the idiopathic form of peritonitis special baneful factors must be at work. The question to be decided is simply whether the well-known chemic and bacterial agencies producing inflammation of the peritoneum can get directly into the peritoneal cavity and produce inflammation there alone, without, at the same time, setting up any other recognizable anatomic changes or clinical manifestations in any other organ of the body, and, lastly, whether they can do all this in the absence of any evidence, either clinically or anatomically, of their point of entrance into the body. Personally, I believe that this possibility cannot *a priori* be negatived. Pathogenic agents can readily, as we know, gain an entrance into the organism and set up the most severe disturbances without leaving any trace as to their point of entry, so that when the disease is once developed, it becomes quite impossible to say where it entered the organism. This is illustrated by tetanus, a disease in which the small cutaneous lesion, through which the pathogenic factor gained an entrance into the system, may be completely obscured and obliterated when the spasms have set in. It is probable that exactly the same process occurs in those forms of traumatic peritonitis following some injury with a blunt instrument or a fall, etc., and in which the skin is not injured; this question has already been dealt with in a previous paragraph. (See p. 721.)

There are two conceivable paths by which pathogenic bacteria might gain an entrance into the peritoneal cavity without leaving any trace at the point of entry into the organism and without leaving any recognizable evidence of their passage through certain organs. In the first place, bacteria or their products may enter the body through the female

genital organs, especially during the time of menstrual disturbance and changes of the parts; this possibly explains the comparative frequency of idiopathic peritonitis after menstruation. In the second place, bacteria and their products may enter the body through the intestine, especially during certain functional disorders (diarrhea, fecal accumulation). An argument against the latter assumption is that peritonitis ought to follow functional disorders of the intestine much more frequently than it actually does if these disturbances are really a prolific source of inflammation of the peritoneum; but, again, it must be insisted that the bowel, when undamaged and histologically intact, does not allow pathogenic micro-organisms to pass, as has been proved experimentally in peritonitis occurring in fecal accumulation. At present it must be considered as definitely settled that the agent causing the inflammation can get through the bowel only when the tissues of its wall are damaged or diseased. This condition of affairs is present when the peritoneum becomes infected by continuity in an attack of enteritis or in some other condition. Under these conditions the peritonitis is not primary, as has hitherto been assumed.

A great deal has also been written on the existence and the non-existence of so-called idiopathic " peritonitis from cold." The various experiments which have been performed on this question will not be described here, as the evidence adduced from them is at present contradictory and ambiguous. Cases, however, are on record of patients developing acute peritonitis after exposure to cold, getting wet when overheated, and after lying for a long time on damp ground. In the light of our present knowledge it certainly cannot be assumed that catching cold alone causes purulent peritonitis, since the presence of pyogenic micro-organisms is necessary before suppurative inflammation can occur in the peritoneum or elsewhere. It might be imagined, however, that exposure to cold might lead to certain circulatory disturbances in some parts of the body which would favor the arrest and the development of bacteria. The question which always remains to be answered in cases of this kind is, Where do these micro-organisms come from and how do they gain an entrance into the peritoneal cavity?

The possible existence, therefore, of an idiopathic form of peritonitis cannot be entirely denied, even from the standpoint of modern etiologic investigation. The fact that this form of the disease is so rare can be explained by the special physiologic activities of the peritoneum described in the section on the General Pathogenesis of Peritonitis, where it was shown that a number of conditions must always be fulfiled before acute general peritonitis can develop. All these considerations, however, do not make the occasional occurrence of idiopathic peritonitis absolutely impossible; but the question cannot be definitely settled in any given case until a pathologic-anatomic examination has been carried out.

I must preface my remarks on this form of peritonitis by saying that my personal experience does not at present justify me in assuming that acute idiopathic peritonitis as defined above ever occurs. In all

the cases which seemed to me to be of this nature, postmortem exam-
ination showed a definite origin for the inflammation of the peritoneum,
and that, in reality, it was a "secondary" form of the disease. (See
case of my own, pp. 723, 724.) In other cases, where there was no
autopsy or the patient recovered, the crucial test was lacking. The cases
recorded by the older writers are often too inexact to base any definite
opinion on as to their nature, and this account must, therefore, be lim-
ited to the more recently recorded cases, which, however, are few and far
between. P. Guttmann, for instance, among 82 cases of recent dif-
fuse exudative peritonitis, mentions 8 cases of idiopathic peritonitis; but
the clinical accounts and the postmortem details furnished by this writer
are far too brief to justify any critical expression of opinion as to
the validity of his claims; I can merely assume that his judgment is
correct and must take his diagnosis on faith. Litten states that he has
seen a very small number of fatal cases of undoubted general peritonitis
in which no point of entry for the infection could be discovered, although
most careful postmortem examinations were made in all the cases. Ley-
den, one of our most excellent observers, has reported three cases of idio-
pathic acute peritonitis in detail. I must confess, however, that even
in his cases I do not see any convincing proof that they were really
acute idiopathic peritonitis. One of the cases, a man of twenty-eight
years, recovered; in this instance the right iliac region was particularly
tender on pressure, and more resistant on palpation than in any other
part of the abdomen. Although the exudate was by no means strictly
circumscribed, and although recovery occurred within two weeks and a
half, the possibility that appendicitis (scolecoiditis) and perityphlitis
may have been the starting-point of the peritonitis cannot be denied.
In the two other cases Grawitz is inclined to believe that the bacteria
gained an entrance into the peritoneum through the genital organs, and
he consequently denies that in either of them the inflammation of the
peritoneum was primary and idiopathic. I am not aware of any cases
of idiopathic acute peritonitis more convincing than those just quoted.

Grawitz has collated 867 cases of peritonitis examined postmortem
in the Pathological Institute in Berlin, and of these, only 13 are headed
as spontaneous or rheumatic (primary suppurative) forms of peritonitis.
From a critical consideration of the latter cases Grawitz comes to the
conclusion that they are not examples of primary peritonitis.

For purposes of illustration four cases which Stooss and Tavel have
quite recently reported may be quoted. In all four there was a pneu-
mococcal peritonitis in children, a form which has always hitherto been
regarded as the type of acute primary idiopathic peritonitis. Let us
examine the course of these cases: Case 1 began with vomiting, abdom-
inal pain, fever, and convulsions. On the second day there were dim-
inished resonance and râles at the apex of the left lung, herpes labialis,
enlargement of the spleen, and not until the tenth day from the onset,
distinct signs of peritonitis. In case 2 there was tonsillitis, then
peritoneal symptoms. Case 3 commenced with attacks of colic, diar-
rhea, and vomiting. In case 4 a previous attack was accompanied

by violent cough; a few weeks later nephritis set in and then peritonitis. All these patients recovered after surgical interference. No critical observer, not even Stooss himself, would venture to assert that the pneumococcus was localized first and foremost or alone in the peritoneal cavity.

[The possibility that the appendix is the primary source of infection in cases of apparent acute idiopathic peritonitis must always be borne in mind. As Hawkins[1] has shown, there may be microscopic abscesses in the wall of the vermiform appendix, which may escape detection at the autopsy of a case of acute peritonitis, the appendix appearing normal to the naked eye, but which, nevertheless, have ruptured and given rise to the peritonitis, which was otherwise unexplained and might have been termed "idiopathic."—ED.]

From a consideration of these facts I believe that idiopathic acute peritonitis, as defined above, and in the strictest sense of the term, is not impossible *a priori*, and that its occasional occurrence is conceivable, but that no indisputable evidence of its actual existence has so far been adduced.

As to a chronic form of idiopathic peritonitis, the literature is more prolific; Galvagni and H. Vierordt, as well as many of the older writers, have contributed to this subject; in addition there are a number of isolated case-reports during the last decade by Quincke, Smidt, Mead, Rehn, Lindwurm-Bauer, Riedel, Rossi, Molinari, Fiedler, A. Fraenkel, and others. The controversy as to the existence or the non-existence of this disease has gone on for some time, and the question has been answered in different ways. Some authors assume with Louis that all these cases are primarily tuberculous, while others are strongly opposed to this view. Seiler has put forward the hypothesis, which, by the way, has never been substantiated by clinical or pathologic evidence, that ascites occurring in childhood, provided it is not due to tuberculosis, is always due to *syphilitic* disease of the liver. Of recent years, many authors have again argued in favor of the idiopathic character of many forms of chronic inflammation of the peritoneum.

Let us formulate once more clearly and distinctly what is to be understood by chronic idiopathic peritonitis; this term can only be applied to peritonitis which develops primarily—*i. e.*, is the only pathologic change whatever in any part of the body when the whole body has been carefully examined; for any lesion elsewhere might conceivably be the starting-point of peritonitis. This involves examination of the body with all the most modern methods of research. It is not necessary that the lesion found should be shown to be the starting-point of the particular peritoneal inflammation, but it is sufficient to find any organic change which is known to be occasionally the point of origin of peritonitis.

That this may actually be the case has already been shown for one particular form of peritonitis, namely, for partial chronic adhesive peritonitis following mechanical irritation of the peritoneum; this form of

[1] H. P. Hawkins, *Diseases of the Vermiform Appendix*, 1895, p. 68.

inflammation, as I have shown, may manifest itself in chronic circum-scribed thickening of the serosa over certain parts of the bowel in chronic fecal accumulation, without the presence of any organic changes in the bowel-wall. But these cases are not now under consideration. We are simply trying to decide whether or not there is such a thing as exudative peritonitis, often with enormous accumulation of serous and serofibrinous fluid in the peritoneal cavity, which can be considered idiopathic in the sense defined above.

As in the acute form, it must be insisted that the only really conclu-sive criterion is the pathologic-anatomic examination of each individual case in which the diagnosis of idiopathic chronic peritonitis is made. What is the outcome of the experience of pathologic anatomists on this point as judged by examination of the available material in the very brief and cursory manner possible in this hand-book?

Two of Galvagni's 12 cases terminated fatally, but details of the postmortem appearances are not forthcoming; one of his cases recovered for a time, but died a year later after having suffered from persistent and obstinate diarrhea for several months before death; the other patient, a woman, died with symptoms of "tuberculous meningitis." Vierordt reports 1 fatal case among 29 patients, in a woman the sub-ject of acute rheumatism seven years before her death. Two years after the attack of rheumatism she developed edema of the lower ex-tremities, ascites, and underwent paracentesis abdominis and later a laparotomy. On autopsy the following conditions, which are of special interest, were found: total calcified pericardium, with hypertrophy of the right ventricle, chronic venous engorgement of the liver, spleen, and kidneys, perihepatitis and perisplenitis, serofibrinous exudate, and ex-tensive adhesions between various loops of intestine. Vierordt himself, it is true, does not consider this case a perfectly typical one, and admits that it is "not quite pure and uncomplicated"; the question arises, Are we ever justified in adducing such clinical conditions as ascites from backward pressure and traumatic peritonitis from puncture and lapa-rotomy (these two operative procedures at the time this report was pub-lished (1869) being performed without antiseptic precautions) as evi-dence that there is such a thing as an "idiopathic" peritonitis? In other words, is it possible to utilize such observations in order to prove the existence of a disease which is not even recognized as a clinical entity? In a case of Bauer's there was: "Serofibrinous peritonitis; the gall-bladder was tightly adherent to loops of the intestine, uni-formly enlarged, and contained two gall-stones; no special lesions in any other part of the body." Since it has been shown by the researches of Tavel and Lanz that chemic peritonitis may develop in cholecystitis without any perforation of the bile-passages, it seems much more feasi-ble to assume that in this case the peritonitic inflammation was due to the entrance into the peritoneal cavity of some chemic irritant; this assumption is, at all events, much more rational than the artificial crea-tion of the conception of an "idiopathic" peritonitis. In the case of "chronic idiopathic" exudative peritonitis reported by Riegel, the

patient had repeatedly been punctured for ascites; at the autopsy there were pleurisy and pericarditis in addition to peritonitis. In Stiller's case, too, peritonitis followed pleurisy, the former disease being in this case secondary and not primary. The well-known case of Henoch's, as has already been shown, was without doubt traumatic in origin. Rossi has reported two cases without autopsy; Fiedler, four cases without postmortem examination. Molinari reports another case of Galvagni's with an account of the postmortem, but in this case the disease from which the patient died followed "disease of the chest" of several months' duration, which was accompanied by fever and hemoptysis, and at the autopsy, besides peritonitis, there was complete obliteration of the pleural cavity by adhesions; in one case reported by Signorini the left pleural cavity was obliterated while the right pleura contained a considerable amount of liquid exudate; Corazza also reports a case in which there was bilateral pleural exudation in addition to peritonitis. Although "tubercle" was not found in any of these cases and there was no bacteriologic examination, the clinical history alone and the anatomic changes furnish ample grounds for advancing critical objections to the diagnosis of "idiopathic" peritonitis.

It can hardly be maintained that the clinical reports and the conditions found after death in these cases support the existence of such a condition as idiopathic chronic peritonitis; the same may be said of a number of other reported cases and postmortem observations which I have not quoted, and in some instances these cases cannot be utilized at all in support of the assumption of idiopathic peritonitis. Most of the authors who adhere to the view that such a condition does exist rely on cases which recovered; and it is precisely the fact that these cases did recover that they adduce to show that the patients were not the subjects of tuberculous peritonitis, but of some benign, primary idiopathic form of the disease.

[As has been pointed out on p. 719, cases of gonococcal peritonitis often recover (Hunner[1]).—Ed.]

As regards such cases, it may be said, in the first place, that a great number of them cannot stand critical examination, even from a clinical point of view; many of them, moreover, show distinct and unmistakable evidence of some definite source of infection which stamps them etiologically as secondary forms of peritonitis. It must be admitted, however, that this does not apply to many of the cases and that it would be very arbitrary, indeed, to assume that all were of tuberculous origin. But does all this prove that cases of peritonitis which cannot be interpreted *in vivo* are really idiopathic and not of some other origin?

It is true that there is a certain clinical similarity between a few of these cases and that they show certain distinctive peculiarities. This must be attributed to the fact that many of the patients were children, and that, moreover, an extraordinary proportion of these patients were little girls and young women about the age of puberty (*ascite des jeunes filles*, Cruveilhier).

[1] Hunner, *Johns Hopkins Hosp. Bull.*, October, 1902, vol. xiii., p. 247.

[Bouilly,[1] from laparotomies on these cases, believes that the peritonitis is tuberculous and due to extension from primary tuberculosis of the internal genital organs.—ED.]

This does not, however, prove that these cases occupy a particular position among the different forms of peritonitis, and that their etiology is entirely different from that of all other forms. Quincke (see p. 698), however, takes the view that these cases must be differentiated from peritonitis in general.

In the foregoing remarks I do not by any means wish to deny that a primary chronic as well as a primary acute idiopathic form of peritonitis may not actually exist. What I do wish to imply is that, on the basis of the observations at present recorded, it cannot be positively proved that this form of peritonitis does exist. In order to establish its existence it will be necessary to point to negative results after the most careful postmortem and bacteriologic examinations; to have reports of clinical observation of doubtful cases which are complete from the onset of the initial symptoms; and to be able to exclude the effect of trauma of any form. "Catching cold" is always brought forward as a cause of the chronic as well as of the acute forms of so-called idiopathic peritonitis; but the only significance which can be attributed to this factor is that it prepares a suitable nidus and a soil for the development of inflammatory processes; certainly it cannot in any way be considered a true cause of inflammation. In all cases attributed to "catching cold" two points in the pathogenesis of the inflammation remain obscure—namely, what are the bacterial or the chemic factors which irritate the peritoneum and where do they come from?

THE PATHOLOGIC ANATOMY OF PERITONITIS.

THE various forms of inflammation of the peritoneum present certain anatomic appearances which differ according to a variety of factors. The most important determining factor is the etiology of the inflammation of the peritoneum; in other words, the anatomic appearances will vary according to the existence of bacterial, chemic, or mechanical irritation of the peritoneum, and according to the combination of any of these different factors which may be simultaneously at work. It is also important to know whether the attack has an acute onset and runs an acute course throughout, or whether it has a gradual and insidious onset and runs a slow course, or, lastly, whether it has an acute onset and then becomes chronic. The point of origin and the path that the inflammatory process travels in the peritoneum—i. e., whether it remains strictly circumscribed in one locality or becomes diffuse—are also important features in determining the anatomic lesions found postmortem. Certain clinical complications that may occur in the course of peritonitis also exert an influence in this direction. These different points will all be referred to in the description of the different clinical forms of peritonitis in later paragraphs.

[1] Bouilly, *Jour. de Méd.*, Paris, October 10, 1897.

A brief description of the anatomic lesions found in peritonitis will first be given.

In acute peritonitis the earliest change is injection of the serosa, which is very red and is often covered with hemorrhagic spots of varying size. In general peritonitis the hyperemia is not uniform, and some parts of the peritoneum appear to be more deeply injected than others. The hyperemia is, as a rule, most pronounced near the starting-point of the inflammatory process; in such cases the increased vascularity of the parts must, as a rule, be attributed to the action of the primary cause of the inflammation, such as internal strangulation of the bowels or volvulus. The visceral layer of the peritoneum is in all cases more hyperemic than the parietal layer.

In fulminating peritonitis, which proves fatal from septic intoxication or from so-called shock, the anatomic development of the process may be arrested at this stage before there is any real exudation. In these cases a pure culture of bacteria can occasionally be procured from the serosa. [The micro-organisms can be found in the omentum or in the lymphatic glands in the anterior mediastinum even when the peritoneum is sterile (Durham).—Ed.]

As a rule, exudation follows the stage of inflammatory hyperemia, the exudate consisting either of fibrin or of serous fluid.

The layer of fibrin first forms a thin, sticky covering tightly adherent to and intimately connected with the structural elements of the peritoneum, as was shown by Virchow and by Rokitansky. The adherent fibrin gives a grayish, dull appearance to the peritoneum. Adjacent organs and parts may become loosely adherent to the inflamed peritoneum. The layer of fibrin is not by any means of the same uniform thickness throughout, but may be much more massive and thick in one place than in another.

It is impossible to enter into a discussion of the general pathology of fibrin formation here. It need only be mentioned that considerable doubt has of late been cast upon the apparently self-evident assumption that the fibrin is derived from the blood. Schleiffarth, for instance, relying on Grawitz's researches, maintains that fibrin in these cases is a product of the metamorphosis of connective tissue, and that the outer fibrillar layers of the serous membranes are converted into fibrin. E. Neumann also strongly advocates the view that "fibrinal" transformation of connective tissue is the chief source of the fibrin, and found islands of epithelium in the fibrin in cases of recent peritonitis. The view that fibrin may also be derived from the blood must, however, still be retained—*i. e.*, that the fibrin is deposited from the fluid exudate found in the cavity of the peritoneum; this applies particularly to cases of peritonitis in which there is an abundant fibrinopurulent exudate. Borst also corroborates the statement made by different authors that in some forms of fibrinous inflammation of serous membranes the fibrin is, in the first instance, formed by swelling and degeneration of the superficial connective-tissue layers of the serosa; he states, however, that in addition there are forms of fibrinous inflamma-

tion in which the fibrin is chiefly derived from the exudate. It may, therefore, be concluded that both forms of fibrin formation can occur. When the peritonitis is not too limited, there is usually, in addition to the fibrin, a serous exudate which varies in amount in different cases. Small quantities—from 200 to 500 grams—follow the law of gravity and accumulate in the most dependent and the deepest portions of the peritoneal cavity—*i. e.*, particularly in the pelvis.

[Treves,[1] however, expressly states that the fluid does not gravitate first to the pelvis, as has been stated, and adds that the recumbent position probably does not encourage such gravitation. From observation during operations he finds that most of the free fluid is in the loin regions.—ED.]

Larger quantities may fill the greater part of the peritoneum and may force the diaphragm upward. In acute peritonitis the amount of fluid is, however, rarely excessive, and seldom exceeds a few liters of fluid. But occasionally as much as 20 liters are poured out in acute peritonitis. When there is such an excessive amount of peritoneal exudate, the intestines are often, although by no means constantly, chiefly in the right half of the abdominal cavity.

The serous exudate in peritonitis is yellowish, yellowish-green, or whitish in color; its specific gravity, 1.015 and higher; the percentage of albumin never under 1 per cent., and usually from 2 to 4 per cent., and occasionally higher—*i. e.*, from 5 to 6 per cent. Even when the fluid is quite clear to the naked eye, it contains a certain number of lymph-corpuscles, but only a very small number of desquamated epithelia. (For further details the reader should refer to text-books on general pathology.)

[An elaborate experimental investigation into the nature of the cells in the exudate produced by the intraperitoneal infection of micro-organisms has been carried out by Beattie,[2] who also examined cases of human peritonitis. In the early stages of acute peritonitis the polymorphonuclear leukocytes migrate from the blood-vessels in great numbers into the peritoneal cavity; in non-fatal cases they diminish after forty-eight to sixty hours, but in fatal cases they persist until the death of the animal; they are the main bacterial phagocytes. Mononuclear phagocytes are present at all stages; they are the most important cells in the exudate, and are chiefly produced by the omentum, for the endothelium covering the viscera may be quite intact when that of the omentum has been shed; they are ameboid, and are specially phagocytic for other cells, but may also ingest bacteria. The presence of large numbers of these mononuclear cells, if actively functional, in inflammatory exudates must be regarded as a favorable sign.—ED.]

Shreds and flakes are often found floating in the exudate; sometimes these solid particles are very small; at other times they form larger continuous membranous masses. They consist of masses of fibrin or of lumps of congregated pus-cells. In severe inflammations the exudate

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[1] Treves, "Peritonitis," *Allbutt's System of Medicine,* vol. iii., p. 611.
[2] J. M. Beattie, *Jour. Path. and Bacteriol.,* vol. viii., p. 129.

is occasionally of a reddish color, and there may be considerable quantities of blood in the fluid (hemorrhagic peritonitis); the latter condition is specially found in peritonitis associated with marked local venous engorgement, as, for instance, in volvulus and in internal strangulation of the bowel, and occasionally in tuberculosis and carcinosis of the peritoneum or in those general diseases which predispose to hemorrhage.

Friedreich was the first to describe a special form of hemorrhagic peritonitis which differs anatomically and etiologically from all the forms mentioned above. His patient, who had heart disease, had been tapped for ascites 16 times in the course of a year and a half. The aspirated fluid was always greenish yellow in color, except at the last tapping, when it was hemorrhagic. At the autopsy the parietal and the visceral layers of the peritoneum were both covered with a continuous membrane that was diffuse yellow in color and pigmented with dark-brown spots; it also showed in different places large and small hemorrhagic erosions, which were quite flat and appeared to be recent. On the peritoneum lining the anterior abdominal wall the new membrane was thick, and could be separated into several lamellæ, between which there were a large number of dark-red tumors about as large as a walnut, nodular, and prominent. These masses consisted of coagulated blood imbedded in the lamellæ of the newly formed membrane. The nodules could readily be separated from the underlying peritoneum, which was very vascular, but otherwise normal, shiny, and uniformly smooth. The intestine was everywhere free from adhesions. Friedreich compares this case with *pachymeningitis cerebralis chronica hæmorrhagica* (hematoma of the dura mater), and calls attention to the possible pathogenetic rôle of the numerous punctures of the abdomen that were performed; he argues that after each withdrawal of fluid the intra-abdominal pressure was reduced so that the pressure exerted on the intra-abdominal blood-vessels was repeatedly diminished; as a natural result, fluctional hyperemia of the parts occurred, followed by the exudation of blood and the other phenomena observed in this case.

[In a case reported by Cheeseman and Ely [1] a hemorrhagic pleural effusion was tapped 22 times, and then cured by the injection of a watery solution of iodin; this was almost immediately succeeded by a hemorrhagic pleural effusion on the opposite side, which was tapped 7 times and then cured in the same way. Seven months later a hemorrhagic peritoneal effusion occurred and was tapped no less than 43 times in five years, and then underwent spontaneous cure. A condition analogous to pachymeningitis hæmorrhagica was conjectured to be present, but as the patient survived, this could not be proved. The subject of hemorrhagic ascites is considered on p. 707.—Ed.]

Since the peritoneum possesses such enormous powers of absorption it follows that, before fluid can accumulate in its cavity, the inflammatory process must produce conditions which effectively prevent the removal of the fluid. These conditions may be partly mechanical obstruction of lymph-stomata, but it is more probable that the inflam-

[1] Cheeseman and Ely, *Amer. Jour. Med. Sci.*, August, 1899, p. 162.

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matory agent—*i. e.*, the bacterial poison—paralyzes the osmotic function of the peritoneum (see introduction).

In addition to these forms of fibrinous, serous, serofibrinous, and hemorrhagic peritonitis there is a purulent peritonitis (empyema peritonæi) which, according to current ideas, is always due to bacterial activity.

The degree of turbidity of serous and serofibrinous exudates, due to pus-cells, varies within wide limits—in some instances it is only slight and the serous character of the fluid predominates and the exudate remains freely displaceable in the abdominal cavity; in other cases the exudate becomes so greatly thickened that nothing but thick, creamy pus is found in the peritoneal cavity. The formation of pus in some forms of diffuse acute, especially puerperal, peritonitis is very rapid, and within thirty-six to forty-eight hours a liter or even more of purulent exudate may be found in the peritoneum.

In acute diffuse peritonitis the exudate is always freely movable at first; it tends, however, like a simple serous one, naturally to accumulate in the most dependent portions of the peritoneum—*i. e.*, in the pelvis, in front of the kidneys, and also occasionally between the loops of intestine. [Treves's views, which are not quite in unison with Nothnagel's on this point, were given on p. 752.—ED.] In protracted cases the adhesions between loops of the intestine and folds of the mesentery which were loose and flimsy originally undergo organization, so that the pus becomes encysted; in this way one or more collections of pus may be found in the abdominal cavity (*vomica abdominalis; cœliopyosis interna*).

Sometimes the exudate becomes sanious, offensive, not uncommonly quite putrid, dirty, discolored, grayish-brown or brownish-red, but not always purulent; occasionally it contains bubbles of gas, even in the absence of perforation of the intestine. This sanious exudate may contain solid masses, such as pieces of fecal material, the whole or part of the appendix, or foreign bodies, for it is more particularly in perforation of the intestine that the exudate becomes sanious in character. In puerperal peritonitis the exudate is often sanious, especially when there is some gangrenous affection of the genital organs or when some portion of the peritoneum becomes infected from a focus of gangrenous inflammation. In sanious acute peritonitis the amount of exudate is usually small, owing to the fact that these cases terminate fatally so rapidly. The peritoneum is, as a rule, of a dirty greenish-red color, often rotten, and may even be necrotic. In the latter event shreds of the serosa may become separated and lie loose in the exudate. The appearance of sanious characteristics is now attributed to the presence of anaërobic bacteria. Lastly, in rare cases, the exudate has been found to be of a gelatinous consistence (*cf.* the section on Benign Tumors—Pseudomyxoma).

[In a subacute case of peritonitis lasting five or six days and due to umbilical infection in an infant Porak and Durante[1] found material

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[1] Porak and Durante, *Soc. d' Obstet. et de Gynec. Ped.*, December, 1902.

looking like wax and of a yellowish color between the intestinal coils; it was composed of fat and fibrin, and was thought to be due to a large, non-motile coccus. There was no pus or exudation of any kind.—ED.]

The presence of gas in the peritoneal exudate in diffuse and in encysted forms of intraperitoneal suppuration has been much discussed. There can be no question as to the origin of the gas when there is perforation of some air-containing organ; there is, however, still some difference of opinion as to the spontaneous origin of gas in cases where this possibility can be excluded. In pleuritic exudates the spontaneous production of gas is universally denied [Dieulafoy,[1] however, describes pneumothorax due to formation of gas by anaërobic organisms.—ED.], but in peritonitic exudates there is not the same uniformity of opinion, for it is always possible that under certain circumstances gas-forming bacteria (Bacillus pyogenes fœtidus; Bacterium lactis aërogenes, etc.) may pass through the intestinal wall even without any gross mechanical lesion of its tissues; under these conditions it cannot be denied that these bacteria, if they once gain an entrance into the albuminous exudate, can give rise to the formation of gas with the same facility in this as in the contents of the intestine.

[The Bacillus aërogenes capsulatus, described by Welch and Nuttall[2] in 1892, gives rise to the formation of gaseous cysts in the organs and tissues of the body after death. It is usually a secondary and terminal infection, and occurs in the wake of other micro-organisms in moribund patients. It has, however, been obtained in pure cultures (Pratt and Fulton,[3] Pakes and Bryant[4]), and was repeatedly isolated from the blood during life in a case of infective endocarditis by Gwyn.[5] Though a most important cause of emphysema and gas-formation after death, there is little if any evidence that it gives rise to the formation of gas during life. In a case of suppurative peritonitis Vallas and Pinatelle[6] described gaseous cysts of the intestine and peritoneum which they regarded as antemortem, but the evidence is not convincing.—ED.]

In any case this event must be rare, and in the great majority of cases of gas-formation in peritoneal exudates, especially when considerable, it may be assumed that the gas entered the peritoneal cavity from the gastro-intestinal tract.

Incidentally, it may be mentioned that the pus found in intraperitoneal abscesses or in abscesses in the immediate vicinity of the intestine may often possess a feculent odor even though there is no putrid decomposition of the pus and no direct communication between the abscess and the lumen of the intestine. In these cases it must be assumed that gas passes through the walls of the intestine. It is a well-known fact, which to my mind has been proved beyond any question by the investigations of Obermayer and Schnitzler, that the healthy, living intestinal

[1] Dieulafoy, *Sem. Méd.*, 1900, p. 375.
[2] Welch and Nuttall, *Bull. Johns Hopkins Hosp.*, 1892, vol. iii., p. 81.
[3] Pratt and Fulton, *Boston Med. and Surg. Jour.*, June 7, 1900, p. 599.
[4] Pakes and Bryant, *Guy's Hosp. Reps.*, vol. liv.
[5] Gwyn, *Bull. Johns Hopkins Hosp.*, 1899, vol. x., p. 134.
[6] Vallas and Pinatelle, *Lyon Médical*, August 18, 1902, p. 215.

wall—and also the wall of the urinary bladder—allows gas to pass through it.

[The Bacillus coli and the anaërobic micro-organisms ordinarily present in the intestine, such as Bacillus putrificus coli (Bienstock), of course, produce very foul-smelling pus.—Ed.]

The tissues of the peritoneum itself in diffuse peritonitis are universally inflamed, though the inflammation is not everywhere of the same intensity; the visceral layer covering the intestine and the mesentery, omentum, and the solid organs are more markedly involved. So long as no adhesions have formed, the fluid exudate accumulates in the spaces of the peritoneal cavity, and, following the law of gravity, collects at the lowest portions of the abdomen, whereas the masses of fibrin adhere to the peritoneum and, after becoming detached again in the form of shreds, float in the fluid exudate. The most advanced changes in the peritoneum occur in the part of the abdominal cavity where the inflammatory process starts—for instance, close to a volvulus or a strangulated loop of intestine; in the latter instance peritonitis attacks the strangulated piece of bowel; this distribution of the inflammation is particularly marked in cases which run an acute course and are examined when the disease has existed only for a short time; in the region of the genital organs in recent puerperal peritonitis, and around the appendix vermiformis in peritonitis ex perityphlitide, etc., the serosa is specially inflamed in the early stages of the disease.

The peritoneum can be stripped off without difficulty; it is very vascular and edematous, and is infiltrated with serum and a large or small number of leukocytes. The lymph-spaces are also frequently dilated and filled with thrombi. The endothelial cells are at first in a condition of cloudy swelling, with nuclear proliferation, and are subsequently shed.

[The inflammatory changes are much more advanced in the peritoneum covering the omentum than elsewhere; thus the endothelial cells of the omentum may be shed, while those on the peritoneum covering the abdominal viscera are still intact. The endothelial cells of the omentum are seen to be ameboid and to thrust out pseudopodial processes; their protoplasm becomes vacuolated, the nucleus divides, and mitotic figures can be seen; the cells are shed and form mononuclear phagocytes. Cellular proliferation also occurs in and outside the blood-vessels and in the lymphoid tissue near blood-vessels (J. M. Beattie[1]).—Ed.]

The superficial layers of the peritoneum often show the changes which, as has been pointed out, lead to the formation of fibrin and pus-cells (Neumann, Schleiffarth, Boris).

In the tissues covered by peritoneum inflammatory changes may occur, particularly in the severe purulent productive forms of peritonitis, and may extend into the substance of underlying solid organs— *e. g.*, the liver—for several millimeters. The most important and significant changes from a clinical point of view are those in the intestine,

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[1] J. M. Beattie, *Jour. Path. and Bacteriol.*, vol. viii., p. 129.

which have always attracted the most attention among clinicians. As a rule, in diffuse acute peritonitis the intestine is more or less distended, sometimes to an extreme degree, some loops of the bowel being enormously dilated. The intestine is rarely contracted. The walls of the bowel are swollen, edematous, at the same time soft and friable, so that it is often difficult to separate the loops of intestine during the postmortem examination of such cases; under these circumstances great care must be taken while breaking down adhesions in order to avoid rupture of the intestine.

Walbaum found histologically, in cases which did not run too rapid a course, that the layers of the bowel under the serous membrane played an important part in the process of inflammation; in particular, he found collections of round-cells accompanying the blood-vessels as broad streaks running from the intensely infiltrated peritoneum right through the muscular layers, and becoming more or less marked toward the lumen of the bowel. The round-cells originate in the blood-vessels, but in addition there is extensive multiplication of the connective-tissue cells. Here and there in the deeper layers of the bowel deposits of fibrin are seen and sometimes hemorrhages. The muscle-fibers themselves show but little histologic change; in 3 cases out of 45 Walbaum found them infiltrated with pus-cells (inflammation in the wall of the bowel originating in a perityphlitic abscess and completely localized); in these cases the muscle-fibers showed necrotic change. Besides the above, the ganglion-cells of Meissner's and Auerbach's plexus constantly show changes which Walbaum described as degenerative. Askanazy also found considerable changes almost constantly in these ganglion-cells, as did Marchand and Orth, who describe the same in cases of typhus fever and dysentery.

The other anatomic changes in acute exudative peritonitis are the following:

In cases which run a favorable course the serous and fibrinous exudates may be completely absorbed, so that *restitutio ad integrum* occurs. Very frequently, however, some of the fibrinous exudate remains in the form of adhesions, which will be referred to below. In cases of hemorrhagic exudation traces of the blood remain behind in the form of slate-colored and dark areas of pigmentation.

When the puriform material is very thin, complete absorption may, it appears, soon occur. Some of the pus-cells in these cases probably wander directly into the lymph-stomata, while others undergo fatty metamorphosis and are subsequently absorbed.

Exudates consisting of pure pus in acute diffuse peritonitis probably never disappear unless they are removed by operative interference or discharge by means of a fistula into some abdominal organ or onto the surface of the body. When death does not occur too quickly in diffuse suppurative peritonitis, or when operative measures are not undertaken early in the disease, encysted collections of pus (abscesses) are formed, the walls of these abscesses consisting of granulation tissue. In the course of the disease septic (hectic) fever develops, or the liver becomes

secondarily involved when the radicles of the portal vein are involved in the suppurative process. In exceptional cases an encysted collection of pus developing after acute diffuse suppurative peritonitis may undergo metamorphosis, so that the contained pus becomes thickened. Occasionally—and this is by no means rare—the pus may burrow into some hollow organ or through the abdominal parietes, and open externally on the surface of the body (peritonitis ulcerosa). This may occur both in a case of freely movable empyema of the peritoneum and in encysted intraperitoneal abscesses. This termination of the disease does not, as a rule, occur for some time, and occasionally is delayed for months. Generally the disease has become chronic before the abscess perforates, but sometimes perforation occurs much earlier—*i. e.*, in the third or the fourth week of the disease. According to the statistics collected from the literature by Kaiser, the prognosis is better when the abscess spontaneously discharges on the surface of the body than when it opens into one of the hollow viscera, such as the intestine or the bladder ; recovery, it appears, is more frequent in the former case than in the latter, although several months may elapse before the patient is quite well. It is noteworthy that in peritoneal abscesses which are encysted from the start the prognosis seems to be better when they rupture into the bowel than when they discharge onto the surface of the body (*cf.* the section on Perityphlitis).

The abscess may discharge externally through any part of the abdominal parietes, but it is remarkable that the fistulous opening is usually either at the umbilicus or in its neighborhood. In perforation into the intestine the peritoneum is more widely destroyed than the mucosa. When this occurs, some of the bowel contents may enter the peritoneal cavity and may lead to sanious decomposition of the exudate. This probably is the reason why the prognosis is less favorable in rupture of a free exudate into the intestine than in rupture of an encysted intraperitoneal abscess into the bowel. Lastly, it is interesting to note that a large proportion of the cases of perforation of purulent exudates through the umbilicus occur in children, and, according to the bacteriologic results of the last ten years, it is the acute pneumococcal peritonitis of children which tends to form encysted collections of pus ; these either burst spontaneously or, as is now the custom, are opened with the best results. In puerperal purulent peritonitis, too, there is a tendency to spontaneous perforation.

The foregoing account has dealt with the anatomic changes seen in cases of diffuse peritonitis with an acute onset and running the most common course—viz., leading to the formation of a fluid exudate. As has been seen, however, a fluid exudate is not always formed. In fulminating cases running a course like severe septic infection (*sepsis peritonealis*), death may terminate the scene before any change except hyperemia has developed. It may be specially mentioned that it is by no means necessary that the inflammatory agents should suddenly enter the peritoneum in overwhelming numbers in order to produce this form of peritonitis and *sepsis acutissima,* for it is well known that this may

occur in cases of appendicitis without perforation and in puerperal infection of the peritoneum.

The change called *peritonitis adhesiva* or *indurata* plays a very important part in the clinical course and sequelæ of peritonitis. The number of possible variations in the distribution and situation of chronic adhesive peritonitis is quite astonishing ; these adhesions are of practical importance in two respects—viz., in the first place, they have a bad effect by forming dense connections between different portions of the bowel ; between the bowel and solid organs ; between two solid viscera ; between the latter and the abdominal parietes ; or, lastly, by causing constriction of the intestine ; in the second place, these adhesions occasionally act as firm protective barriers which effectually prevent the spread of grave morbid processes, such as perforation and purulent peritonitis. From these two points of view adhesive peritonitis will be considered in a special section to which the reader should refer for the clinical and etiologic details.

A large number of investigations have been published on the histologic processes concerned in the production of adhesive peritonitis ; the bibliography on this subject will be found in Graser's and in Roloff's papers on the subject. These investigations were specially undertaken with the object of making out the nature of the processes which lead to the formation of intraperitoneal adhesions after certain operative procedures on the peritoneal cavity. The most important point to be determined was the rôle of the endothelial cells. Although Graser found peritoneal wounds of the peritoneum occasionally united by first intention,—*i. e.*, by coalescence of the endothelial layer leading to closure of the wound and adhesion,—it must be assumed that in general the endothelial layer is removed or damaged before connective-tissue adhesions can form between the layers of the peritoneum. It is quite impossible to enter into the histologic detail of these processes here, and my remarks will be confined to a summary of the anatomy and the etiology of adhesive peritonitis.

In one group of cases adhesions are the relics of an acute attack of peritonitis in which the anatomic changes did not progress further than the stage of fibrinous exudation and degeneration of the serosa. In a second group of cases the adhesions persist, while the fluid exudate, whether serous or seropurulent, is absorbed. Lastly, in a third group of cases, adhesions form as a result of peritonitis which runs a slow and protracted course.

These different methods of formation are, however, insignificant as compared to the much larger number of cases ·in which adhesions develop from the very outset of the disease ; under these conditions the formation of adhesions is very insidious and slow, or possibly, in some cases, subacute. The adhesive and indurative inflammation may be diffuse and involve the greater portion of the peritoneum, but is more commonly limited to a circumscribed and often very small area of the peritoneum. To this category belong cases of pelvic peritonitis starting from the female genitalia, adhesive peritonitis near the vermi-

form appendix, and that in the vicinity of hernial sacs or around an inflamed gall-bladder; in addition, there are many forms of localized adhesive peritonitis which will be described *seriatim* in the section on Chronic Peritonitis.

The fibrous adhesions are either very delicate and thin, or coarse and thick; in the latter case they may form veritable masses of cicatricial tissue. When the adhesions are numerous and voluminous, the loops of intestine may be adherent to the abdominal walls, to other loops of intestine, or to other organs. The loops of intestine may be so firmly matted together and in such a complicated manner that it is surprising that their contents manage to pass through the bowel, for it is hard to understand how the peristaltic action can go on under these conditions. Bauer has compared the appearance of a mass of this kind to that of a uterine fibroid, for both look like a large, undefined mass of connective tissue traversed by large tubes and canals. Klebs has called the condition *peritonitis deformans.*

[Malcolm [1] has described a case in which, on opening the abdomen, a smooth, rounded mass filling the lower part of the abdomen was found. It felt like a cyst, and the condition was like that found when a broad-ligament tumor raises the pelvic peritoneum high up into the abdomen. As there were no intestines visible above the supposed cyst, it was percussed and found to be resonant and composed of matted intestine. He refers to two unpublished cases in which such a mass was cut into.—Ed.]

Henoch reports a case in a child of four years in which chronic peritonitis following injury had led to the formation of dense cicatricial callosities in some parts of the peritoneum which were thought to be new growths during life; after death the peritoneum, the subserous tissues, and the exudate were organized into a bluish-white, semitranslucent, cicatricial mass of tissue about $\frac{1}{2}$ to 1 cm. thick, which gave a peculiar grating sound when cut with a knife.

In cases of this class the large and the small omentum, as well as the mesentery, are always involved, and are thickened and considerably contracted. Owing to this retraction of the mesentery and the omentum, the intestinal canal is often considerably shortened, and the small intestine may be reduced to one-half its normal length; when this occurs, the mucosa of the shortened bowel is thrown into numerous folds which lie in close proximity to one another, while the intestinal glands remain intact.

In this way the whole peritoneal cavity may become obliterated. Occasionally the peritoneal adhesions inclose some serous fluid, while sometimes there are collections of pus or the thickened and calcified remains of old abscesses. The adhesions are sometimes pigmented.

Traube has described the following peculiar condition in diffuse adhesive chronic peritonitis—viz., numerous reddish or blackish, thread-like shreds of connective tissue, varying in length from a few centimeters to 15 centimeters, and attached to the small and large intestine

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[1] J. D. Malcolm, *Lancet,* 1901, vol. ii., p. 76.

and to the parietal peritoneum. They were attached to the peritoneum by a broad base, and then tapered down to a point at their free end. Attached to the mesentery were numerous flattened or rounded tendinous and tough cords, several millimeters thick, terminating in arborescent end-filaments. The coils of intestine were nowhere adherent to the abdominal parietes or to one another. Traube probably correctly interpreted these findings to signify that the patient—a woman suffering from puerperal peritonitis—became the subject of an adhesive form of peritonitis, and that the resulting adhesions were at first greatly stretched and subsequently ruptured by the violent peristaltic action of the bowel-walls accompanying a persistent and severe attack of diarrhea which supervened in this case.

Circumscribed adhesive peritonitis may develop in many different parts of the abdomen, may assume many various forms, and may lead to a number of different results. In this condition the most distant points of the peritoneal cavity may become firmly connected with each other. In a previous paragraph I quoted Treves as follows : " There is hardly any conceivable combination of adhesions which has not been met with."

The same author has given an admirable account of the factors responsible for the production of these adhesions, and has described the anatomic conditions which lead to the formation of adhesions between abdominal organs which are normally far removed from one another. Thus, adhesions have been found between the ascending colon and the ovaries, the transverse colon and the uterus, the sigmoid flexure and the cecum, etc. These adhesions can originate only when the parts involved are permanently in an abnormal position or are displaced at the time when adhesive peritonitis is in progress ; it must be assumed that in many cases the intestine is unduly distended with gas when adhesive peritonitis develops, and that pieces of the bowel normally distant from one another thus become approximated. It is quite impossible to enumerate all the various possible connections between abdominal organs which are occasionally seen ; some of these have already been described, and I shall have occasion again to refer to some of these anomalies of position in subsequent sections.

Circumscribed adhesive peritonitis forms either flat surface membranes varying in thickness and consistence, or cords which may be broad and band-like (" peritoneal false ligaments "), or rounded or even perfectly cylindric, and which vary in length and thickness in different cases. Obre described an adhesion which started from a loop of intestine near the ensiform cartilage, and extended to the parietal peritoneum in the inguinal canal of one side, and was 44.5 cm. long.

[J. Hutchinson, Jr.,[1] described a long, tough cord, of the consistence of catgut, which extended from the great omentum to the right broad ligament of the uterus. It was six inches in length and was regarded as being due to elongation of the pedicle of the hydatid of Morgagni, the hydatid of Morgagni being adherent to the great omentum.—Ed.]

[1] J. Hutchinson, Jr., *Trans. Path. Soc.*, vol. xlvii., p. 100.

Treves has called attention to the fact that in addition to the anatomic position of the organs the traction and pressure exerted on the adhesions by the peristaltic movements of the bowel, as well as by the friction to which they are exposed when the adherent organs change their position, all have an important effect on the form of the adhesions. In general we may say that chronic adhesive peritonitis involving organs which are fixed in positions, such as the female genital organs, leads to the formation of flat fibrous plaques, while in chronic peritonitis in the immediate vicinity of the intestine, where there is consequently constant and incessant motions of the bowel, round and cord-like adhesions are formed. It will be sufficient here to give this brief aphoristic review of the subject, since detailed consideration will be postponed to the section on Chronic Peritonitis, where the formation of adhesions will be referred to again.

One of the most remarkable forms of chronic indurative peritonitis I have ever seen occurred in an Italian, aged seventy-eight years, who died after a six weeks' illness from subacute nephritis. At the age of eighteen he had been treated for several months in the hospital at Verona for fever (malaria?); during this illness he suffered from pain in the left side; for the next sixty years, with the exception of an attack of varioloid, he stated that he had been perfectly well. At the autopsy there were a slight degree of atheromatous degeneration of the vessels, moderate hypertrophy of the left ventricle, and nephritis. All the abdominal organs were otherwise healthy and there was no trace of chronic peritonitis anywhere except on the spleen, which was enlarged to twice its normal size. Its capsule was white, and as it was quite impossible to cut through it, it was necessary to *saw* through it. The capsule was of a uniform thickness of about 1 cm. all over; in the center of the capsule there was a somewhat yellowish and softer layer; the inner surface of the capsule attached to the spleen and the external layer were as hard as stone and calcified. The substance of the spleen showed marked fibrosis.

The development of modern surgical treatment has provided opportunities not only for the study of peritonitis from the anatomic and descriptive standpoint, but also from the clinical and practical point of view. Mikulicz in particular has suggested a classification of peritonitis, separating some of the main forms of infective peritonitis according to their anatomic characters. This procedure is practical and meets the case. While adhering in the main to the subdivision proposed by Mikulicz, I shall, nevertheless, expand his original classification a little as follows :

A general infective acute peritonitis—in which the inflammatory agent simultaneously, or at least rapidly, invades the whole peritoneal cavity. The rapid spread of the infection is favored by intestinal peristalsis, which speedily carries the infection all over the peritoneal cavity. This form is seen in perforation of the bowel into the general abdominal cavity, in invasion of the peritoneum by microbes from the female genital organs, in infection from the appendix when it is not perforated and not adherent, and, formerly, before the days of asepsis, in most laparotomies. Anatomically, there may either be no exudate at all, or it may be fibrinous, serous, serohemorrhagic, purulent, or sanious. The most important point is that large portions of the peritoneum are uniformly infected and still no adhesions are formed.

A Progressive Fibrinopurulent Peritonitis (Mikulicz).—In this form the whole course of the disease is less severe, the infection does not involve the whole of the peritoneum at once, but is gradually progressive. The disease usually starts from some one point of infection, where the inflammatory reaction leads to the formation of a protective wall of adhesions, which for a time interferes with the further spread of the inflammation and also prevents the whole of the peritoneum from being infected at the same time. Gradually, however, either through a weak spot in the gut-wall or along the lymph-channels, the infection spreads to the adjacent parts of the peritoneum, so that fresh purulent foci continue to be formed until eventually the greater part of the peritoneum is involved in this process.

Lennander distinguishes, and justly too, from the above-described method of dissemination a form of peritonitis characterized by multiple encysted abscesses (called by Tietze, "disseminated"). Here inflammation does not progress, but is limited to several encapsulated foci of pus, absolutely distinct from each other. We will quote his words as to their probable origin : "At the time that infection of the abdominal cavity takes place the infective material is, in consequence either of peristalsis or abdominal pressure or movements of the body generally, carried almost at the same moment to different parts of the abdominal cavity. We must assume the infective material possesses but little virulence."

According to Mikulicz, the mode of progression in this form of peritonitis is probably not entirely irregular ; he believes that the spread of infection from one part to another depends on definite anatomic conditions. Though all the different parts of the peritoneal cavity are in free communication with one another, there are dividing partitions between them, there being anatomic conditions so arranged that the formation of adhesions in these parts can most effectually prevent the further extension of the infection. Thus, for instance, the transverse colon with its mesocolon separates the abdominal cavity into an upper and a lower half—viz., the supra-omental and infra-omental space. Communication between these two spaces can occur with the greatest facility along the ascending and the descending colon, and this communication can readily be closed by the formation of adhesions. In the supra-omental space the stomach and the liver may form a partition capable of preventing the extension of subphrenic abscesses to other parts of the peritoneum. In the infra-omental space the root of the mesentery and the mesentery itself may play a similar rôle. This topographic-clinical aspect of the subject suggested by Mikulicz is certainly very fruitful. Following in these lines, Rauenbusch, of Ponfick's laboratory, has collected a number of postmortem results, which, from a purely topographic point of view, he divides into the following four groups of localized purulent progressive forms of peritonitis : (1) Supra-omental ; (2) infra-omental ; (3) spreading from above downward ; (4) spreading from below upward. To group (1) belong chiefly subphrenic abscesses. Group (2) much more frequently presents clin-

ically, on account of the large surface of peritoneum involved, the symptoms of general peritonitis and nearly always starts from inflammation in or around the appendix.

Localized purulent peritonitis, in which, owing to the insidious formation of adhesions in advance of the infection, the spread of the inflammation is effectually prevented; this leads to the localization of the peritonitic process in one circumscribed area of the abdomen. Types of this form are perityphlitis, perimetritis, and subphrenic abscesses.

A diffuse chronic adhesive form of peritonitis which frequently follows acute inflammations; it may become arrested; it follows tuberculous infection of the peritoneum with relative frequency, and occasionally only leads to adhesions between certain organs; sometimes the remains of fluid exudate are found inclosed between different layers of the adhesive tissue.

A partial adhesive form of peritonitis: this form is found to vary greatly in extent and in its site, as has already been shown in the preceding paragraphs.

THE SYMPTOMS OF PERITONITIS.

It is impossible to give an inclusive clinical description of all the forms of peritonitis. The acute diffuse suppurative, the fulminating septic, the acute perforative, the circumscribed purulent, the tuberculous, the local adhesive, and several other anatomic and etiologic modifications of the disease all present such different clinical pictures that a comprehensive description of these several forms is quite impossible and must ultimately resolve itself into a description of the syndrome presented by each individual variety. In this section I shall, therefore, first give a simple review of the symptoms that occur in peritonitis, and then describe the different forms of the disease from the clinical standpoint. I am fully aware of the fact that it is difficult, or rather impossible, to group the different varieties of peritonitis under distinct headings and categories, for at best any such arrangement must be arbitrary and open to numerous objections; from the very nature of the problem a schematic subdivision of the subject cannot be made. In order to arrive at a clear and comprehensive description of the subject it will, however, be necessary to divide it up as suggested above. Since this subdivision is entirely for practical and clinical use, it has been made on those lines, and no attempt has been made to draw up a classification of the subject on a strictly scientific basis, which, as intimated above, seems to me impracticable. I believe that this treatment of the subject will meet the requirements of the practitioner at the bedside in the most satisfactory manner.

ANALYSIS OF INDIVIDUAL SYMPTOMS.

The symptoms of peritonitis can be arranged in a number of groups, as follows:

(*a*) Symptoms which are the immediate result of inflammation of the peritoneum itself and characterize the disease—*i. e.*, pain, exudation.

(*b*) Symptoms due to functional or anatomic changes in organs directly involved in the process—*e. g.*, the intestine, the bladder, the diaphragm, the abdominal muscles—*i. e.*, vomiting, constipation, meteorism, intestinal paresis, painful micturition, hiccup, rigidity or paresis of the abdominal muscles.

(*c*) General symptoms—fever, cardiac and circulatory disturbances, modification in the urine, changes in the general constitutional condition of the patient, septicemia, and toxemia.

GROUP A.

Pain is the chief symptom. The three special features of the pain of peritonitis are its spontaneity, exacerbation on pressure, and its persistence. They are observed in the majority of cases, but occasionally other conditions are present which lead the tyro to make an erroneous diagnosis. Thus perforation of the appendix following perityphlitis may at first be taken for intestinal colic, subphrenic abscess the result of gastric or duodenal ulcer, or for gall-stone colic.

In acute cases pain is never absent. Absence of pain in a few exceptional cases of perforative " peritonitis " after cessation of the agonizing pain following rupture of the bowel, or in some cases of septic " peritonitis " (in which the patients are occasionally free from spontaneous pain and from tenderness on pressure), must be explained by the fact that the condition is inaccurately described as peritonitis. In all these cases the patients die from the shock of the perforation or from sepsis before the anatomic changes of *peritonitis* have had time to develop. Pain, however, is always present in fully developed acute peritonitis, and is, moreover, extremely severe; occasionally it is so violent that the patient, if not in bed at the time of the onset, may faint. In fact, diffuse acute peritonitis is one of the most painful of all diseases.

The severity of the pain is explained by the facts that such an extensive surface is involved in the inflammatory process and that such a large number of nerve-fibers are simultaneously irritated. This also explains why the severity of the pain is in proportion to the rapidity with which the process develops, and is most severe in acute perforative peritonitis where the whole peritoneum becomes inflamed at the same time.

The sudden onset of pain, which is violent from the very first, occurs only in acute perforative peritonitis. In other forms of peritonitis the pain gradually increases in severity—sometimes slowly, sometimes rapidly, but always by degrees, until, eventually, it reaches its maximum intensity (the excruciating pain that appears suddenly in the perforative form is in reality due to the perforation and not to inflammation of the peritoneum). In many instances it is possible to gauge the extent and progress of the inflammation by the pain. Extremely severe tenderness may persist over the starting-point during the

whole course of the disease—for instance, in the ileocecal region when diffuse peritonitis starts from the appendix. Too much stress should not, however, be laid on this point, for this rule is not without exceptions, and the point of greatest pain, even at the onset, does not always correspond to the origin of the inflammation. The initial pain, for instance, in appendicitis may be most intense in the umbilical region or in the lower half of the abdomen on the left side.

The degree of pain and the patient's complaint of pain to some extent, of course, depend on the individual sensitiveness of the subject; but the general expression of suffering and the clinical features of the pain are characteristic and constant in all cases. It is continuous, and even in the mildest cases there is pain in the abdomen throughout the whole course of the disease. The slightest movement or pressure in some cases increases the pain to such an extent that it becomes quite unbearable, and for this reason the patients lie perfectly still and motionless, avoiding all unnecessary movements of the trunk and of the legs, which are often drawn up. The breathing is shallow, purely costal, and rapid, in order to avoid, as far as possible, deep inspiratory excursions of the diaphragm. The head and arms are the only parts of the body restlessly moved to and fro, as an expression of the patient's mental anxiety; but occasionally, however, they are also kept still. In very exceptional cases, particularly in some instances of perforative peritonitis, the patients, in the hope of relieving their fearful torture, toss about restlessly or may even get out of bed. The patients dread the pressure of the medical man's hand during examination, and try to avoid all pressure from the bed-clothes, as the slightest pressure over the abdomen may give rise to loud expressions of pain. Defecation, coughing, or sneezing may be veritable torture. On the other hand, it is sometimes, although not always, possible to pinch up a fold of skin over the abdomen without causing pain.

[Head[1] definitely states that there is no true cutaneous tenderness in peritonitis, and that if the skin only is picked up between the finger and thumb in acute peritonitis, the patient feels no pain, but directly the deeper structures of the abdominal wall are included in the gentle pressure, there is intense pain. The referred cutaneous pain of appendicitis may disappear when perforation into the general cavity of the peritoneum occurs, but it may persist when there is peritonitis (Sherren[2]). —Ed.]

From time to time exacerbations of the pain occur without any discoverable cause, and occasionally assume a colicky character, only with the peculiarity that, in addition, the *constant* pain persists and does not diminish. This colicky pain is due to intestinal peristalsis, for any contraction or movement of the intestines must necessarily stimulate still further the nerves of the peritoneum, which are already greatly irritated.

With the increase of exudation and, still more, when the abdomen becomes tympanitic, the pain becomes less severe; but this is not neces-

[1] H. Head, *Brain*, vol. xvi., p. 94.   [2] S. Sherren, *Lancet*, 1903, vol. ii., p. 816.

sarily a favorable sign, and, on the contrary, may have a very grave significance and point to paralysis of the intestine and cessation of intestinal peristalsis. The disappearance of pain when a considerable amount of exudation has collected is attributed by Lennander to the fact that in the presence of fluid the friction of the intestines against the parietal peritoneum is much diminished. According to him, the parietal layer is the only sensitive portion of the peritoneum.

In diffuse acute peritonitis the whole abdomen is, as a rule, uniformly painful and tender on pressure. Occasionally the pain radiates to the chest and may extend as high up as the shoulders. Sometimes one part of the abdomen is specially painful, or the site of persistent and continuous pain, which, as I have said, is usually the starting-point of the inflammatory process—for example, the uterus and the adnexa, the ileocecal region, or the neighborhood of the kidneys. Sometimes, however, there is no special localization of the pain, and it is not more severe at the point of origin of the inflammation. In the former case the local tenderness may be due to the fact that the inflammation is specially severe in this particular spot ; in other instances the severity of the pain in one spot may be due to certain physiologic factors which are not yet understood ; thus, to take an example, the frequency with which exceptionally severe pain occurs in the middle of the abdomen around the umbilicus.

In chronic peritonitis the pain is not so severe, but it has one characteristic feature of the acute form—viz., it is increased on external pressure. Spontaneous pain may, in fact, be entirely absent in chronic peritonitis and be felt only on pressure. Tenderness may also be absent, at least during some periods of the disease, for it is incorrect to say that pain is absent throughout the whole course of chronic peritonitis. The only form of chronic peritonitis in which this is possible is the strictly circumscribed adhesive form, which runs a slow and insidious course. In chronic tuberculous, carcinomatous, and the progressive purulent forms of peritonitis there may be no pain during long periods of their course, a fact which should be remembered in clinical practice.

In circumscribed peritonitis the pain is limited to the affected part. A detailed description and some exceptions to this rule will be given in full in the sections on the various forms of circumscribed peritonitis (perityphlitis, etc.).

The intensity of the pain in acute diffuse peritonitis, in addition to leading to characteristic changes in facial expression and attitude of the patient's body, also produces modifications in the action of the heart and in the circulation, which will be described below.

To consider now the second characteristic symptom, which is only subordinate in importance to the pain—viz., the **exudate**. This, as has already been shown in the section on the Anatomy of Peritonitis, may either be fluid or solid, and in the former case either quite free in the peritoneal cavity or encysted ; the quantity of the exudate may be considerable or insignificant ; there may be only one-tenth of a liter or it

may amount to several liters ; it may be serous, fibrinous, seropurulent, fibrinopurulent, entirely purulent, hemorrhagic, sanious, sanio-gaseous, or of the consistence of connective tissue.

Small quantities of free exudate, not amounting to more than several hundred cubic centimeters, may escape detection altogether, as they gravitate to the lowest parts of the peritoneal cavity—*i. e.*, the pelvic region or the flanks. Their diagnosis becomes easier in proportion to the amount of fluid in the peritoneal sac ; generally speaking, the detection of small quantities of fluid is less accurate in acute exudative peritonitis than in ascites, depending on backward pressure ; this, however, is merely due to the fact that the pain in the former condition renders the manipulations necessary to detect such small quantities of fluid very difficult or impossible, while a more thorough examination is possible in ascites from backward pressure. For the methods of physical examination to be employed in looking for the presence of fluid in the peritoneal cavity—*i. e.*, inspection, percussion, palpation— the reader should refer to the section on Ascites, where all the details are given ; in order to avoid unnecessary repetition, this will not be gone into again here, and only the following additional points will be given :

It is well known that an elliptic line drawn horizontally around the abdomen corresponds to the boundary between the dull percussion-note over the ascitic fluid and the clear and loud note obtained over the intestine floating above the fluid ; when the patients are lying on their back, this line is perfectly even, but in inflammatory exudative forms of ascites its contour may be changed. Gerhardt states that if this line is drawn very carefully in the latter affection, it sometimes has a jagged course. This peculiarity must be attributed to the existence of adhesions between some of the loops of the intestine which prevent the fluid from occupying a uniform horizontal level in all portions of the abdominal periphery. In the section on Tuberculous Peritonitis reference will be made to some anomalies in the distribution of resonant and dull notes in tuberculous and carcinomatous forms of peritonitis described by Thomayer. My own experience does not justify me in attaching much importance to these statements, for the points laid down by Thomayer are subject to numerous exceptions ; this is unfortunate, since his conclusions would be very valuable if only they were constantly true.

It is impossible to make any definite statements as to the rapidity with which the exudate forms, for it varies in peritonitis, as it does in pleurisy, within wide limits. In some cases it is possible to detect considerable quantities of exudate within forty-eight hours, while in other cases the amount of exudate is small, even after a much longer interval. In fact, the most serious form of peritonitis,—viz., *sepsis peritonealis*,—notwithstanding the development of the most virulent septic symptoms, runs its course with only a very small amount of exudate or even without any at all.

Although occasionally the pain is relieved as soon as considerable quantities of exudate accumulate in the peritoneal sac, this advantage

is nullified by the development of a number of other distressing symptoms, such as interference with the movements of the diaphragm, due, of course, to the combined result of the accumulation of fluid in the peritoneal sac and of the meteorism which soon comes on.

It is, of course, impossible to make out the composition of the exudate from the physical examination of the patient, but some help may be obtained from other factors, such as the underlying cause, the course of the disease, the temperature, etc. Occasionally cutaneous edema or a burrowing abscess develops and shows the septic character of the peritonitis. Generally, however, puncture of the abdominal wall is the only way of making out with certainty the nature of the exudate.

Although the presence of a fluid exudate in the abdomen and the characteristic pain of peritonitis are relatively the most valuable symptoms of peritonitis, they may both be present in the absence of peritonitis. In the section on Intestinal Obstruction in general and of Volvulus in particular attention was called to the fact that a large amount of hemorrhagic fluid may occasionally exude from the strangulated part of the intestine, and that, under these circumstances, particularly when other symptoms of volvulus or of internal strangulation (pain, collapse) appear at the same time, the picture of acute diffuse peritonitis may be simulated; but examination of the peritoneum may show complete absence of inflammation or only the slightest trace of inflammation at the point of strangulation.

Attention must be called to another source of error which is common, especially in chronic cases. It may happen that the fluid contents of the intestine, especially in the colon, imitate the presence of fluid exudate in the peritoneal sac; this may occur even with the small intestine, when, from gravity, loops of the bowel have dropped down into the flanks. Under these conditions the lateral dependent portions of the abdomen give a dull note on percussion, which disappears when the patients turn over on their side; this occurs because the loops of intestine, being filled with fluid, are so heavy that they change their position in the abdomen with every alteration in the position of the patient. The fact, moreover, that no fluctuation can be obtained over these loops does not necessarily militate against the diagnosis of peritoneal effusion, for fluctuation, as is well known, may be absent in peritonitis when there are adhesions. Nevertheless, I consider the differential diagnosis between a free peritoneal exudate and the presence of fluid in the intestine possible in the great majority of cases; in arriving at this diagnosis the following points must be borne in mind: When the dulness in question in the flanks is produced by the fluid contents of the intestines, distinct splashing can often be obtained over this area when a series of rapid short taps are given; another point is that the dulness is entirely absent in the lower part of the abdomen,—*i. e.*, above the symphysis pubis,—although the amount of exudate may appear to be very considerable; lastly, there is usually some difference in the dulness on the two sides, which is generally much more marked in the case of fluid in the intestines than in free peritoneal exudate.

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The detection of the exudate may be specially difficult when the fluid is encysted, as in chronic peritonitis. Under these conditions the most important criteria—the alteration of the percussion-note when the patient changes his position and fluctuation—are absent. If the encysted peritoneal effusion is in certain definite situations, such as the neighborhood of the umbilicus or in the right iliac region, the resulting dulness may lead the medical man to a diagnosis of localized peritoneal exudate ; the only way, however, to make quite certain is to aspirate or to obtain a distinct fluctuation. (For the details the reader should refer to the section in which the different forms of circumscribed peritoneal abscess (perityphlitis, subphrenic abscess, etc.) are described.)

Fibrinous exudates may occasionally lead to friction between the two layers of the peritoneum and to a rough friction-rub audible on auscultation, which resembles in its origin and characters the same phenomenon in pleurisy. Usually the friction-rub is produced by movement of the two layers of the peritoneum with respiration, less frequently by the peristaltic action of the intestine. It is heard most frequently and distinctly in the upper part of the abdomen, particularly over solid organs (liver, spleen), which move a good deal with respiration, and, occasionally, over tumors, but, for obvious reasons, is only rarely heard over the intestine. Generally speaking, a peritoneal friction-rub is rare, but when present, and both palpable and audible, the diagnostic significance is reliable.

Delicate and localized peritoneal adhesions cannot be diagnosed, and can at best be suspected only when certain definite groups of functional disorders appear. Thick and firm peritoneal bands may, however, when in specially favorable positions, such as the front of the abdomen, or more particularly when in the neighborhood of the female sexual organs, sometimes be felt. These adhesions will be dealt with in detail in the description of chronic peritonitis, where attention will be directed to the tumor-like indurations occasionally palpable in *peritonitis deformans*, and still more frequently in tuberculous peritonitis, which are often mistaken for tumors, and, therefore, lead to considerable difficulty in diagnosis.

GROUP B.

In contrast to the symptoms enumerated above, which may suitably be called obligatory, those considered in this group are less constant and are, generally speaking, more often seen in acute than in chronic peritonitis.

Vomiting is frequent, almost constant, in acute peritonitis, especially when the onset is rapid, with the possible exception of acute perforative peritonitis. It is impossible, however, to characterize it as a symptom of any one particular form of peritonitis. When the onset is gradual and the disease runs a chronic course from the outset, vomiting is usually absent. In acute peritonitis vomiting is one of the earliest symptoms, and in several of my cases it appeared before the pain.

For instance, a woman, while perfectly well and attending to her ordinary duties, was suddenly seized at 11 A. M. with nausea, vomiting, weakness, and

rolling noises in the abdomen, but kept about by sheer force of will; an hour later vomiting, at first of food, later of greenish mucus; soon after the vomiting violent pain in the whole of the abdomen, followed by an attack of peritonitis of medium severity; the patient recovered, and the cause of this attack, therefore, remained obscure.

Vomiting usually appears before the second day, and rarely later; it generally persists from the beginning of the attack for a number of days until the disease takes a favorable turn, and the peritoneal irritation stops (vomiting in addition to the pain must be considered one of the most important indications of the severity of this irritation), or until a large exudate is formed or the disease proves fatal. The mere act of vomiting, of course, increases the peritoneal pain and consequently greatly distresses the patients.

It need hardly be mentioned that vomiting, especially in the early stages, is reflex in character, and is due to irritation of the nerves of the peritoneum. In addition, the nerves of the stomach may be involved, as shown by the fact that occasionally drinking leads to retching or vomiting. At first the vomited material consists of whatever the stomach contains; later it becomes greenish, bile-colored, and slimy; the older writers on peritonitis called particular attention to the *vomitus herbaceus* of the disease. Occasionally the vomit is feculent; this constitutes the syndrome of so-called ileus paralyticus, the pathogenesis of which will be considered below.

Hiccup is mentioned here, for although it does not occur constantly in acute peritonitis, it is a frequent and very distressing symptom. The occurrence of hiccup does not necessarily prove that the nerves of the diaphragm are involved,—in other words, that there is inflammation of the diaphragmatic layer of the peritoneum,—for its appearance in any form of peritonitis is readily explained by the fact that the phrenic nerves send sensory fibers to the peritoneum (Henle, Schwalbe).

The symptoms due to the fact that the intestines are involved are important. The **stools** differ in the various forms of the disease, and also according to its course. In chronic and in acute circumscribed peritonitis no definite rules can be laid down as regards the stools, as they depend on a number of concomitant conditions which have nothing to do directly with the inflammation of the peritoneum. As a rule, however, acute diffuse peritonitis is accompanied by constipation, which is occasionally very obstinate and dates from the onset of the disease, lasting from five to ten days, even when no opium is given. Sometimes, it is true, the constipation is slight and the bowels act naturally about every other day. In one form of acute diffuse peritonitis— namely, in puerperal (septic) peritonitis—there is, as a rule, diarrhea. All observers agree on this point, and my own experience further bears out this statement; I have not infrequently seen 10 and even 15 and 17 motions a day in puerperal septic peritonitis. Similarly, diarrhea is common in the pneumococcal peritonitis of children.

In the section on the Anatomy of Peritonitis attention was called to the involvement of the intestinal wall in the morbid process. Most

authors are agreed that it accounts both for the peculiar character of the motions and also for the meteorism, to be described later, which so frequently occurs in inflammation of the peritoneum. The inflammatory edematous infiltration of the intestinal wall is regarded as the real cause of the constipation, as it produces a paretic condition of the muscular coat ; that this paresis occurs in acute diffuse peritonitis is undoubtedly true, but it does not seem to me to be by any means proved that the constipation is exclusively due to structural changes in the intestinal wall nor to the paretic condition of the muscular coat, which is said to depend on these changes. The facts that constipation begins with the onset of the acute inflammation of the peritoneum and that it is absent in many cases of chronic peritonitis seem to indicate that some other factors are responsible for its occurrence. In my opinion the vigorous stimulation of the inhibitory nerves starting from the inflamed peritoneum in acute cases is chiefly responsible for the initial constipation. The diarrhea that is so frequent in puerperal peritonitis has been explained in various ways, some of which seem to me extremely inadequate. Rokitansky believed that in puerperal peritonitis the peritoneum, not only of the small, but also of the large, intestine was inflamed, and that this accounted for paralysis of the colon, which thus became a wide, flaccid tube through which the soft contents of the small intestine can flow unhindered and unchanged. Other authors attempt to explain the diarrhea in puerperal peritonitis as due to anatomic lesions (catarrh and ulceration) of the intestinal mucosa. I wish to call special attention to another point—viz., that many cases of puerperal peritonitis accompanied by diarrhea are septic. Is it not rational to assume that in these cases the bacterial toxins have something to do with the diarrhea ? I believe that the appearance of diarrhea in septic puerperal peritonitis and in the pneumococcal form of children is connected with the action of the particular toxin formed by the corresponding pathogenic microbe (see p. 130).

Meteorism plays an important rôle, but is by no means constant. It is, of course, absent in the circumscribed and in the majority of the chronic forms of peritonitis ; on the other hand, it is a characteristic feature of the clinical picture of acute diffuse peritonitis, in which it is only exceptionally absent—for instance, in perforative peritonitis. Meteorism may become enormous, and the gigantic distention of the abdomen in acute peritonitis must be attributed much more to the meteorism that accompanies the inflammation than to the peritoneal exudate. The abdomen is markedly distended, particularly in the middle and in the upper portions, but the flanks may also be very greatly swollen ; the skin over the abdomen is smooth, shiny, and tense. The more flaccid the abdominal muscles, the greater the distention ; this explains the enormous distention in puerperal cases, where the abdominal muscles are greatly relaxed and offer little resistance. On the other hand, the powerful abdominal muscles may exert so much resistance to the gases within the abdomen that the shape of the abdomen is either not changed at all or only slightly altered. This condition is of great medical in-

terest, inasmuch as the dangerous consequences of marked meteorism (described on p. 137 together with its physical signs) become specially manifest under these circumstances, as the diaphragm is more forcibly and persistently displaced upward when the abdominal muscles are unyielding than in patients with flaccid abdominal walls, which give way and so relieve the intra-abdominal pressure. (For the pathogenesis of peritonitic meteorism the reader should refer to what has been said on p. 138.) To what was said there we may add that it is impossible at present to settle the question whether the alterations of the nerve plexus of the bowel-wall, described by Walbaum and Askanazy, are responsible for the meteorism.

The **paresis of the intestine** in acute diffuse peritonitis just mentioned is not necessarily present. The condition of intestinal peristalsis is as follows : Clinical experience shows that in many cases of acute diffuse peritonitis the bowels act normally, and that intestinal peristalsis, therefore, is normal. In puerperal peritonitis there may even be increased peristalsis. Here, however, it might be argued with some reason that the diarrhea is not due to structural changes in the intestine, which, in their turn, might produce increased peristalsis, but to other factors which act indirectly (see above). Clinical observation, however, and animal experiments performed by Grünbaum at my suggestion show that increased peristalsis may also occur in other forms of peritonitis and at the very onset of the disease. Intestinal paresis cannot, therefore, be considered a constant symptom of acute diffuse peritonitis. With these limitations the following statements may be made as regards paresis and paralysis of the bowel in peritonitis :

This condition may appear very early in the course of the disease, especially in perforative peritonitis, where it is most probably due to reflex inhibition of intestinal peristalsis. Paresis gives rise to meteorism, since gas continues to collect and accumulate in the bowel as it is not driven onward. In general, the paresis comes on more gradually, and in these cases Stokes's explanation—viz., that the bowel-wall is infiltrated with a serous exudate and that consequently its muscular activity is impaired—may apply. As soon, however, as a considerable quantity of gas accumulates in the intestine, the vicious circle is completed, for gaseous distention of the intestine leads to further impairment of its muscular powers and eventually to overdistention and paresis, or to definite paralysis. It is impossible to decide at present whether toxic paralysis of the bowel-wall plays an important rôle in these cases.

Complete paralysis, involving either a small or a large part of the intestine, produces a group of symptoms—ileus paralyticus—which is occasionally seen in acute peritonitis, and is characterized by complete cessation of peristalsis. In the sections on Occlusion and Paralysis of the Bowel this syndrome was considered at great length, and the reader should, therefore, refer to them for the details.

[If the abdomen is carefully listened to with a stethoscope for five minutes and no gurgle is heard during that time, it may safely be concluded that the whole intestine is paralyzed (Greig Smith).—ED.]

The **shape of the abdomen** varies greatly in the different forms of peritonitis. Some forms of peritonitis have no influence on the outline of the abdomen; this is true in the majority of the circumscribed adhesive forms and in strictly localized abscesses; some of the latter varieties of peritonitis may, however, indirectly modify the appearance of the abdomen—*i. e.*, by producing kinking, stenosis, or other lesions of the bowel, which ultimately alter the outline of the abdomen. Many of the chronic exudative forms produce the same appearance as ascites, while acute diffuse peritonitis produces the most marked effects as regards ascites and meteorism. In some of the cases of peritonitis the behavior of the abdominal muscles is of some importance; thus, occasionally, more especially in acute perforative peritonitis, they become rigid from reflex irritation, which is sometimes accompanied by a similar condition in the muscular coats of the intestine. As a result, the abdominal wall becomes flattened and firmly contracted; this may be so marked that the surface of the abdomen may become retracted below its normal level and boat-shaped; this condition may persist throughout the whole course of the disease until death, provided the fatal issue is not delayed for a considerable time after the perforation. This "défense musculaire" of French authors possibly plays a part in diminishing the strength of the peristalsis, and so the intensity of the pain. It appears that the sudden and violent irritation of the sensory nerves of the visceral and parietal layers of the peritoneum is responsible for this effect.

[Bishop[1] considers rigidity of the abdominal walls the earliest available sign of acute peritonitis. It is essentially an early sign, and does not persist. When considerable septic absorption has taken place, these muscles become paralyzed; it should be looked for before morphin has been given. It does not occur in the chronic forms of peritonitis, such as tuberculous peritonitis.—ED.]

Lastly, another symptom belonging to this group—viz., **painful micturition**—is frequently present in acute peritonitis. It is due to irritation of the serous covering of the urinary bladder. When this sign appears, micturition is very painful, owing to the traction exerted on the inflamed peritoneum by the contractions of the bladder. Sometimes voluntary micturition is impossible, as the detrusor vesicæ may become paretic in the same manner that the muscular coat of the bowel becomes paretic in this disease.

GROUP C.

General Symptoms.—In chronic peritonitis general symptoms are, as a rule, absent. Even in acute peritonitis general symptoms are not always present, and when they do occur, are not by any means constant in character. The appearance of general symptoms depends primarily on certain etiologic peculiarities, more particularly on the virulence of the bacteria. The anatomic fact that the peritoneum is

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[1] E. Stanmore Bishop, *Lancet*, 1903, vol. ii., p. 741.

inflamed does not alone determine the appearance of the general symptoms referable to this lesion.

The **temperature** varies so greatly in peritonitis that no general rules can be given as to its character. There is no definite type of fever in acute peritonitis, and the temperature presents greater variations even than in inflammation of the pleura ; this is probably due to the fact that the anatomic lesions which may accompany inflammation of the peritoneum and the different etiologic factors responsible for the inflammation vary so greatly in peritonitis. In order to avoid unnecessary repetition I shall confine my remarks here to a sketch of a few peculiarities of the temperature in peritonitis in general, and must refer the reader for detailed descriptions of the course of the fever to the sections dealing with the various forms of peritonitis.

In acute diffuse forms of peritonitis, which start with or without a chill, the temperature usually rises rapidly and may assume a continuous type, rarely exceeding 104° F., for a varying time ; according to the anatomic course of the disease, the temperature drops after a shorter or longer interval from the onset of the disease ; sometimes the temperature becomes irregular, and often oscillates rapidly just before the death of the patient—*i. e.*, during the agonal stage. Again, in other cases the temperature slowly rises from the onset of the inflammation.

Occasionally an initial rise of temperature is followed by a drop to normal or below ; this is sometimes seen in septic peritonitis when the disease involves the general peritoneal cavity.

These differences in the temperature-curves manifestly depend on the nature and virulence of the infection responsible for the inflammation, and also on the quantity of the poison that gets into the peritoneal tissues at one time. The toxins of the individual bacteria probably act differently as regards rise of temperature, but nothing definite is known about this. Madelung, Lennander, Helferich, Grazer, Krogius, and others insist upon the importance of the difference between the rectal and the axillary temperature in diffuse peritonitis. It often exceeds the normal (1° F.) difference, the rectal being 2° to 3.5° or 5.5° F. higher than the axillary. Thus the temperature may be considerably raised inside the body when the skin appears cool. The importance of this in diagnosis is apparent, and Krogius looks upon the low temperature of the skin in these cases as a symptom of collapse.

In perforative peritonitis, particularly when the peritoneal cavity is suddenly flooded with the contents of the bowel through a large perforation, as in perforation of a gastric or duodenal ulcer or in rupture of a large abscess, the temperature occasionally drops below normal at once (collapse temperature) and remains so until the death of the patient.

It is a remarkable fact that the quality of the exudate in no way influences the course of the temperature ; it was formerly thought that it did, but it is now recognized that in cases of diffuse septic peritonitis the temperature often does not rise above 100.4° F. It would, therefore, be a serious error to exclude the presence of a purulent exudate

from the absence of fever or from the fact that there is only a slight rise of temperature.

[The severity of the general peritonitis may be regarded as being inversely as the height of the temperature, so that in really serious cases the temperature may be subnormal (Barnard).[1]—ED.]

In circumscribed purulent peritonitis the temperature varies greatly. There may be initial fever, the temperature then gradually becoming apyretic, or there may be continuous fever throughout the whole course, or again there may be intermittent or remittent types of fever with marked chills.

In tuberculous peritonitis the character of the fever also varies, and, as a rule, a number of other factors influence the temperature (pulmonary tuberculosis, etc.).

These forms of peritonitis which run a slow and insidious course and ultimately lead to the formation of localized peritoneal adhesions are usually afebrile from start to finish.

The great importance of the **pulse** and the general condition of the circulatory system in peritonitis is obvious; in fact, the older physicians spoke of a distinct *pulsus abdominalis*. This character of the pulse, however, is present only in cases of acute peritonitis that run a special course. The characteristic features of the so-called *pulsus abdominalis* are that it is exceedingly rapid (120 to 140 beats or even, toward the end, 160 to 170 beats) and very small, thin, and easily compressible, though, as a rule, quite regular. The highest pulse-rates with the minimum of tension and volume are observed in septic peritonitis. But there is no parallelism between the temperature and the rapidity of the pulse-rate; the temperature may be only slightly raised, or may be normal or subnormal, while the pulse-rate is enormously increased. This want of proportion between the temperature and pulse, whether it appears at the onset or during the course of the disease is of the greatest importance from a clinical, and particularly from a prognostic, point of view, for it is almost invariably an omen of the gravest significance. Doubtless the cause of this circulatory disturbance is the action of bacterial toxins. Owing to the great absorptive power of the peritoneum, large quantities of toxins are quickly carried into the general circulation, where they exert their deleterious action. It is at present doubtful which part of the circulatory apparatus or its nerve-supply is attacked—whether muscles or nerves of the heart or whether the nerves of the blood-vessels themselves are damaged. Romberg and Paessler have shown experimentally that in rabbits the pneumococcus, the Bacillus pyocyaneus, and the Bacillus diphtheriæ act by paralyzing the vasomotor center in the medulla oblongata. This leads to diminution of blood-pressure and a redistribution, in consequence, of the blood. The vessels of the splanchnic area are dilated, while those of the brain, muscles, and skin are emptied. The heart itself is not directly affected, but suffers secondarily through imperfect blood-supply. Heineke supplemented these results by experiments on the cause of death in rabbits in

[1] H. L. Barnard, *Clinical Jour.*, September 30, 1903, vol. xxii., p. 377.

whom he produced perforative peritonitis. He found it due to paralysis of the vasomotor and respiratory centers in the medulla oblongata—that the former causes the circulatory disturbance, while the heart is not directly affected. This circulatory disturbance in cases of peritonitis resembled precisely that of the experiments of Romberg and Paessler, and shows that the paralysis of the bulbar centers is due to the absorption into the blood of bacterial products from the peritoneum.

[In some cases where operation shows that the peritoneum is full of pus the pulse-rate may be only 100. Barnard[1] explains this on the supposition that the stomata of the peritoneum are so efficiently blocked as to prevent toxic absorption.—Ed.]

In the slowly progressive forms of circumscribed or diffuse acute peritonitis unaccompanied by severe toxic symptoms the heart-beat and pulse-rate, as a rule, run parallel with the course of the fever; the pulse in such cases may be full, with good tension, and only moderately rapid. If the process takes an unfavorable turn, the pulse soon becomes more rapid, smaller, and of lower tension.

In cases of very chronic peritonitis and in subacute cases that run a very slow course special cardiovascular manifestations rarely develop.

The general constitutional condition and the appearance of the patient are in no way typical, either in the chronic or in the subacute forms of the disease. Occasionally the patients begin to emaciate and look very miserable, as a result of chronic suppuration, loss of appetite, and poor nourishment; but these signs are not peculiar to peritonitis, and, of course, occur in a large number of other conditions. In acute diffuse peritonitis, on the other hand, the clinical picture which rapidly develops after the onset of the inflammation is perfectly characteristic and typical.

As already stated elsewhere, in acute diffuse inflammation of the peritoneum the patient remains motionless in one position on account of the intense pain; respiration is, as a rule, rapid, shallow, and costal. The facial expression is that of great suffering, anxiety, and intense pain, and, in addition, shows evidence of the general collapse that the patient falls into; according to the course of the disease, the collapse develops rapidly or slowly in different cases. In the septic and perforative forms of peritonitis the great loss of vitality and collapse are particularly striking, and are shown by the following signs: the nose appears pointed, the eyes are sunken, the extremities cold, the heart's action weak, the pulse thready; there are cyanosis and, occasionally, cold perspiration. (The reader should refer to pp. 387–398 for other clinical details and for the pathogenesis of all these symptoms, which are the same as those seen in acute intestinal obstruction with strangulation.) For this reason, too, it is often difficult or impossible to make a differential diagnosis between acute diffuse peritonitis and acute intestinal obstruction. Only one difference in the pathogenesis may be particularly insisted on here—viz., that in collapse from strangulation importance should be chiefly attached to the "nervous reflex theory,"

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[1] Barnard, *loc. cit.*

whereas in peritonitis the "intoxication theory" should be considered the most important factor; this at least applies to a great majority of the cases of peritonitis, and probably to all the cases of septic peritonitis.

[Charters Symonds [1] has drawn special attention to "the period of repose" which occurs soon after the onset in nearly all cases of acute peritonitis; it is marked by a cessation of all acute symptoms, and the patient passes, unaided by sedatives, into a condition of repose in which there is relief from pain and sickness; the sense of comfort may be interpreted as evidence of improvement if the signs of grave abdominal mischief are neglected. This period of reaction is suitable for operation. —Ed.]

While the patient presents this distressing general clinical picture, the **sensorium,** as a rule, remains perfectly clear; the patients are, of course, often very markedly depressed, but all the intellectual faculties remain quite clear. It is only in rare cases that consciousness is impaired or that delirium, stupor, or coma supervenes. The psychic symptoms which occasionally occur must be referred more to the general toxemia than to anemia or edema of the brain, as these phenomena are most commonly seen in the grave septic forms of peritonitis (compare the paragraphs on this form of peritoneal inflammation).

The **urine** in acute diffuse peritonitis presents some special features which are in part due to the fever, when it is present, and in part to the circulatory disturbances connected with the fall of blood-pressure. The quantity of the urine is diminished, and there is a tendency to lithatic deposit; it occasionally contains nucleo-albumin and serum-albumin, and sometimes acetone and diacetic acid. The most important change in the urine, however, is the presence of indican, the quantity of which may be enormous; there is probably no other condition, with the exception, possibly, of acute intestinal obstruction, in which the amounts of indican in the urine are so large. I consider the excretion of large quantities of indican to be one of the most constant signs of acute diffuse peritonitis; in fact, I should always hesitate about diagnosing this condition unless there were greatly increased quantities of indican in the urine. I remember only a single case in which indicanuria was absent in diffuse purulent peritonitis; postmortem the pancreas showed marked induration and atrophy. (The reader should refer to page 166 for the pathogenesis of indicanuria.)

Whereas indicanuria is a regular and constant accompaniment of acute diffuse peritonitis, it is not by any means of other forms of the disease; in chronic circumscribed adhesive peritonitis indicanuria is completely absent, and in all the other forms the presence or absence of abnormally large quantities of indican in the urine depends on the functional condition of the intestine in each individual case; the perverted function in these cases may, of course, depend either on the peritonitic process or on other causes; in either case indicanuria may occasionally appear.

[1] Charters Symonds, *Brit. Med. Jour.*, 1899, vol. i., p. 520.

[Lennander[1] points out that the urine may contain the micro-organisms which are responsible for the peritonitis, and that they may be present within twenty-four hours of the onset.

Leukocytosis occurs, provided the patient's resistance is not too much reduced, but in very severe forms of peritonitis there may not only be no leukocytosis, but a diminution, or leukopenia. In 16 cases of acute peritonitis the maximum was 46,000, the average 18,875, and the minimum 4400 (Da Costa[2]). The leukocytosis is the ordinary polymorphonuclear, neutrophile form.—Ed.]

Lastly, a certain number of complications which are occasionally seen in acute diffuse peritonitis must be mentioned—viz., rapid splenic enlargement, pleurisy, and endocarditis. The occurrence of the two last complications can readily be understood in the light of the ease with which an infection can pass through the diaphragm *via* the lymph-channels. Von Recklinghausen, it will be remembered, was the first to call attention to this possibility. The combination of pleurisy and peritonitis is not, of course, always explained in this way, for the two conditions may be concomitant and may both be due to simultaneous affection of the pleura and the peritoneum by the same hemic infection. The latter possibility must be borne in mind particularly in those cases of peritonitis in which the spleen, being also infected, becomes palpably enlarged.

Cases of chronic circumscribed peritonitis may occasionally run their course without producing any symptoms whatsoever. When the entire process is confined to the formation of slight inflammatory adhesions over solid organs (the liver, spleen, uterus), around old hernial sacs, and in a number of other situations, there may be no functional disturbance to point to the existence of peritonitis. On the other hand, these forms of inflammation of the peritoneum may occasionally indirectly produce the most serious consequences, such as intestinal obstruction (this part of the subject is dealt with in a special section).

ACUTE DIFFUSE PERITONITIS.

ATTENTION has already been called to the fact that in practice it is necessary to divide peritonitis into several categories. It is, however, impossible to base this classification on any scientific principle, such as the morbid anatomy or etiology of these classes, and it must be arranged from the practical clinical point of view. In this section the clinical pictures presented by the following forms of acute diffuse peritonitis will first be described: (*a*) *Peritonitis acuta diffusa sine perforatione;* (*b*) *peritonitis acuta diffusa e perforatione;* (*c*) *peritonitis septica* (*sepsis peritonealis*); (*d*) *peritonitis puerperalis;* (*e*) *peritonitis fibrinosa-purulenta progrediens.*

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[1] Lennander, *Deutsch. Zeitschr. f. Chir.*, 1902, vol. lxiii.
[2] Da Costa, *Chemical Hematology*, p. 408.

ACUTE DIFFUSE PERITONITIS NOT DUE TO PERFORATION (Peritonitis Acuta Diffusa Sine Perforatione).

The special description of this form of peritonitis need only be short and limited to a few general remarks, for the general clinical picture already given on pages 764–779 is essentially a description of this form of peritonitis.

Etiologically speaking, all those forms of peritonitis which are due to the gradual invasion of the peritoneum by inflammatory agents and not to sudden deluging of the peritoneum by these agents belong to this category. The infective agents may get into the peritoneum from some neighboring organ *via* the lymphatic or blood-vessels, or they may invade the peritoneum gradually—that is, a few at a time, or more commonly may find a suitable nidus for their growth somewhere in the peritoneum and gradually multiply and spread from this primary focus. All these various clinical possibilities have been described in the section on the Special Clinical Etiology of Peritonitis.

The leading symptoms appear in each case with stereotyped regularity. The most important symptom is the peculiar pain of peritonitis, which contrasts with the initial pain of perforative peritonitis in not beginning with full severity, but by gradually increasing in violence; at the start the pain may be strictly localized in the position where the peritonitis begins; later it spreads diffusely over the abdomen. Exudation always occurs, although sometimes it may be fibrinous in character or so scanty that it either escapes detection or cannot be recognized with absolute certainty; but even in such cases the other symptoms, and especially the character of the pain, will clear up the diagnosis. The more acute the onset of the peritonitis,—*i. e.*, the more rapidly the anatomic process spreads diffusely over large portions of the peritoneum, —the more constant is vomiting. When the spread of the inflammation is gradual, vomiting may be absent, whatever the cause of the peritonitis. Thus I have seen vomiting absent in peritonitis due to typhoid, appendicitis, carcinoma of the stomach, in puerperal infection, and injury. Constipation is the rule, but in cases of peritonitis coming on in the course of typhoid fever diarrhea may persist when it was present before the inflammation of the peritoneum set in. Besides puerperal peritonitis, which is often accompanied by diarrhea and will be specially dealt with below, peritonitis supervening on tuberculous or other forms of intestinal ulceration may be associated with diarrhea, and I have met with it in traumatic peritonitis.

Meteorism, paresis of the bowel, which may even lead to the symptom-complex of paralytic intestinal obstruction, and, lastly, painful micturition may all be present and produce the clinical picture which has been described in the general part. The same applies to the action of the heart and the general constitutional condition which have already been described. The character of the temperature-chart, however, in this form of diffuse peritonitis requires a special description.

It is only in exceptional cases that there is no rise of temperature

in acute diffuse peritonitis; it is remarkable that this absence of pyrexia seems, from my personal observations, to be specially frequent in cases due to injury. As a general rule, fever is present either with or without an initial chill; the subsequent course of the fever is subject to the greatest variations in different cases. The type of fever may be continuous, remittent, or even intermittent, and in some cases periods of apyrexia are followed by a second elevation of the temperature. The following case may serve to illustrate these conditions: Onset of the disease with a chill and fever up to 104.2° F.; on the second day, fever of from 100.4° to 102.2° F.; then no fever for eighteen consecutive days, in spite of the presence of excessive pain and the formation of an exudate; then for ten days moderate fever; then again for the next nine days and up to the time the patient was discharged, apparently cured, no more fever. The absolute height of the temperature rarely exceeds 104° F.; even in rapidly fatal cases, in which the duration of the disease does not last more than six or eight days, the temperature does not necessarily rise above 103° F. In cases that terminate favorably the temperature drops gradually, although exacerbations may occur at any time.

These considerable variations in the temperature-curve must clearly be due to differences in the nature of the inflammatory agent (bacterial toxins) in different cases. At the present time, however, it is quite impossible to make any definite statement as to the rôle of these different factors in modifying the temperature. It must be specially insisted upon, however, that purulent exudates, even when considerable, need not raise the temperature above 101.4° F. throughout the whole course of the disease. When purulent peritonitis runs a slow course, particularly when there is a sacculated collection of pus between several loops of intestine or in some other part of the abdomen, there may be fever of a marked remittent or intermittent type,—so-called hectic fever,—which gradually produces great emaciation and exhaustion and thus slowly causes the death of the patient, provided, of course, that spontaneous perforation does not occur or that the patient is not operated upon and a different termination thus brought about.

Course, Duration, Prognosis.—The duration of acute diffuse peritonitis—the perforative and the septic forms are not included—varies greatly. In the mildest cases which recover the duration is never less than two or three weeks; more severe cases last four to six weeks. When operative interference is undertaken, the duration cannot be predicted, and may, owing to variations in the course of the illness, extend to months before the patient is able to attend to business; even then a number of sequelæ, especially adhesions between different loops of intestine, are apt to persist.

In cases that run an unfavorable course death rarely occurs before the end of the first week—it is only severe septic or perforative cases that terminate fatally within the first forty-eight hours. When the disease gradually and steadily progresses from bad to worse, death, as a rule, ensues in the second week; when the course fluctuates, death gen-

erally occurs from the third to the sixth week; sometimes, however, the course of the disease is prolonged for many months, and there are many ups and downs in the patient's condition before death finally closes the distressing scene.

The possible anatomic variations in the course of the disease have already been described in the section on the Pathologic Anatomy of Peritonitis; the different clinical pictures and the course of the disease correspond to the differences in the anatomic basis. When improvement begins, the symptoms all become less severe: the febrile symptoms gradually disappear; the exudate is absorbed; finally, tenderness on pressure and spontaneous pain disappear. It is not always easy to make out whether complete absorption of the exudate has actually occurred; adhesions may persist and may not produce clinical symptoms for a long time after the patient apparently recovers from the acute attack of peritonitis.

Purulent exudates of small bulk may also occasionally undergo absorption, with disappearance of all the symptoms produced by them. When the purulent exudates are very large, they either spontaneously discharge through an opening situated in one of several different parts of the abdomen, or when not surgically opened, may produce a septic fever of an irregular type which persists until death ensues from exhaustion. Even when the pus is spontaneously evacuated or is let out by a surgical operation, a variety of dangerous sequelæ may develop— viz., adhesions between different loops of intestine, perforation into dangerous positions, persistence of sacculated circumscribed abscess cavities, extension of the suppurative process to veins.

Acute diffuse peritonitis is always a serious and, as a rule, a very grave condition. In the prognosis the local and general symptoms must be taken into account. When the exudate is abundant, the prognosis is not necessarily bad; when the exudate is purulent or sanious, the prognosis is worse.

[The formation of pus is a comparatively favorable sign in diffuse peritonitis, and cases in which it occurs are not so fatal as those in which serum only is poured out, there being no diapedesis of leukocytes either because the process is too rapid or because the patient fails to react. In these cases there is slightly turbid, blood-stained serum, without any fibrin on the surface of the intestines, which is roughened and eroded; the stomata are not occluded, and the endothelium of the peritoneum is removed so that the most rapid absorption of toxins takes place from the peritoneum (Barnard).[1] Durham's[2] experimental work, showing that acute peritoneal infection may prove fatal with very little evidence of ordinary peritonitis, has already been referred to (*vide* p. 730.—Ed.]

In the latter case spontaneous recovery can never be expected; in the former, only when the amount of purulent exudate is very small. When the accumulation of pus is considerable, the only hope of recovery

[1] H. L. Barnard, *Clinical Jour.*, September 30, 1903, vol. xxii., p. 378.
[2] H. E. Durham, *Jour. Path. and Bact.*, vol. iv., p. 362.

lies in spontaneous discharge of the pus into some hollow viscus or through the skin, or in operative interference; under these conditions the life of the patient may occasionally, though rarely, be saved, and even with this most favorable result the patient's life remains in danger for a long time afterward from the adhesions which usually form, etc. Marked meteorism, as well as paresis of the muscular coat of the bowel, is a direct danger to life.

The general symptoms are still more important in making a prognosis. The temperature itself has not much bearing on the prognosis, and it would be a serious mistake to give a bad prognosis because the onset was acute and ushered in with high fever, or, conversely, to give a favorable prognosis because the onset of the disease was slow and unaccompanied by high fever. The condition of the circulatory system is of the greatest importance. Early and severe collapse, with rapid loss of strength and the signs of a failing heart, should arouse the worst fears, although even this condition may suddenly change and improvement occur after all. These severe general symptoms must be attributed to the grave toxic character of the infection; it is toxemia which especially determines the course, prognosis, and even fatal issue of the attack.

PERFORATIVE PERITONITIS (Peritonitis Acuta Diffusa e Perforatione).

In perforative peritonitis the entrance of the infective agent into the peritoneum does not occur gradually, but the contents of an organ suddenly pass into the peritoneal cavity, either in small or in large quantities. This can occur only when there is a solution of continuity in the wall of some organ—*i. e.*, when it becomes perforated. It is true that the term "perforative peritonitis" is usually taken to mean that perforation of an air-containing organ has occurred, such as the stomach or the intestine; in other words, the term is frequently used to describe pneumoperitonitis. This is not correct, however, for the term "perforative peritonitis" is equally applicable to peritonitis following rupture of an abscess or the passage of the contents of the gall-bladder or the urinary bladder into the peritoneum. Properly speaking, peritonitis due to laparotomy also belongs to this category.

Etiology.—In the description of the general pathogenesis of peritonitis the general factors giving rise to peritonitis when an organ is opened or an abscess bursts and their contents suddenly pass into the peritoneal cavity have been dealt with. In order to avoid unnecessary repetition, the reader should refer to that section.

The mechanism of perforation of certain organs into the peritoneal cavity and of the resulting peritonitis has been described several times elsewhere—viz., in the paragraphs on traumatic and spontaneous rupture of the intestine, on the special clinical etiology of peritonitis, of appendicitis, and of perityphlitis. Under these headings the various methods by which peritonitis may arise in certain diseases of the intestine, stomach, liver, bile-ducts, pancreas, spleen, kidneys, and urinary bladder were described.

It is only necessary to add that perforative peritonitis may also occur as the result of rupture of abscesses, whether extraperitoneal (paranephritic, parametric, psoas abscess, etc.) or intraperitoneal (this applies particularly to suppurative perityphlitis), or when the pus tracks through the diaphragm from the pleura or the lungs.

Perforation is most frequent in the vermiform appendix and the lower part of the ileum (typhoid ulcer); then in other parts of the intestine, and less often in the stomach. Rupture of abscesses followed by diffuse peritonitis is again most frequent in perityphlitic suppuration. Perforative peritonitis is comparatively rarely due to disease of the solid abdominal organs.

Anatomy.—Some time must, of course, elapse before the anatomic changes of peritonitis can develop. All signs of peritonitis may, therefore, be absent in cases in which death from collapse occurs very soon after the onset of the disease—*i. e.*, after eight to sixteen hours. In such cases gastric or intestinal contents may be found in the peritoneal cavity, which is quite free from any signs of inflammation.

When an abscess or some organ that does not contain air ruptures into the peritoneum, the resulting inflammation is almost invariably putrid or purulent, and presents all the anatomic characters that have already been described in the general account of the pathologic anatomy of peritonitis. When, however, air-containing viscera, such as the stomach or intestine, rupture into the peritoneal cavity, a few special points require description.

When the abdomen is opened, gas frequently escapes with a hissing sound. When the gas is free from odor, perforation of the stomach is probable (Czerny). The perforation is often large and clearly visible, particularly in gastric ulcer. In other cases the perforation is so small and insignificant that it requires most careful search; this, for instance, is a frequent event in gangrenous perforation following volvulus, particularly when the opening in the intestine is subsequently closed by a fibrinous exudate or becomes adherent to other parts. Great care must also be taken not to confuse artificial tears, which are very easily made in the bowel when its walls are friable, with the perforation that occurred during life and caused the death of the patient.

The quantity of the exudate varies greatly, and largely depends on the duration of the disease, while the latter, in its turn, is dependent on the nature of the affection; the more toxic the cause, the shorter, as a rule, the course of the disease and the smaller the exudate. The character of the exudate varies: in perforation of the bowel followed by pneumoperitonitis it is often sanious and has a feculent odor; it may also contain solid particles that are derived from the part of the bowel in which the perforation occurred; the so-called fecal concretions occasionally found in perforation of the appendix are of special interest.

The extent and degree of the inflammation may be very limited when death occurs rapidly. When the inflammation is well advanced, the bowel will be covered with a discolored fibrinous exudate; the peritoneum itself has a greenish tint, is friable, and is at times necrotic

and readily torn into shreds which cling to the affected portion of the bowel. In the solid organs, particularly on the convex surface of the liver, on either side of the suspensory ligament, there may be greenish-grayish areas of discoloration. These discolorations are in all probability due to the chemic action of the gases that are liberated in the peritoneal cavity during this process. The distention of the bowels varies : it may become very marked, even meteoristic, when the perforation, originally small, is subsequently closed by fibrous exudate.

Clinical Features.—In considering the clinical aspects of perforative peritonitis, a distinction must be drawn between cases due to perforation of air-containing viscera and cases due to perforation of viscera which do not contain air. In the former category two groups of symptoms must be distinguished—viz. (1) Those caused by the rupture of the stomach and intestine *per se* and by the resulting pneumatosis ; (2) those caused by the inflammation of the peritoneum itself. The former symptoms correspond to those described on p. 679, and the latter are the symptoms of ordinary peritonitis described in the preceding section. The combination of these two groups of symptoms makes up the clinical picture of what is commonly called perforative peritonitis.

In perforation of the stomach or the intestine, when their contents enter the peritoneal cavity suddenly, the resulting symptoms also come on suddenly. Occasionally there are no prodromal symptoms whatever—the patient may be perfectly well up to the moment when the perforation occurs (when this is due to an external injury), or at least the patient may appear to be perfectly well (for instance, when the underlying process which eventually leads to perforation of the stomach runs a latent course, as is sometimes the case in gastric or duodenal ulcer and in perforation of the vermiform appendix). In other cases symptoms precede the development of perforative peritonitis which belong to the primary disease responsible for the perforation of the viscus—as, for instance, in the lesions of the stomach or intestine just mentioned or in typhoid fever, carcinoma, volvulus, or any of the numerous processes which may lead to ulceration of the gastro-intestinal tract.

The occurrence of perforation is marked by the peritoneal pain due to the tearing of the wall of the bowel, and is followed by collapse ; the abdomen may become retracted, boat-shaped, and rigid, or it may be distended and present the physical signs of gas in the free peritoneal cavity with more or less certainty. Death may ensue while this clinical picture is presented, and occasionally this occurs within a few hours after the onset of the disease. At the autopsy there may be no anatomic evidence of peritonitis, death being due to shock. Logically speaking, therefore, it is hardly justifiable to say that these are the clinical manifestations of perforative peritonitis, for, as a matter of fact, they are merely those of perforation. The symptoms directly due to the inflammation of the peritoneum come on gradually in the course of four to twelve hours. At this stage the abdomen becomes

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generally painful; the pain is very severe; the presence of fluid exudate can often be made out; vomiting now occurs in cases in which it was not already present; any retraction of the abdomen previously present disappears, and the abdomen becomes distended and meteoristic instead—it is possible, however, in cases in which death occurs within the first twenty-four to thirty-six hours after the onset of the first symptoms, for the abdomen to remain rigid and retracted until death, even when inflammation of the peritoneum is fully developed; the urine contains indican.

Some of the older observers noticed that occasionally in peritonitis due to perforation of the stomach vomiting was entirely absent when the tear in the stomach-wall was large. Traube explains this on the assumption that when the stomach contracts, its contents escape in the direction of least resistance—*i. e.*, through the perforation into the peritoneal cavity. Ebstein further has shown that vomiting may be absent in perforation of the stomach, or that it may stop if it was present before, when the perforation occurs into the lesser sac of the peritoneum (bursa omentalis) instead of into the free peritoneal cavity. When the perforation in the stomach-wall becomes closed in any way, vomiting may be present as usual.

[In perforated gastric ulcer Fenwick found vomiting in 29 per cent., Finney in 40 per cent., and English[1] in 31 out of 42 cases, or 74 per cent.—Ed.]

The condition of advanced and serious collapse, which appears soon after the perforation, continues unchanged, but is now due to absorption of bacteria or their toxins by the peritoneum, causing toxic poisoning. The pulse becomes very rapid—up to 160—and thready, and the respiration purely costal in type. The temperature varies in some cases—as, for instance, after perforation of a gastric or duodenal ulcer: there may be no rise of temperature at all, even though postmortem a purulent exudate is found in the peritoneal cavity; in other cases the temperature may rise rapidly and may be accompanied by rigors; in traumatic perforative peritonitis I have seen the temperature go as high as 104° F. and remain at this level for several days, only falling when the patient became moribund. When perforation occurs in the course of a febrile disease, the temperature may fall temporarily, possibly for a few hours, as a result of the perforation, but it has a tendency to rise again in a short time. The temperature may also rise directly the perforation occurs, and be accompanied by a chill (compare p. 680). Occasionally, too, the fall of the temperature, which is often seen when collapse occurs, may not take place, so that the temperature-chart does not point to the existence of peritonitis and does not in any way suggest the probability of a perforation.

The prognosis of peritonitis is dealt with in another portion of this work; the course of the disease, as has already been said, is, as a rule, rapid and unfavorable; death generally supervenes within a few days, sometimes even within a few hours. There can be no doubt, at the

[1] T. C. English, *Medico-Chir. Trans.*, vol. lxxxvii.

same time, that recovery does occasionally occur, but, unfortunately, is very rare ; recovery may occur either spontaneously or as the result of operative interference. Leube, who has had a very wide experience with this disease, reports recovery in only two cases of peritonitis following perforation of a gastric ulcer which were not operated upon. The consensus of opinion is that this rare termination in recovery occurs only in those cases in which the stomach is empty when the perforation occurs. Surgical interference in cases of perforation of the stomach or duodenum and jejunum is of much more favorable prognosis when the traumatic or spontaneous tear takes place on an empty stomach ; for microscopic and cultural investigations prove that the mucous membranes of these parts are sterile after six to twelve hours of starvation. On the other hand, the prognosis of perforation of the intestine lower down is uniformly bad. Even under such unfavorable conditions as perforation of a typhoid ulcer recovery may occasionally occur ; this has been established by a number of reliable observers, among them Traube. The smaller the amount of gas that escapes, the smaller the opening and the more rapid the agglutination of the edges of the perforation by fibrin, the better the prospect of recovery. In the cases that recover the course of the disease is very tedious, and the patients are ill for weeks or even months.

The diagnosis of peritonitis with escape of gas into the peritoneal cavity can often be made with certainty ; in cases where there is perforation of a solid organ, the diagnosis can be made only from the rapid appearance of the symptoms of peritonitis, on the one hand, and the presence of some local lesion, on the other hand, which is capable of leading to a rupture of the affected organ.

It is always important to make out the part of the gastro-intestinal tract in which the perforation has occurred. As a rule, the history of the case is a valuable guide (gastric ulcer, typhoid, etc.), but where this information is absent or unreliable and it is necessary to have recourse, nevertheless, to surgical interference,—as, for instance, in a case of peritonitis perforativa acutissima,—it is useful to bear in mind some points which, when the abdomen is opened, may help the surgeon in rapidly discovering the exact position of the perforation. Attention has already been called to the significance of absence of vomiting in cases of gastric perforation. Another point in favor of this diagnosis is the absence of any odor (Czerny), for the gases escaping from the stomach are usually without odor. When the fluid contained in the peritoneal cavity is bile-stained and there is, in addition, gas in the peritoneal cavity, the diagnosis of perforation of a duodenal ulcer seems probable. [Testing the exudate with litmus-paper may be of some use, an acid reaction pointing to perforation of the stomach or duodenum.—ED.] There is no clinical means of deciding whether the perforation is in the large or in the small intestine ; the only possible available method would be to count the bacteria in the exudate and to determine their species ; this might sometimes give some slight indication, but the results of such an examination could never be absolutely diagnostic ; it is, moreover,

impossible to carry this out quickly enough to be of any practical value, for the exudate is only obtained at the time of the operation.

SEPTIC PERITONITIS.

From a practical clinical standpoint we are justified in isolating a special form of peritonitis—viz., "septic peritonitis." It is necessary to define clearly the conception which it is intended to convey by this term. Many authors regard as septic all forms of peritonitis that are caused by bacteria. This classification is incorrect, for nearly all forms of peritonitis must then be included under the category of septic peritonitis, since very few cases seen in ordinary practice are caused by purely chemic or mechanical irritation of the peritoneum. It is also impracticable to classify septic peritonitis according to the anatomic appearances presented, for they vary greatly in different cases. The clinical point of origin of the inflammation of the peritoneum may also vary greatly, and while it is true that the signs of septic intoxication appear most frequently in the puerperal form of peritonitis, it may also appear in a number of other conditions that lead to the development of septic peritonitis.

I regard as septic peritonitis all those cases in which a characteristic syndrome is presented as a result of the intoxication of the nervous system, and in which the general constitutional condition of the patient is specially affected. As this differentiation is exclusively based on clinical observation, it is often difficult to determine where ordinary acute diffuse peritonitis ends and the septic form begins; the typical cases of the septic form are so clearly defined, however, that we are justified in describing it separately as a distinct clinical entity.

The characteristic feature of septic peritonitis is that, in addition, to the ordinary clinical signs of diffuse peritonitis, which have been described under the heading of the general symptomatology of peritonitis, certain other phenomena appear which must be attributed to the action of some chemic poison on the general organism and in particular on the nervous system; the general signs of diffuse peritonitis in these cases may either be well marked or may only be indicated; the latter signs, moreover, it must be clearly understood, are caused directly or indirectly by anatomic changes in the peritoneum, whereas the septic signs are due to toxemia.

It is beyond the scope of this work to enter into the whole subject of septic intoxication or to review all the published work on this subject; and, as a matter of fact, the last word has by no means been said on this question, and investigations into the nature of septic intoxication are not concluded. Bumm differentiates two distinct forms of so-called septic peritonitis according to differences in their etiology— viz., the one produced by streptococcic poison, the other caused by putrid poison : the former is septic peritonitis in the strict sense; the latter should be spoken of as putrid peritonitis.

As pointed out above, the Streptococcus pyogenes must be considered

the most common cause of diffuse acute peritonitis in general ; the same micro-organism also produces the most severe varieties of the septic form of the disease. In pure cases of the latter kind there is occasionally a pure culture of the streptococcus covering the whole peritoneum, particularly when the inflammatory anatomic changes are only slightly developed. It may, therefore, be concluded that this streptococcus furnishes the septic poison. As the same microbe is, however, also found in other forms of peritonitis without septic symptoms, it must be concluded that the septic syndrome develops only in cases where the bacterial poison produced by the streptococcus is manufactured in highly virulent and exceptionally large quantities, and is also very rapidly absorbed in large quantities into the general circulation. In some cases, in fact, the toxemia is so intense that death may supervene from this purely chemic intoxication before the inflammatory changes in the peritoneum, which would naturally result, have had time to develop. These are cases of peritoneal sepsis proper in which highly virulent toxins, absorbed with great rapidity from the peritoneal cavity, bring about a general deleterious effect (chiefly through impairment of the vasomotor center in the medulla) before even the local effect (peritonitis) has completely developed. Peritoneal sepsis cannot be separated from septic peritonitis, but must be regarded as an anatomically abortive form of the latter.

In order to explain the fact that the chemic action of the bacterial poisons is often so rapid and so violent, several facts are put forward, borne out by numerous recent experiments on peritonitis. First and foremost is the degree of virulence of the particular bacterium concerned.

Further, it must probably be assumed that paralysis of the peritoneum occurs as the primary event whenever large numbers of bacteria suddenly invade the peritoneal cavity (as in perforation or from the puerperal uterus). The reactive inflammatory change of the serosa, consisting chiefly in the formation of a protective wall of fibrin, does not in these cases occur with sufficient rapidity to afford adequate protection against the absorption of the bacterial poisons. The poisons are consequently absorbed with great rapidity into the general circulation, and in this way the septic symptom-complex is produced. When the anatomic changes of peritonitis develop, the absorption of the bacterial poisons occurs more slowly, but the quantity absorbed may still be sufficiently large to produce definite clinical symptoms of septic intoxication in addition to the local signs of peritonitis. Lastly, in a third group of cases, the toxic effects may be entirely absent from the clinical picture.

It is conceivable that the physiologic injury to the peritoneum just referred to may be produced by factors other than the bacterial poisons ; some physical factors may produce the same effect—*e. g.*, when aseptic sponges are brought in contact with the peritoneum during the course of an operation ; drying the peritoneum by exposure to the air, and, lastly, the action of antiseptic fluids which damage the peritoneum.

In addition to septic forms of peritonitis produced by streptococci,— and possibly other forms of bacteria, especially staphylococci ; Seitz also

describes toxemia due to the Bacterium coli,—another form which Bumm has appropriately called putrid peritonitis must be recognized. The existence of this form of peritoneal inflammation is very soon recognized—by the sense of smell, for the peritoneal cavity emits an odor of decomposition due to the presence of offensive putrefactive bodies.

This form is produced by the invasion of putrefactive bacteria of various kinds, which may be cocci or bacilli (Bumm). They find a suitable nidus in dead or necrosing tissues or in portions of the peritoneum that are separated from living tissue, where they produce decomposition and putrefaction and lead to the formation of putrefactive alkaloids. The latter are absorbed into the general circulation and produce the symptoms of putrid intoxication. Quite recently the anaërobic bacteria have been considered important in connection with the origin of putrid septic peritonitis.

Clinically, these processes occur under a variety of conditions. In the puerperal state putrid decomposition may occur in necrotic fragments of the uterine mucous membrane, cervix, vulva, in the placenta and decidua ; while after laparotomies putrid decomposition may supervene in the blood or in transudates in the wound itself ; and in perforation of the intestine, provided the perforation still remains in free communication with the inflammatory exudate.

Anatomy.—The anatomic changes in septic peritonitis are by no means constant. In the most severe cases, where death occurs with fulminating rapidity, the anatomic lesions are often very slight ; occasionally, in fact, nothing more is found at the autopsy than hyperemia of the peritoneum—*i. e.*, merely the anatomic evidence of the commencement of inflammation. Death in these cases occurs so rapidly that the advanced changes of peritoneal inflammation have not time to develop. In other cases the layers of the peritoneum are reddened, and, in addition, the peritoneal cavity contains a small amount of greenish-yellow exudate, amounting to some 300 to 400 cubic centimeters, and a few flakes of fibrin loosely adherent to the inflamed surface of the peritoneum ; there are no firm adhesions, but there may occasionally be delicate adhesions between different loops of intestine. As a rule, it is true, the ordinary appearances of acute purulent peritonitis are present, even in cases that ran a short course not exceeding sixty to seventy-two hours ; here, in addition to the anatomic changes proper to this form of peritonitis, there is a purulent exudate, which, in the putrid forms, may be sanious and offensive.

In addition to these lesions in the peritoneum, other changes are found in distant organs, which are the result of the general septicemia ; these conditions will merely be mentioned, and no attempt to describe them in detail will be made here. The mucous membrane of the stomach and of the intestine may be in a condition of catarrhal swelling ; ulceration, as has already been mentioned above, is rare, at least so far as my personal experience goes. The spleen is enlarged. The well-known parenchymatous changes are seen in the heart muscle, the liver, and the kidneys. In addition, pleurisy and pericarditis, bron-

chitis, and articular and periarticular suppuration are common—in short, the complete anatomic appearances of septicemia.

Clinical Features.—As an illustration of the clinical appearances of septic peritonitis the following sketch, taken from the records of Traube's clinic from the year 1863–64, may be given (in H. Fischer's report of puerperal affections) :

The patient was a cook twenty-eight years old ; on March 23d, simple and easy labor ; shortly after delivery a chill and violent abdominal pain. On the evening of the twenty-fourth her condition was as follows : the patient is in very depressed condition ; there are dark-blue rings under the eyes ; the facies betrays great suffering. The sensorium is free. The woman suffers from constant nausea, but does not vomit. The temperature, 104.2° F.; the pulse, 172; the respiration, 44. The radial pulse is small, but of high tension. The abdomen is enormously distended and very tender to the slightest touch. The percussion-note over the abdomen is meteoristic ; in the right iliac region, however, there is slight dulness. The patient is constipated. The lochia are scanty, thin, and discolored. Otherwise nothing abnormal can be discovered in the external or internal genital organs. There is a slight degree of albuminuria. In the night of the twenty-fourth to the twenty-fifth, constant vomiting of green material. Constant restless delirium. On the morning of the twenty-fifth extreme collapse, extremities cold, pallor of the face and lips, and universal cyanosis. Pulse barely perceptible ; the abdomen remains very tender on pressure, but the patient no longer complains of spontaneous pain. Constant vomiting. No bowel motions. Death at noon. Autopsy : A considerable amount of purulent exudate is found in the abdominal cavity. Small collections of pus in the broad ligaments. The whole tissue of the neck of the uterus is edematous and covered with numerous yellowish spots ; the mucous membrane of the uterus is breaking down and is covered with a very thin, cloudy fluid, and here and there with tough masses that can be removed only with difficulty. The attachment of the placenta looks normal.

The history and account of this case present the main clinical features that appear in addition to the ordinary syndrome of peritonitis in cases which justify the diagnosis of septic infection ; the alarming change in the general constitutional condition of the patient and, more important still, the cerebral disturbance must both be attributed to septic poisoning.

In cases of peritonitis that run an ordinary course the sensorium remains free until death, whereas in the septic form of the disease the sensorium is frequently involved. The clinical manifestations of cerebral disturbance may take different forms. Sometimes the patients become delirious, confused, and lose all sense of time and place, get completely out of touch with their environment, and may even have hallucinations. As a rule, the delirium is of the quiet type ; exceptionally, however, it may become maniacal ; in the latter event it is often difficult to decide whether the delirium is due to the toxemia, secondary meningitis, or is only a manifestation of a concomitant puerperal mania. In other cases the patients are merely restless, toss about in bed, and display dissatisfaction and impatience, in marked contrast to their ordinary mental attitude and behavior. In rare instances the women relapse into a condition of somnolence or even of sopor. In many cases, however, the patients become indifferent and display a marked want of interest in what is going on ; it is remarkable that occasionally the violent pain disappears or becomes greatly relieved as soon as this condition of apathy develops. There is often a marked contrast between the com-

plete subjective euphoria of these patients and their severe and alarming objective state.

In every form of acute diffuse peritonitis there is always some degree of collapse, and in the septic form the general prostration is extraordinarily marked. The patient's general condition indicates intense collapse; the bodily movements are wanting in strength and energy, and are tremulous and hesitating; the position of the patients in the bed is also characteristic, for they sink down to the lowest part, where they lie, so to say, in a heap; the extremities are livid and cold; the face is pinched and tired, with blue furrows under the eyes; the pulse is wretchedly weak and very rapid. The face may at the same time be of a peculiar yellowish, subicteric hue—*i. e.*, of a shade that is peculiar to grave septic infections. The coldness of the extremities, of the face, the ears, and the thready character of the pulse—in short, all the severe symptoms—must apparently not be attributed, as hitherto assumed, to alterations in the action of the heart muscle, due to the effect of the septic poison, but, according to the latest experiments, to toxic vasomotor paralysis.

The following may be added in regard to the temperature in septic peritonitis. (See also the remarks on the temperature in peritonitis in general on page 775.) The fever usually begins with a chill, which is often violent; in many cases the chill is replaced by one or several spells of "goose-skin," but occasionally both conditions may be absent. The temperature usually rises to a considerable height—as high as 104°, even 106° and 108° F. As a rule, the character of the temperature-chart is at first that of a continuous fever; in cases in which death rapidly supervenes the temperature rises still higher toward the end, or occasionally falls. The course of the fever in cases that run a more protracted course may vary greatly, and the curve may assume a remittent, an intermittent, or a slowly falling continuous type. Further, in some cases the temperature does not rise above normal at all, or at most only a little above normal—*i. e.*, to 100.6° to 101.5° F. These are the most malignant cases, and rapidly prove fatal within thirty-six to forty-eight hours. These cases manifest the most marked symptoms of the intense toxemia—viz., profound collapse, extremely rapid and very low tension pulse; during the course of this short but severe syndrome the local signs of peritonitis are often practically absent or merely point to the presence of the earliest changes of peritonitis.

Another feature of special interest in the clinical picture is the occurrence of diarrhea, which has already been mentioned several times. As a rule, diarrhea is described as a common symptom of "puerperal" peritonitis; this is not, however, quite correct. It is true that diarrhea is particularly common in peritonitis occurring in parturient women—in fact, more frequent in this class of patients than in any other, but this is only the case because the inflammation of the peritoneum in puerperal cases has the characters of a septic disease. The frequency of diarrhea in puerperal peritonitis is remarkable: there may be as many as 15 to 20 actions of the bowels in twenty-four hours.

It is clear that in septic peritonitis other signs of sepsis occasionally make their appearance, such as cutaneous hemorrhages, marked icterus, etc. On the other hand, the signs proper of peritonitis—*i. e.,* those directly due to the anatomic changes of the peritoneum—may be only slightly marked or even entirely absent; there may, for instance, be no exudate, or only the smallest amount; this is apparently due to the fact that death occurs so rapidly that none of these phenomena have time to develop. The longer the course of the disease, the more marked, as a rule, the signs of peritonitis proper. A striking and peculiar symptom in many of these cases is the slight degree of spontaneous pain and of tenderness on pressure over the abdomen.

The **prognosis** of the septic form is very bad, and the majority of the patients die; occasionally, however, though this is distinctly rare, cases in which all the characteristic features are present recover. The factors that determine the prognosis have already been described under the heading of Diffuse Peritonitis (p. 781). When sepsis has fully developed, the course of the disease is generally short, seldom exceeding two weeks, and occasionally, particularly in puerperal cases, very acute; I have seen death occur within twenty-six hours after the onset of the first symptoms. In other cases the course of the disease is slow and insidious, especially when the septic process becomes localized in some part of the body and thus leads to the development of pleurisy, arthritis, etc. In other instances the septic inflammation of the peritoneum itself proceeds slowly and gradually. Cases that ultimately recover always run a long and protracted course.

PUERPERAL PERITONITIS.

Inflammation of the peritoneum following delivery or the puerperium is one of the most serious of the various morbid conditions that were formerly grouped under the dreaded name of "puerperal fever." Even at the present day the prognosis of this disease is as gloomy as before, and the treatment as unsatisfactory and hopeless as in the past; but from an etiologic point of view, at least, the mystery no longer exists, which formerly obscured the conception of puerperal peritonitis and the whole group of so-called puerperal fevers. It is unnecessary to describe or even enumerate all the various febrile conditions which, up to a few decades ago, were included under the latter name, for this section is concerned only with the inflammation of the peritoneum that may occur in the puerperium.

Peritonitis following labor or the puerperium or rather caused by delivery always has the same pathogenesis and etiology; in each individual case, however, there are some anatomic differences and some corresponding clinical differences. In some cases the inflammation of the peritoneum is limited to the uterus, the adnexa, and their immediate neighborhood; this form of puerperal peritonitis will be described under the heading of Local Peritonitis; in other cases the inflammation becomes diffuse and is one of the most serious forms of peritonitis known; it is this form of puerperal peritonitis that will be described here.

Etiology.—Until quite recently there was chaotic confusion as to the nature and etiology of puerperal peritonitis in particular, and of puerperal fever in general; it is only within the last decades that it has become recognized that this form of peritonitis is produced by the action of bacteria or of their poisons. In the section on the General Pathology of Peritonitis a number of bacteria capable of producing peritonitis were enumerated; among these the Streptococcus pyogenes is the most important in the pathogenesis of puerperal peritonitis; in fact, it is probably exclusively concerned in the etiology of puerperal inflammations of the peritoneum, as has been shown by Pasteur, Mayrhofer, Rindfleisch, von Recklinghausen, Waldeyer, Klebs, and Orth in the earlier days of bacteriology; later, when the perfection of bacteriologic technic that we owe to Koch became the common property of investigators, a large number of fresh researches on this subject established the etiologic rôle of the Streptococcus pyogenes beyond a doubt. In the great majority of cases, then, of puerperal peritonitis the Streptococcus pyogenes must be regarded as the specific inflammatory agent (as this micro-organism is, in fact, the specific infection in almost all anatomic forms of puerperal fever). The only question to be answered is whether other micro-organisms also play a rôle in these processes. This query can be answered in the affirmative, for the Streptococcus erysipelatis has been found in the exudate of puerperal peritonitis (Winckel); in addition, possibly, the gonococcus and some kinds of staphylococci; the presence of these and of some other bacteria that I have not mentioned must, however, be considered quite exceptional.

The microbes or their toxins gain an entrance to the peritoneal cavity from the genital tract through the numerous lymph-spaces that are injured during parturition; they do not pass so freely through the blood-vessels. The point of entry is chiefly in the cervix and in the endometrium, less commonly in the external genitals and the vagina. In those exceptional cases in which infection of the peritoneum occurs before delivery the microbes in all probability enter the peritoneum through some of the erosions and ulcerations that are so common on the vaginal portion of the cervix uteri in pregnant women; some authors, it is true, claim that peritoneal infection may occur without the existence of any visible lesions of the sexual organs.

Since Semmelweiss, in 1847, first brought forward the views on the origin of puerperal fever that are now universally accepted, all investigators are agreed that this infection occurs only when infectious agents are introduced from without. The pathogenic germs are conveyed by the hands of the medical man or of the midwife, by improperly sterilized instruments, and by vessels, bandages, and linen. Pathogenic microbes may in this way enter the genitals either before, during, or after labor. The correctness of this view, which is of fundamental importance in understanding the pathogenesis of puerperal fever and in its prevention, is now, fortunately, universally recognized, and it is useless to waste words in trying to *modify* this generally recognized truism. None of the other etiologic factors formerly credited with a rôle in the patho-

genesis of puerperal fever exist. Puerperal fever, and consequently puerperal peritonitis, is pathogenetically a disease that is due to wound infection. The only point on which some authorities still differ is as to the mode of introduction of these germs; for while some writers claim that infection of the peritoneal cavity occurs only in the manner described above, others believe that it may also occur through other channels. Many writers have at different times claimed that the disease could be transmitted through the air of lying-in wards. That puerperal infection can be air-borne has not yet been proved, and there are many reasons against this idea, but the possibility of infection by air-borne microbes cannot be absolutely denied; still, if it ever does occur, it is exceedingly rare.

Again some writers speak of auto-infection in puerperal peritonitis, meaning that micro-organisms that may be present in the sexual organs before labor may lead to infection postpartum, although infection may not have occurred by contact or by examinations during labor. Space does not permit of a résumé of all that has been published on the bacteriology of the micro-organisms that are normally present in the female genitalia, but it may be pointed out that among gynecologists there is a great diversity of opinion in regard to the possibility of auto-infection as defined above. Some are strong advocates of the possibility of auto-infection; thus, Ahlfeld summarizes his opinion as follows: "Micro-organisms are always present in the vagina that under favorable conditions may produce fever and death." Others again, such as Winckel and Fehling, vigorously oppose the idea of auto-infection, and doubt whether it can ever occur. It is impossible to enter into the details of all these investigations, and the final decision on these questions must be left to the gynecologists; from a practical point of view, however, it is impossible to overestimate the importance of the fact that micro-organisms can be transferred by direct contact, as the recognition of this fact is of the greatest importance in the prevention of the disease.

Puerperal infection and peritonitis in general are more common in primiparæ than in multiparæ; this is due to the fact that in the former the genital organs are more likely to be extensively lacerated; for the same reason postpartum infection of the peritoneum is more frequent when the fetus is large than when it is small, and when labor is protracted, particularly in the first stage. In cases of retained placental membranes or blood-clots in the uterus the danger of infection of the peritoneum is also greater.

Pathologic Anatomy.—There are several ways in which diffuse puerperal peritonitis may originate—*i. e.*, there are several paths by which the streptococci may get into the peritoneal cavity. By far the most common means of ingress are the lymph-channels. These vessels are of great importance in causing the spread of pathologic processes from the vagina to the uterus, and thence to the peritoneal covering of the genital organs. Streptococci that find a nidus in wounds of the vagina, the uterine cavity, the insertion of the placenta, and particularly of the cervix uteri, spread from these points *via* the lymph-

channels ; the lymph-channels will be found dilated, with thickened walls ; their contents are either granular and solid, like a thrombus, or yellowish, like pus (metrolymphangitis puerperalis). Cruveilhier, Virchow, and Buhl described this peculiar affection of the lymph-channels long ago ; Waldeyer was the first to prove that the mass of the so-called thrombus consisted in large part or often entirely of numerous spheric cocci, later recognized as Streptococcus pyogenes, and pus-cells.

The muscular tissue of the uterus may also occasionally be affected, especially in the more severe cases, in which the uterine walls may contain numerous small abscesses, while in very severe cases the whole wall may be necrotic and disintegrating up to the level of the peritoneum ; this condition, however, is exceptional.

The streptococci travel by the lymph-channels, and pass from the point of entrance by way of the paravaginal—*i. e.*, parametral—connective tissue to the subserous connective tissue of the broad ligaments, and so to the peritoneum, where they set up inflammation. They may travel still further through the lymph-channels of the abdominal cavity into the thoracic duct, or through the stomata of the diaphragmatic peritoneum and the diaphragm into the pleura and the pericardium.

This form of puerperal fever, in which the process extends in the way just described, is called lymphangiotic or phlegmonous puerperal fever. Virchow has also called the severe forms of this affection erysipelas puerperale malignum internum.

The process rarely spreads by the blood-vessels. Infection of the veins on the surface of wounds in the puerperal genital canal (metrophlebitis puerperalis) may, it is true, lead to very severe forms of thrombophlebitic sepsis (pyemia), and may even give rise to metastatic embolic inflammation of the pericardium, the joints, the lungs, and pleura ; but peritonitis produced in this way—*i. e.*, by streptococci—in the blood-stream must be considered very rare. Peritonitis by continuity is another possible result of thrombophlebitis of infective origin involving the vessels of the uterus and the adnexa, and though commoner than the one just mentioned, is also rare.

In all these cases the Streptococcus pyogenes is again the most common cause of the inflammation. There are not sufficient grounds for stating that a mixed infection always occurs when microbes directly invade the blood-vessels. It is true that such mixed infections occasionally occur ; staphylococci and saprophytes, especially members of the coli group, are comparatively often found in the metastatic abscesses. Some other anatomic conditions must be mentioned which may be the cause of peritonitis. Infective endometritis may extend to the tubes and produce pyosalpingitis, which in its turn may in various ways lead to diffuse peritonitis ; the pus may either leak into the peritoneal cavity through the ostium abdominale—this is the most frequent event—or the pyosalpinx may become so enormously distended that it ruptures ; or, lastly, it is at least conceivable that infective micro-organisms may pass through the wall of the tubes, even without any gross perforation.

Diffuse peritonitis may originate from purulent oöphoritis in the same manner as from pyosalpingitis.

Lastly, the possibility that a parametric abscess or an intraperitoneal abscess in the pelvis may rupture into the peritoneal cavity must be borne in mind. All the different possibilities just mentioned may be the cause of diffuse peritonitis at a late period of the puerperium. Diffuse puerperal peritonitis proper, let me repeat, in the great majority of cases originates from metrolymphangitis.

The detailed description of the changes in the sexual organs in the course of diffuse puerperal peritonitis must be omitted here, as it is not an essential part of this subject. The peritonitis itself leads to the development of clinical manifestations which have already been described in the general part of this volume, where the anatomy of diffuse peritonitis was described. It is only in the very mild cases that the exudate is fibrinous or largely serous; in cases that are at all severe the exudate is purulent, occasionally hemorrhagic or putrid. The amount of exudation varies: it is often very large, and may amount to as much as one liter in thirty-six to forty-eight hours. Corresponding to the anatomic origin of the disease in the genitalia, the most marked changes —the thickest layers of fibrin, the largest abscesses—are found in the pelvis in the vicinity of the female sexual organs.

In contrast to the majority of cases in which there is a large exudate some cases of puerperal peritonitis run their course without much exudation; this is seen in the most severe cases of all—*i. e.*, in the septic form, where occasionally there is only a trace of exudate or even none at all; this peculiarity will be referred to later.

A remarkable and common feature of diffuse puerperal peritonitis is the marked meteoristic distention of the intestine.

It will merely be mentioned here that pleurisy, endocarditis, pericarditis, and splenic enlargement often occur in this disease and can be explained most satisfactorily from the etiology of this particular form of peritonitis (general infection); in some of the cases it must be assumed that the infection of the peritoneum steadily spreads and that eventually the infective agent succeeds in working its way through the diaphragm.

Clinical Features.—The clinical manifestations of diffuse peritonitis of puerperal origin depart more than any other form from the ordinary type of the disease described above. Although these main symptoms can all be recognized, there are, in addition, a number of variations and deviations from the common syndrome. The clinical features due to sepsis are specially prominent and have just been described in a special section, but there are, in addition, a number of symptoms in diffuse puerperal peritonitis which require detailed description.

The pain, the exudate, the attacks of vomiting, and the meteorism are essentially the same as in other forms of acute peritonitis. It is remarkable, however, how often the pain seems to start from the lower part of the abdomen, especially from the uterus; the latter organ and the tissues surrounding it are particularly painful on pressure, so that

the origin of the peritonitis can generally be readily traced to these parts. In addition, the tympanitic distention of the abdomen is specially prominent. This, however, is not due to the fact that in puerperal peritonitis a particularly large amount of gas is developed, or that the muscular wall of the bowel is especially affected, but to the specially distensible condition of the abdominal walls, which have been stretched and relaxed during the long period of pregnancy preceding the onset of peritonitis.

It has long been recognized that in puerperal peritonitis, in contrast to other forms of peritonitis (which are usually accompanied by constipation), the bowels are loose; the number of evacuations may vary greatly: I have seen as many as 17 motions in twenty-four hours. The motions may be the same as those in acute infective diarrhea; in other instances they may contain mucus and blood and thus resemble those passed in severe cases of acute enteritis. I have already pointed out (see p. 772) that these attacks of diarrhea are probably due to the septic intoxication, and that clinical observation strongly supports this view. Analysis of my cases shows that puerperal peritonitis may be accompanied by continuous constipation; but the general aspect of these cases does not differ from that of ordinary diffuse peritonitis. Conversely in all those cases—and they constitute the majority—in which diarrhea is present other signs of a general septic intoxication are also present.

The course of the fever is in many instances exactly the same as in other forms of diffuse peritonitis. (See p. 775.) In cases in which, in addition to metrolymphangitic peritonitis, there is some metrophlebitis, the character of the fever becomes peculiar, and there are chills due to embolic occlusion of veins with septic thrombi and to the passage of infected material through the blood-stream. Occasionally there are spells of "goose-skin" or attacks of chills in the course of purulent lymphangitic peritonitis without phlebitis; but these attacks remain single and there is not the common and horrible condition in which the patients are tortured to the limit of endurance with chill after chill in rapid succession and sudden rises of temperature alternating with these chills; the latter picture is seen only in infective metrophlebitis. The onset of the fever is described differently by various observers; thus Winckel, for example, states that at the onset there is "almost constantly" a severe chill, lasting for a considerable time (from half to three-quarters of an hour); Ahlfeld, on the other hand, says: "The onset of peritonitis is, as a rule, insidious, and the disease is rarely ushered in with a chill; as a rule, there is a mild sensation of chilliness immediately after delivery or in the course of the first or second day after labor. It is only when the inflammation of the peritoneum is due to rupture of an abscess into the peritoneal cavity that there is a genuine chill." From my own observations I am more inclined to agree with Winckel, as I have much more often than not seen the onset of peritonitis accompanied by a chill; on the other hand, I do not feel justified in stating that an initial chill is a constant occurrence. The

peritoneal pain occasionally precedes the onset of the fever; but, as a rule, it follows rapidly on the rise of temperature. As to the exact time when fever and other signs of peritonitis appear, I cannot corroborate the statement that this is usually ("almost without exception") during the first or the second day after delivery. All that I feel justified in saying is that, as a rule, the average time of onset is the second day after delivery. I have records, however, of a considerable number of cases in which the disease did not come on before the third, fourth, fifth, sixth, seventh, or eighth day.

Another feature which must be mentioned is that the lochia, as a rule, become scanty or more or less offensive. The secretion of milk, if it has begun, as a rule rapidly ceases.

The prognosis in puerperal forms of diffuse peritonitis is even worse than in the other varieties of diffuse peritonitis without perforation. Happily, it is true, a certain proportion of recoveries is recorded even in this disease; in favorable cases the course of the disease is acute, and a return to the normal occurs gradually and by degrees, the symptoms slowly diminishing and becoming less marked; occasionally the patient recovers from the disease after many vicissitudes and variations in the clinical aspect of the disease, the variations being due to different secondary results depending on the presence of a purulent exudate in the peritoneal cavity; in these cases complete recovery is often delayed for weeks or months. Puerperal peritonitis, however, is rightly the cause of the greatest anxiety, for apart from the occurrence of metrophlebitis and septicemia, extremely severe complications in themselves, the disease far more often assumes a general toxic or septic character than any of the other forms of diffuse peritonitis; it is, moreover, specially prone to be complicated by conditions such as pleurisy, pericarditis, arthritis, etc., all localized manifestations of the general septicemia. Under these circumstances death results very rapidly—often on the fourth to the seventh day of the disease, though in some cases not until the second or the third week. In these cases the prognosis is extremely gloomy and recovery is very rare; many observers indeed state that in the presence of these complications the disease is invariably fatal, a statement with which I agree so far as regards the markedly septic cases.

PROGRESSIVE FIBRINOPURULENT PERITONITIS.

The cases included in this group form an anatomic and clinical transition from the circumscribed to the diffuse forms of peritonitis. Mikulicz, who introduced the title used above, has given the best description of the condition. To quote his own words: "The characteristic feature of this form is that at first only a circumscribed area of the peritoneum is affected, the boundaries of which are formed by peritoneal adhesions or by fibrinopurulent masses which unite the adjacent layers of the peritoneum. In this way a more or less resistant wall is formed which protects the healthy portions of the peritoneum from sudden infection. Within the affected (encysted) area a fluid,

purulent exudate collects in one or more foci, separated from one another by peritoneal adhesions. The boundaries of the area affected by peritonitis gradually extend by the formation of fresh adhesions and purulent foci. The protective wall between the affected and the healthy parts of the peritoneum persists, however, throughout the course of the disease, unless the whole of the peritoneum eventually becomes involved in the process. This, however, probably occurs only in rare instances in this form of peritonitis, and, as a rule, the patients succumb to the disease before this happens and at the autopsy large or small portions of the peritoneum, particularly those at a considerable distance from the morbid process, are found to be free from inflammation. The longer it lasts and the slower the course of the disease, the firmer the adhesions and the more definite the boundaries of the peritonitis, and the greater the change in the character of the exudate. The latter is first thin and seropurulent or sanious, and later becomes more distinctly purulent. In very chronic cases nature may effect a cure by causing contraction of the adhesions, thickening of the purulent exudate, and evacuation of the pus through the skin or into some internal organ ; or, lastly, gradual absorption of the pus may occur."

It has already been pointed out that Lennander distinguishes another form from this, in which multiple encapsulated abscesses exist, but do not owe their origin to spreading by contiguity. The original infection attacks several parts of the peritoneum at one and the same time, and this happens through accidental distribution of the infective material.

I only wish to add the following remarks to Mikulicz's clear description. The progressive purulent form of peritonitis may originate from circumscribed inflammation of any part of the peritoneum ; most commonly, of course, the starting-point is a perityphlitic, a paranephritic, or a subphrenic abscess, but it may also start from one of the less common primary foci, such as a suppurating mesenteric or retroperitoneal gland, etc. (The reader is referred to p. 763 for a description of the spread of this form of peritonitis and for all the anatomic and pathogenetic details of this process, and to the same section (pp. 750–764) for the pathology of peritonitis in general.) The size of the various collections of pus which are separated by septa may vary greatly ; occasionally, when they lie between several coils of intestine or between the folds of the mesentery or between the mesentery and the intestine, they are no larger than a walnut or even a hazelnut.

The clinical picture is often so clear and characteristic that the diagnosis is simple, especially when a perityphlitic, parametric, or some other primary focus is detected and the progressive character of the affection is recognized. The localized pain gradually extends to other parts of the abdomen, and sometimes, under favorable conditions, it is possible to follow the progress of the inflammatory exudation and suppuration by palpation and percussion. The temperature is especially important ; it may either be remittent, intermittent, or continuous in type, and if intermittent and accompanied by chills and rigors, it may

assume a septic type. Between the attacks of fever there may be periods of complete apyrexia; under these circumstances each fresh rise of temperature indicates an advance of the peritonitic process. The pulse is, as a rule, quickened, the general strength fails, and the patients soon become very anemic and emaciated; there is also loss of appetite. Other symptoms, such as meteorism and intestinal disturbance, are very inconstant, and complications involving the kidneys, the pleura, etc., do not properly belong to the clinical picture, but should be regarded as occasional complications.

When the patient is examined for the first time with the disease fully developed, it is often difficult or impossible to make out the starting-point of the disease, though the existence of progressive purulent peritonitis is usually easily recognized. As a result of operative interference in such a case, either for diagnosis or for treatment, a single collection of pus only may be opened, especially if simple puncture is made, while other purulent foci may remain unopened; this may readily lead to diagnostic errors. It need hardly be mentioned that occasionally simple puncture is quite futile, even though pus is present; but under these conditions the diagnosis can often be made from the clinical signs.

Although progressive fibrinopurulent peritonitis is a very serious disease, the prognosis is not absolutely bad. It is true that many of the patients die; the protective wall of adhesions may eventually give way, and the whole peritoneum suddenly become deluged with pus, which condition, of course, proves fatal; or the patient may become exhausted from the hectic fever or deficient nutrition; or dangerous complications, such as pleurisy, pericarditis, etc., may develop and precipitate the fatal issue. On the other hand, the case may recover not only as a result of surgical intervention, but spontaneously. The anatomic nature of this disease, however, makes it possible for discharge of pus to occur without the disease being thereby cured, for only one of the many purulent foci may thus be evacuated. On the other hand, we know that the purulent foci are, as a rule, so small that spontaneous absorption and resolution are never out of the question.

No definite statements as to the duration of the affection can be made. Occasionally the interval between the onset of the inflammation and until death is only two or three weeks; in these cases sudden perforative peritonitis with rapid death is likely to occur; in other cases, if a favorable turn once sets in, either spontaneously or as a result of surgical interference, the course of the disease may become indefinitely protracted and extend over many months.

TREATMENT OF ACUTE DIFFUSE PERITONITIS.

Acute diffuse forms of peritonitis are still unsatisfactory from the point of view of treatment; in fact, when the underlying morbid lesions are duly considered, it seems very doubtful whether medical science will ever be able to have much effect on this disease; certainly

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there is very little prospect of success in cases where the morbid changes characteristic of the more severe forms of the disease are fully developed. It is true that at the present day, thanks to the advances of surgery, the treatment of this disease is not quite so hopeless as in the past. But the good results occasionally seen after operative interference are obtained in cases operated upon before the anatomic changes of diffuse peritonitis have fully developed.

In view of these facts the greatest importance must be attached to prophylactic measures; in other words, every effort must be made to prevent the development of diffuse peritonitis.

Prophylaxis.—From a review of all the various causes of this dangerous disease it is evident that in some groups of etiologically related cases prevention is easy, whereas in other categories it is impossible. The first category includes puerperal peritonitis, which, as a matter of fact, is extraordinarily rare now as compared with its frequency in former times. The epidemics which formerly proved so fatal in numerous institutions are hardly ever seen now in efficient hospitals. At the present day cases are exceptional and sporadic, or at most occur in small groups in the practice of an isolated medical man or midwife. Asepsis and antisepsis in combination with absolute cleanliness have brought about this splendid result, which is due to Lister and Semmelweiss' work. It is not necessary, however, to enumerate here the technical and medical regulations necessary to prevent puerperal peritonitis; these measures will be found in works on obstetrics, to which the reader should refer.

In traumatic peritonitis the same obtains, at least in cases due to surgical interference. In injuries of other kinds, however, peritonitis can, as a rule, be prevented when efficient antiseptic and other precautions are adopted sufficiently early.

The medical man who opens a circumscribed peritoneal abscess early enough to prevent perforation and entrance of pus into the general peritoneal cavity is adopting a decidedly prophylactic method of treatment, for he directly prevents diffuse peritonitis. The advocates of early operation in perityphlitis rely on this fact as one of their strongest arguments and base their recommendation of frequent and early interference in this disease on the recognized fact that opening an intraperitoneal abscess effectually prevents diffuse peritonitis from spontaneous rupture of such a focus. What has been said of perityphlitis applies, of course, with equal force to other forms of circumscribed suppuration in the peritoneum.

The most difficult form of peritonitis to treat prophylactically, and which, in fact, it is often quite impossible to prevent, is that following perforation of gastro-intestinal ulcers. What physician would dare to advise a laparotomy or resection of the bowel in such cases before perforation had actually occurred, even supposing the diagnosis of tuberculous, catarrhal, or decubital ulcers could be made with a reasonable degree of certainty (a positive diagnosis can probably hardly ever be made) or would urge this treatment in the course of typhoid fever or

of dysentery ? Merely to ask is to answer this question ; and it is quite unnecessary to multiply arguments to prove that such a course would be unwarrantable. As regards the question whether it is not justifiable to operate in cases of gastric ulcer which show a tendency to perforation, I agree entirely with Leube's view that such interference for prophylactic reasons is unwarranted, if for no other reason than that it is impossible in any given case to recognize that perforation is impending. This, of course, also applies to duodenal ulcer.

The prophylactic treatment of peritonitis is by no means limited to the various points just enumerated. In the section on the Etiology of Peritonitis attention was called to the fact that the anatomic changes caused by the inflammation of the peritoneum require a certain time for their full development (at least several—*i. e.*, four to ten—hours). In other words, some time must elapse between the infection of the peritoneum by pyogenic microbes, etc., and the development of the anatomic lesions characteristic of peritonitis. The time when the peritoneum becomes invaded can often be determined very accurately ; sometimes, in fact, as in sudden perforation, the exact moment can be fixed by means of the pain caused by the perforation. In the interval between the rupture or perforation and the onset of the first signs of peritonitis —*i. e.*, in the first few hours after the accident—treatment can, or rather should, most certainly be undertaken. These measures, properly speaking, are still to be classed under the heading of prophylaxis. The whole situation in these cases, in the light of our present knowledge, urgently calls for only one form of treatment, and that is operative. The surgical treatment of fully developed peritonitis, which is much more general and broad than the simple prophylactic interference just sketched, will be described in detail below, under the surgical treatment of other forms of peritonitis. A connected description of the general and purely medical methods of treatment will be given here.

Medicinal and General Measures.—The first and most important measure is to place the patient at rest. The patients often keep perfectly still of their own accord, because the pain almost forces them to do so. When they do not lie still voluntarily, measures should be taken to insure that they do. The medical man must use his own judgment and must decide in each individual case to what extent the patient should be moved passively for the sake of careful physical examination. It is hardly justifiable to forbid absolutely these passive movements, for a careful examination of the patient is often absolutely necessary in order to find out, for instance, whether there is a perforation and whether, consequently, an operation is required. When the condition is quite clear without further examination of the patient, no one, it is to be hoped, would be callous enough to insist on a more exhaustive physical examination of the patient merely for the sake of "an exact clinical examination."

Lennander insists, particularly in peritonitis of the lower half of the abdomen, upon a definite position—viz., that the lumbar region should form the lowest point of the abdominal cavity. To this end he

raises the foot of the bed, and by thus assisting the return of blood from the lower extremities, prevents venous thrombosis. Similarly, he raises the upper part of the trunk to minimize the passage of infectious material from the lumbar to the subphrenic region.

The necessity for keeping the intestine quiescent is almost as important as, and possibly even more important than, general rest. Peristalsis increases the pain, and its effect, therefore, is not only distressing to the patient, but does definite harm. For peristalsis is the active factor in the spread of peritonitis by disseminating the bacteria or bacterial poisons widely throughout the peritoneal cavity ; in this way fresh areas of the peritoneum are constantly being infected ; the peristaltic movements of the bowel-wall also prevent the inflammatory process from being localized, which is so important in these cases. Lastly, peristalsis may effectually prevent the fibrinous exudate from closing up perforations or small ruptures. For all these reasons it is an essential part of the treatment in these cases to use all the available means at our disposal to stop peristalsis and to prevent any increase of the intra-abdominal pressure.

A patient suffering from diffuse peritonitis, and in particular from perforative peritonitis, should be forbidden to take anything at all into the stomach during the first few days after the onset of symptoms of peritonitis. Not only solid food, but all liquids and water, should be prohibited ; he should not even be allowed to swallow small pieces of ice or cold effervescing waters, both of which are usually given to allay thirst and stop the tendency to vomit that these patients suffer from, for it must be remembered that anything taken into the stomach stimulates peristalsis, and thus vomiting is readily set up and is more severe than when the stomach is kept completely empty. In order to alleviate the thirst which often tortures the unfortunate patients small pieces of ice should be allowed to dissolve in the mouth, and the water not swallowed, but spat out ; if the pain is not so severe as to prevent irrigation of the bowels, small quantities of water should be allowed to flow into the rectum. Intravenous or subcutaneous transfusion of a normal (0.6 per cent.) solution of sodium chlorid is most effective. Subcutaneous injection is sufficient in practice, is simple, and is easily carried out. The solution is injected warm twice daily, from $\frac{3}{4}$ to 3 pints at a time, and preferably into the lower extremities. This is continued until fluid can be safely given by mouth. When the general condition makes it necessary that some food should be given to sustain life, the only permissible way is by the rectum, or, in cases of urgency, by subcutaneous injection of oil.

From what has been said it is quite clear that laxatives must be avoided altogether ; the same applies to the use of purgative enemata. In this respect I appear to agree with the great majority of medical writers on this subject. The view of a few clinicians who advise the use of all these measures is in marked opposition to thousands of careful clinical observations which clearly indicate the course to be pursued in these cases. Baldy, for instance, claims never to have seen any bad

effects from the use of saline laxatives followed by a copious evacuation of the bowels in cases of incipient or even of fully developed peritonitis. Treves, it is true, limits the use of laxatives and gives them only at the commencement of peritonitis and in the circumscribed forms of the disease, and fully condemns their use in diffuse peritonitis. It is well known that formerly diffuse puerperal peritonitis was treated with laxatives from the very onset; this method, though still employed by a few practitioners, has largely been discarded, and, I believe, rightly so.

[Years ago the late Mr. Lawson Tait strongly associated the use of magnesium sulphate in general peritonitis, so as to prevent intestinal distention, stagnation of the contents of the bowel, and toxic absorption. This use of purgatives, which has, of course, exactly the opposite effect to morphin, has been largely accepted and employed in practice in acute peritonitis. Recently magnesium sulphate in solution has been introduced directly into the intestines by means of a syringe during the course of operations on cases of peritonitis (McCosh,[1] Weir,[2] Sheild,[3] Hutchinson,[4] Morton[5]). Mayland[6] has introduced sulphate of magnesia into the bowel, after letting out its contents, through the evacuating incision. Sheild introduced the nozzle of the syringe into the stump of the vermiform appendix in cases of peritonitis of appendicular origin. Magnesium sulphate and glycerin can also be given as an enema. Barnard[7] speaks highly of calomel, given every hour in one-grain doses, so as to open the bowels and maintain a moderate diarrhea; in some cases at the London Hospital 40 and 50, and in one case 72, grains of calomel have been given. In addition to its purgative effect, the antiseptic action of calomel is important.—ED.]

The use of opium, on the other hand (or of morphin), is of the greatest importance; Graves and Stokes were the first among the great clinicians to introduce these drugs into the treatment of perforative peritonitis, and later into the treatment of diffuse peritonitis. Numerous medical men since then have recognized their great value and warmly advocated their use. It is true that opium and morphin have not the slightest influence upon the morbid process itself, but they undoubtedly modify the symptoms in a number of different ways. Morphin more especially quiets peristalsis and in this way gives the patient all the benefits that accrue from arrest of the intestinal movements; in addition, it relieves pain and restlessness, allays thirst, stops vomiting and hiccup, and, under certain circumstances, may exert a beneficial effect in cases of shock following perforation, and, finally, when all hope of recovery is abandoned, it gives the patient the boon of euthanasia.

The dose of opium, particularly when given in the early stage of the disease,—and chiefly when employed in perforative peritonitis,—should be

[1] McCosh, *Annals of Surgery*, vol. xxvi., p. 691.   [2] Weir, *ibid.*, vol. xxvi., p. 236.
[3] A. M. Sheild, *Brit. Med. Jour.*, 1901, vol. ii., p. 1864.
[4] J. Hutchinson, Jr., *ibid.*, 1902, vol. i., p. 77.
[5] C. A. Morton, *ibid.*, 1902, vol. i., p. 77.
[6] A. E. Mayland, *ibid.*, 1899, vol. i., p. 842.
[7] H. L. Barnard, *Clinical Jour.*, September 30, 1903, vol. xxii., p. 376.

fairly large—*i. e.*, 10 to 15 minims of the tincture or 0.05 to 0.1 gram of the extract two to four times at intervals of one hour; subsequently it should be given at longer intervals or in smaller doses. Penzoldt recommends the general plan of allowing the dose of opium given at a time and the total daily dose to fluctuate between the maximum dose and half the maximum dose in adults (tinctura opii simplex, 0.75–1.5 pro dosi; 2.5–5.0 pro die; opium, 0.075–0.15 pro dosi; 0.25–0.5 pro die). The tincture is preferable, because it is more easily given and because the dose can be more conveniently regulated; if the tincture produces nausea, opium or the extract of opium may be given instead. If the stomach will retain nothing, the drug may be administered in the form of suppositories containing 0.1 of the remedy, three or four daily. It is when pain is but slightly influenced by the opium that morphin should be given hypodermically; the latter remedy, however, does not exert so marked an effect on peristalsis.

It need hardly be pointed out that individual peculiarities must determine the exact dose in each case. Anemic and weakly subjects, old people, and children should receive smaller doses. In the septic form of the disease, with cerebral symptoms and cardiac failure, in which, moreover, the pain is comparatively slight or is entirely absent, opium is contraindicated, as well as in cases where there is excessive meteorism, except when the prognosis is absolutely fatal and the drug is given only to make death more comfortable. On the other hand, in morphiomaniacs or opium-eaters the dose should be larger than ordinary. Bearing all these points in mind, the general rule can be formulated that the dose should be regulated according to the pain.

Of late years intravenous and subcutaneous injection of large quantities of fluid has become important. It was first thought of as a means of quenching great thirst, but is, besides, used for other ends. Thus Michaux (quoted by Friedrich) regards it as a specific remedy in peritonitis, and Friedrich, on the ground of clinical experience, attaches great importance to it. According to clinical experience and experimental research it enables one to prevent the depletion of the body of its normal amount of fluid; thereby the blood-pressure is raised and the heart protected from the dangers of insufficient blood-supply. Lastly, it is believed, by Sahli and others, that the infective poison is to a certain extent actually washed out of the body.

[Barnard[1] says that the use of massive subcutaneous saline transfusions has entirely changed the point of view at the London Hospital as to cases of general suppurative peritonitis. This method was originally practised in Kocher's clinic at Berne, where as much as 20 or 30 pints had been transfused into the subcutaneous tissues by four needles. Barnard says that 15 pints can be transfused into a man in twenty-four hours; little boys have had 4 pints directly after operation and 4 more a few hours later. The effects are very remarkable and most beneficial, and are due to several factors—(1) The replacing of fluid lost by the body in various ways; (2) by filling the vascular system, the tendency

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[1] H. L. Barnard, *Clinical Jour.*, September 30, 1903, vol. xxii., p. 376.

to absorption of toxins from the pus, etc., in the peritoneal cavity is diminished and there is a tendency for fluid to flow in the opposite direction—viz., into the peritoneum ; (3) dilution of toxins in the circulation. Kraft[1] recommends that transfusion should be performed before operation in peritonitis, so as to retard absorption from the peritoneum.—ED.]

Putting aside unreasoning optimism and all attempts to exaggerate the efficacy of treatment in actively influencing the course of this disease, it must be admitted that the measures just described—*i. e.*, absolute rest, strict diet, opium, etc.—practically exhaust all the available general measures and drug treatment. Numerous other measures, it is true, especially local applications to the abdomen, are occasionally employed, but it is very doubtful whether they exert any appreciable influence on the symptoms they are intended to relieve. This applies to the use of cold applications, which are generally employed. However useful cold applications may be in circumscribed inflammation, their value in diffuse peritonitis is very doubtful. It is only necessary to remember the anatomic conditions present—viz., the wide extent and the partially hidden position of the inflamed membranes—to realize that cold can exert very little effect. In addition it must be remembered that occasionally the cold and the weight of a heavy ice-bag are distressing, or even quite unbearable ; the latter disagreeable effect, it is true, can be avoided by using a Leiter coil. Sometimes the pain is somewhat lessened and a soothing effect is produced by the application of heat to the abdomen ; as I am fully convinced that heat is no more capable of exerting a bad effect on the course of acute diffuse peritonitis than cold is capable of exerting a favorable effect, I usually allow the patient's sensations to decide whether heat or cold should be applied in any given case. If the practitioner wishes to proceed carefully and cautiously, he should apply Priessnitz compresses. Some sort of application to the abdomen is necessary in almost all cases, because most patients are anxious to have the affected part treated locally.

Local depletion (leeches) may be dispensed with in diffuse peritonitis. Very occasionally I use them as an anodyne when there is extreme pain in some strictly localized part of the abdomen which is not properly controlled by opium.

Formerly general mercurialization (inunctions with gray ointment and calomel internally) was much in vogue ; at the present day this form of treatment has been abandoned by the majority of clinicians, and rightly so.

Antipyretics are quite useless in the treatment of this disease. The fever is either so slight that their use is quite unnecessary (a reduction of temperature in cases with high fever is of no importance in view of the gravity of the general disease) or definite harm even may result from their influence on the heart.

No further description will be given of the other therapeutic measures that have been recommended in this disease. Careful analysis shows

[1] L. Kraft, *Hospitals Tidende*, 1903, p. 33.

that the treatment of the horrible disease known as diffuse peritonitis is unfortunately practically limited to the measures already mentioned. Recent knowledge as to the pathology of the disease makes it quite clear why we are so helpless in this disease. Until some means is discovered of arresting the growth of bacteria in the peritoneal cavity, of preventing their inflammatory action on the peritoneum, and, in addition, of counteracting the deleterious action of the bacterial toxins on the general organism, the treatment must remain in its present unsatisfactory condition. It is impossible to predict whether serum therapy will open a new path, as experiments on this subject are still in their infancy. The reports of clinical cases, moreover, are still too few in number and too divergent to justify any discussion at the present time of this important problem.

The medical practitioner's chief aim in acute diffuse peritonitis—and I can only repeat what I have said above—must be to act from a prophylactic point of view, and when the disease is fully developed, to insist on quiet and the administration of opium.

There are, however, a few additional measures which can be employed to combat some of the more distressing and dangerous individual symptoms. In regard to the most important symptom—the pain—the essential points have been described. The chief remedy for the pain—viz., opium or morphin—is also useful in restraining the tendency to vomit and sometimes in preventing another very distressing symptom that occasionally appears—viz., hiccup. When opiates fail to relieve the last two symptoms, ice, iced effervescing water, black coffee, or a few drops of chloroform given internally may be successful. In addition a small dose of chloral or atropin subcutaneously may be tried for vomiting or hiccup; as in all other conditions that produce these symptoms, the heart should, of course, be carefully watched. The treatment of meteorism is usually quite useless and is at best purely symptomatic. (The reader should refer to what has been said on pp. 145–147.)

Cardiac weakness and collapse require a special description. In this connection it is necessary to distinguish between the so-called primary reflex collapse that is seen at the commencement of perforative peritonitis, and the collapse seen later in the course of peritonitis, particularly in the more or less pronounced septic forms, which is due to weakness of the heart's action. The best remedy for the primary collapse is removal or relief of the pain; the treatment, therefore, is based purely upon removal of the cause, and consists in subcutaneous injections of morphin to soothe the irritation of the sensory nerves. In addition, a hypodermic injection of caffein or camphor may be necessary in these cases. This treatment is also useful at a later stage of the disease when signs of cardiac weakness appear, when, too, the subcutaneous injection of normal salt solution is important. Since the treatment of peritonitis and its complications, generally speaking, is so unsatisfactory, I am glad to be able to indorse fully the use of digitalis as recommended by Frommel, provided always that the drug can be

given by mouth without producing vomiting; a very rapid pulse with falling arterial tension is the indication for digitalis. I have had no personal experience with strychnin, which has also been recommended in this condition. [Crile's [1] experimental work shows that in shock due to exhaustion of the vasomotor center strychnin and digitalis, which stimulate the center, are of little value and may do harm. He speaks highly of the good effects of adrenalin; it must be given with caution, and Bloodgood [2] recommends that it should be given subcutaneously rather than intravenously. Crile has invented a rubber suit by means of which, when inflated by an air-pump, pressure can be brought to bear on the surface of the body and the peripheral resistance of the circulation increased; in this way great benefit results until vasomotor tone is reëstablished.—ED.]

When the septic manifestations of the disease become very marked, large quantities of alcohol (brandy, strong wines) should be given. I shall not discuss the value of this remedy in these cases, but there is no more efficient measure in this hopeless condition, and to my mind alcoholic drinks are far less harmful in sepsis than salol, salicylate of soda, or large doses of quinin. Gersuny (Moskovicz) reported favorable results from hot (104° F.) baths; these appeared in hopeless cases to act as a powerful stimulus to the heart's action, thus tiding over the stage of collapse.

Lastly, as to the treatment of the symptoms due to paralysis of the bowel, especially stercoraceous vomiting, it may be briefly stated that washing out the stomach appears to be the only measure for this condition.

SURGICAL MEASURES.

After this review of the almost hopeless therapeutic measures, it is gratifying to draw attention to the part played by surgery during the last twenty years in the treatment of acute diffuse peritonitis. Surgery alone cannot, of course, cure peritonitis, but it can and does bring about conditions which favor the curative processes of the organism. Many hundreds of operations have been performed; Krogius, in 1901, published 680 cases of diffuse purulent peritonitis originating in the appendix. The mortality is still appalling (of the 680 cases 194 recovered and 486 died), but there can be no doubt it was higher before this era.

The literature of the last few years contains a steadily increasing number of successful cases, so that at the present time we are justified and even obliged to point out that so long as there is no medical measure of any kind which can be considered effective and reliable, such as some specific or serum treatment, the surgical treatment of acute diffuse peritonitis must be included among the valuable means of combating this disease.

Operative interference may be indicated in two radically different

[1] G. Crile, *Boston Med. and Surg. Jour.*, March 5, 1903, vol. cxlviii., p. 247.
[2] J. C. Bloodgood, *Progressive Medicine*, 1903, vol. iv., p. 86.

categories of cases of diffuse peritonitis—viz., first, before the inflammatory process proper has had time fully to develop—*i. e.*, at the very onset of the disease ; second, when inflammation and exudation are already fully developed.

In the first place, by operative interference it may be possible to find the origin of the infection, and prevent it from doing any further harm, as in perforating gastric ulcer, disease of the appendix, intra-peritoneal or extraperitoneal abscess. Further, appropriate surgical technic may act locally upon a case of peritonitis in its earliest stage and so prevent it from spreading.

Peritonitis in its fully developed exudative stage is quite a different matter. Generally speaking, surgeons who have recently expressed their opinion are agreed on the main point—*e. g.*, Tietze, Mikulicz, Lennander, and others—and one is in duty bound to side with them from a general pathologic standpoint. The real use of laparotomy in these cases is to empty the cavity of its infective (toxic) exudation, and possibly, by drainage, etc., to a certain extent to prevent its reaccumulation ; with the exudation one removes to a great extent bacteria and their products ; the deleterious effect of the latter upon the circulatory system has been referred to previously. On the other hand, the fact must not be overlooked that diminution of intra-abdominal pressure is not always accompanied by danger ; for thereby lymph-canals and vessels which were closed by actual pressure are opened up, and grave general infection may result from reabsorption of infective material from the peritoneal cavity.

Operative Interference at the Onset of the Inflammation.—In this category are the operations necessary in perforation of the intestine, of the stomach, of some circumscribed abscesses, the gall-bladder, etc. ; here interference is warranted at once—*i. e.*, within a few hours after the perforation into the free peritoneal cavity occurs.

From a practical point of view there are the following possible clinical entities :

Perforation of Gastric Ulcer.—According to the statistics of Mikulicz in *Die chirurgische Behandlung des chronischen Magengeschwürs*, which include all the literature up to the end of 1896, 35 cases were operated upon from 1885 to 1893 ; of these, 1 was cured and 34 died (mortality of 97.15 per cent.) ; from 1894 to 1896, 68 operations with 32 cures and 36 deaths (mortality of 52.94 per cent.) ; the more favorable results of more recent years are unmistakable and are apparently due to two factors : In the first place, the more extended experience of individual operators may have something to do with it ; in the second place, however, this favorable result must be largely attributed to the fact that operators, recognizing the good results to be obtained, decided to operate earlier in the course of the disease. There is absolutely no doubt that the chances of recovery are increased by early surgical interference—*i. e.*, really before the anatomic changes of general peritonitis have had time to develop. From the statistics available at present, it appears that the chances of recovery are approximately four times more

favorable when the operation is performed within the first twelve hours after the perforation than when undertaken at a later stage of the disease.

[This corresponds with Mayo Robson's [1] statistics that if perforated gastric ulcers are operated upon at once, there is hardly any mortality; if within twelve hours of rupture, a mortality of 16.6 per cent.; if within twenty-four hours, 63.6 per cent.; if within thirty-six hours, 87.5 per cent., and if delayed for forty-eight hours, recovery seldom occurs.—ED.]

It is noteworthy, however, that the operation is more likely to be successful in cases where the stomach is empty at the time perforation occurs; this factor, of course, is not within our control. From consideration, therefore, of the fact that only an infinitesimal number of the cases of peritonitis following perforation of a gastric ulcer recover without operation, and then only if the stomach was empty at the time of the perforation, whereas operative interference may occasionally, though rarely, save a case even though the stomach is not empty, the conclusion is inevitable that operative interference is indicated in every case of perforation of the stomach, provided the operation is technically possible.

What has been said of gastric ulcer applies with equal force to the closely allied condition of duodenal ulcer, which may, as we know, often be difficult to distinguish from it.

[In 25 cases of duodenal ulcer, 12 were operated upon within thirty hours of the perforation and 8 recovered, while 13 were operated upon more than thirty hours after perforation and all died.[2]—ED.]

Perforation of Typhoid Ulcers.—The general results of surgical interference are far less favorable in this lesion than in the preceding ones; this is quite intelligible, for the operation in typhoid ulcerations is performed under less favorable circumstances, because it is performed upon patients who previous to the operation have been suffering from a grave infectious disease. Finney has recorded 52 cases of operation, with 17 recoveries after perforation.

[In a subsequent paper Finney [3] collected 112 cases of operation for typhoid perforation, with 23 recoveries. In Keen's [4] 150 cases, which include Finney's figures, the percentage of recoveries was 22.7. Up to March, 1903, Elsburg [5] collected 289 laparotomies for typhoid perforation with 75, or 25.9 per cent., of recoveries.—ED.]

According to the statistics of Gesselewitsch and Wanach, who include 5 cases of their own, 11 cases of typhoid perforation out of 63 operated upon recovered. As spontaneous recovery is so rare in this condition, this result must be considered very satisfactory. It appears, therefore, that laparotomy in diffuse peritonitis following perforation of

[1] Mayo Robson, *Brit. Med. Jour.*, 1903, vol. ii., p. 242.   [2] Collin, *Thèse*, Paris, 1894.
[3] J. M. T. Finney, "Studies in Typhoid Fever," *Johns Hopkins Hosp. Reps.*, 1901, No. 3, p. 155.
[4] W. W. Keen, *Philadelphia Med. Jour.*, November, 1899.
[5] C. A. Elsburg, *Annals of Surgery*, part cxxvii., p. 71.

a typhoid ulcer must be considered a justifiable procedure. The authors mentioned above express themselves as follows : " Cases occurred in children, men, and women between the ages of seven and forty years, in ambulatory, mild, and severe cases of typhoid ; the perforation occurred early—on the eighth to the ninth day—and late—in the seventh week ; the operation was performed after two hours and after four to five days after the perforation ; the abdominal cavity in some cases contained gas and feces ; in other cases it did not ; the perforative opening was found in some cases, and in other cases it was not ; in some cases the abdominal cavity was washed out, while in others it was not ; the abdominal wound was sometimes closed and sometimes left open, etc. ; none of these factors appeared to exert any appreciable influence on the subsequent course of the disease. We are unable, therefore, to formulate any general or special rules or regulations on which to base the prognosis of laparotomy performed for the cure of typhoid perforation ; the recovery of these cases must so far be attributed to a fortunate concatenation of favorable circumstances in some of the individual cases." I merely wish to add one remark to this statement (which Gesselewitsch and Wanach arrive at on the basis of a number of recorded cases, and is probably correct in a general way), namely, that, so far as I can learn from the more or less condensed accounts, the best results were obtained in cases operated upon within the first twenty-four hours after the perforation.

[In 11 cases under Osler's [1] care, 5 recovered after operation. The danger of delay is shown by the following figures : Of 15 cases operated upon within twelve hours, 4 recovered ; of 20 cases operated upon between the twelfth and twenty-fourth hours, 6 recovered ; while of 13 operated upon in the second twenty-four hours, only 1 recovered. This subject has also been dealt with by Cushing [2] and McCrae and Mitchell. [3] —Ed.]

There is so little known at present about the surgical treatment of perforation of other forms of ulcers of the intestine into the general peritoneal cavity that no generalizations can be formulated on the basis of our present experience. It is best in these cases to act on the general principles which have been shown to hold good in other forms of perforation. The question of surgical interference in cases of diffuse peritonitis originating in the appendix will be fully dealt with in the section on Appendicitis and Perityphlitis.

The rupture of an abscess, it being immaterial whether it is intraperitoneal or extraperitoneal, into the general peritoneal cavity is always a most dangerous accident, and a spontaneous cure must always be very rare in such cases. Immediate laparotomy is the only means by which even a slight chance of recovery can be hoped for.

The question whether laparotomy should be performed in all these cases can be decided only after careful consideration of the patient's

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[1] W. Osler, *Principles and Practice of Medicine*, 1901, p. 47, fourth ed.
[2] H. W. Cushing, "Studies in Typhoid Fever," *Johns Hopkins Hosp. Reps.*, 1901, No. 3, p. 209.   [3] T. McCrae and J. F. Mitchell, *ibid.*, vol. x.

general condition. Many surgeons postpone operation until the shock following the perforation has passed off, while other surgeons are strongly of the opinion that the chance of recovery becomes less and less with every hour that is allowed to elapse after the perforation, and claim that the outlook is least favorable when the actual anatomic lesions of peritonitis have begun to develop. Not being a surgeon, I do not purpose to give a dogmatic opinion on these points, but I should certainly advise operation in all cases where the patients are not already manifestly moribund, and in which the operation itself is not likely to prove fatal by its effect on the nervous system and the heart.

Operation in Cases where Diffuse Peritonitis is Fully Developed.—There is an almost universal consensus of opinion to the effect that in marked cases of septic peritonitis all operative interference is useless and should not be undertaken. This applies more especially to so-called *sepsis peritonei*, in which there is a complete absence of exudation or even of marked inflammatory changes in the peritoneum. But even in cases where the inflammatory changes are more pronounced and in which there is a considerable quantity of exudate, but in which clinical features of sepsis are well marked, the operation must be considered useless, for in these instances the immediate danger does not lie in the local condition of the peritoneum, but in the general intoxication that has already taken place and that becomes clinically manifest in the reactions of the nervous and the cardiovascular systems. The operation can, of course, only affect the local condition of the peritoneum and not the systemic infection. A few isolated cases are on record, it is true, in which the evacuation of the peritoneal exudate seemed to have a favorable effect on the "septic character" of the clinical syndrome. In cases of pure sepsis peritonei, however, without an exudate, death can never be prevented by surgical intervention.

The conditions are somewhat different and, as a rule, more favorable in cases of acute diffuse peritonitis with free seropurulent, purulent, or sanious exudate in which the clinical picture is dominated by the local symptoms and by phenomena directly due to the presence of exudate in the peritoneal cavity, but in which there are no symptoms of a general toxemia. The prognosis, however, is very grave, even when an operation is performed ; but it has improved since operative interference has become more frequent in such cases. Recovery has followed operation on patients in an advanced collapse in whom the intestines were found to be "swimming in pus."

[In 45 cases collected by Dwight,[1] 15 recovered, several of whom were in a condition of profound collapse and toxemia ; the cases with streptococci seldom recovered. The object was to diminish the number of bacteria present in the peritoneum to the greatest possible extent by free drainage, and at the same time to avoid in every way diminishing the patient's powers of resistance.—ED.]

It is true that all the published cases are not so favorable as those

[1] E. W. Dwight, *Med. and Surg. Reps., Boston City Hosp.,* 1902.

recorded by Berruti (quoted by Winckel), who saved 12 out of 13 patients "with exudative and purulent" peritonitis by laparotomy. Operation, however, has been followed by such favorable results, particularly in cases of diffuse acute peritonitis starting from the appendix or the female genital organs, that it has a definite place in the treatment of this disease. These good results were, moreover, obtained in cases of several days' standing. A résumé of the literature on this subject up to 1896 will be found in the latest communications of Körte, Tietze, and Krogius. In the recurrent forms of multiple abscesses encysted in the peritoneal cavity the treatment is essentially surgical. The great reluctance formerly to adopt surgical measures is well brought out in F. F. Kaiser's historic review. The powerful advocacy of Kussmaul, who recommended surgical treatment thirty years ago in subacute and chronic forms of purulent peritonitis, had no effect on the prejudice against operative measures which then prevailed. The rapid strides of abdominal surgery in the last two decades has, however, revolutionized current opinion. Mikulicz first pointed out the true value of surgery in slowly progressive forms of fibrinopurulent peritonitis. Later observations have confirmed this absolutely.

Not being a surgeon, I must, as elsewhere, omit any technical description of all the surgical details of these operations, and refer my readers to surgical works for the necessary information.

ACUTE CIRCUMSCRIBED PERITONITIS.

INFLAMMATION strictly localized to a single and small area of the peritoneum is common, but in these cases peritonitis is often merely a concomitant result of the primary disease, and the clinical picture is entirely dominated by the symptoms of the original condition, which incidentally produces some localized peritonitis. In other cases this form of peritonitis develops into a well-defined clinical entity, which consists entirely of the symptoms of inflammation of the peritoneum. In the former category the structural changes in the peritoneum are confined to vascular injection, formation of fibrin, and possibly a little serous effusion, and the process remains strictly limited to the organs originally affected—*i. e.*, the liver, the gall-bladder, the spleen, the appendix, a coil of intestine, the female sexual organs, etc. In the latter category there is suppuration, with the formation of small or large collections of pus (perityphlitic, subphrenic, parametric, etc., abscesses).

As in every other disease, there are various transitional forms between these two varieties of localized peritonitis; the commonest and the most interesting form of localized peritonitis is that originating from the appendix, for it shows all transitions from a slight fibrinous exudate to perityphlitic abscesses, progressive purulent peritonitis, or even diffuse acute peritonitis.

In order to obtain a clear and comprehensive review of this subject the different forms of acute circumscribed peritonitis will be divided

into several groups. The grounds on which this classification is based are the morbid changes ; in other words, the extent of the inflammatory process in the peritoneum. I must add, however, that, as in the case of acute diffuse peritonitis, the clinical differences are the most important, and it is essential that the practical standpoint should be given the first place in this classification. The most important form is circumscribed purulent peritonitis or intraperitoneal abscesses. These abscesses are often so characteristic in their position and give rise to such typical clinical pictures that they have been considered and described as special diseases. This applies with special force to peritoneal inflammation originating from the vermiform appendix ; for this form of peritonitis ("la grande maladie de l'abdomen") is so characteristic and typical in its clinical aspects, and is of such great practical importance, that it is essential to describe it in full detail.

A careful study of the features of localized intraperitoneal suppuration, including its morbid anatomy, shows that in the great majority of instances there are a few points of selection from which the process starts. These two situations are, first, the neighborhood of the appendix in the right iliac fossa ; and next in importance and frequency, the region of the female sexual organs in the pelvis—in other words, perimetric and parametric abscesses ; the latter group is considered in detail elsewhere in this series, and will not be dealt with here. Further, on the grounds of their clinical characters and topography, a special group must be recognized of the abscesses found under the diaphragm (abscessus et pyopneumothorax subphrenicus). An account will also be given of the varieties of local peritonitis which present unusual features in their situation and origin. Progressive fibrinous suppuration of the peritoneum, a condition that may develop in any one of these forms of circumscribed peritonitis, forms a transitional stage, as we have seen, between the above conditions and acute diffuse peritonitis.

The non-purulent forms, although fairly frequent, are much less important than suppurative peritonitis.

APPENDICITIS.

Inflammation of the Peritoneum starting from the Intestine in the Right Iliac Fossa (Scolecoiditis ; [1] Perityphlitis).

Inflammations due to intestinal lesions often occur in the right iliac fossa, and either remain limited to the bowel or extend from it and involve the adjacent peritoneum. These forms of inflammation have at different times been called by a variety of names, according to their situation—*i. e.,* iliac phlegmon, typhlitis, perityphlitis, paratyphlitis, and

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[1] The word appendicitis, though only recently introduced into medical literature, bids fair, I am sorry to say, to become universally adopted. This term—a Latin word with a Greek termination—runs counter to all the dictates of etymology. I venture to suggest the name "scolecoiditis" here. The Greek anatomic term for the vermiform appendix is σκωληκοειδὴς ἀπόφυσις (from σκώλεξ, worm). "Apophysitis" can hardly be employed, since there are several apophyses in anatomic nomenclature. Scolecoiditis, on the other hand, completely covers the idea to be expressed—viz., "inflammation of the vermiform" appendix.

of late years appendicitis and periappendicitis. As in the great majority of cases the inflammation is localized in the neighborhood of the cecum (typhlon), the term "perityphlitis" is, generally speaking, very appropriate, and expresses no opinion as to the exact point of origin of the inflammation. As this term is universally employed in the literature, it will be used in this article to describe localized inflammation of the peritoneum in the right iliac fossa.

Pathologic experience based upon operation chiefly has shown that many of the cases of peritonitis in the right iliac fossa are solely due to disease of the appendix. For these cases, of course, a fresh word is required to describe accurately the actual conditions. The term "appendicitis," which may be applied to these cases, was introduced, so far as I know, by Fitz. It is, however, badly constructed from an etymologic point of view, and I should propose to substitute the name "scolecoiditis."

B. Grohé has recently written an exhaustive history of appendicitis (scolecoiditis) in which he describes the changes that have occurred in the views of different authors in the last few years; he refers to 1250 publications, but even this list could be added to; fresh researches and reports of cases, moreover, are constantly appearing. The following review will be confined to some of the main features brought out in Grohé's paper, and chiefly to those points which indicate important changes or advances in our views as to the pathology or the treatment of this disease.

The disease was first recognized during the second and third decades of this century; before that time scattered cases only were put on record, or cases were reported as appendicitis (scolecoiditis) which were erroneously interpreted as such, or vice versâ.

[The appendicular origin of the disease was, according to Pye Smith,[1] fully recognized by Addison in 1836 in his *Elements of the Practice of Medicine*, while John Burne[2] wrote a monograph on the subject in 1837. As an interesting antiquarian point it may be mentioned that the illness of Erasmus in 1530 has been thought to have been appendicitis,[3] and that Kelly[4] has drawn attention to a fecal abscess in the right iliac region containing round-worms recorded in 1642. The history of the disease in Great Britain and in France has recently been considered by Howard Kelly.[5]—ED.]

Wegeler, Louyer-Villermay, and Mélier, in 1827, described a few cases with autopsies in which the vermiform appendix was affected and was regarded as the starting-point of the disease. This correct view was, however, soon superseded by an inexact one, and nearly all the investigators who published similar cases studied the syndrome more

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[1] Pye Smith, *Brit. Med. Jour.*, 1902, vol. ii., p. 82; *A Text-book of Medicine* by Hilton Fagge and Pye Smith, 1902, vol. ii., p. 402.

[2] J. Burne, *Medico-Chir. Trans.*, London, 1837.

[3] Pye Smith, *Brit. Med. Jour.*, 1902, vol. ii., p. 638.

[4] A. O. J. Kelly, *Proc. Phila. Path. Soc.*, 1900, p. 126.

[5] Howard A. Kelly, *Glasgow Med. Jour.*, vol. lx., p. 81; *Bull. et Mem. Soc. de Chirurg.*, June 10, 1903, p. 632.

from a clinical than from an anatomic point of view, and believed that the cecum itself was more important as the primary focus than the vermiform appendix. Puchelt, who introduced the name of "perityphlitis," was largely responsible for this, while Albers is also to some extent to blame, for he made the first attempt to investigate the pathologic anatomy of the right iliac fossa and originated the name "typhlitis." Grisolle, it is true, entered a vigorous protest against the view that simple inflammation of the cecum following fecal accumulation could produce suppuration in the right iliac fossa, and insisted that perforation of the cecum or of the vermiform appendix was the most important factor; his views, however, were almost ignored, and had no influence on contemporary medical opinion.

Rokitansky later called attention to the possible relationship between the concretions occasionally found in the appendix and the anatomic changes of perforation and peritonitis which might be attributed to the presence of these bodies. Volz's paper on "Perforation of the Vermiform Appendix Produced by Enteroliths" established the principles that govern this accident and pointed out clearly the relationship between perforation due to this cause and the peritoneal lesions produced by disease of the appendix. Bamberger next contributed a collective investigation and a general summary of the clinical picture of this form of appendicitis. Oppolzer later proposed to separate perityphlitis— i. e., inflammation of the peritoneum covering the appendix—from paratyphlitis—i. e., phlegmonous inflammation of the retrocecal connective tissues.

The pathology and treatment of inflammations in the right iliac fossa received a fresh stimulus and were dealt with from a fresh point of view when antiseptic surgery was introduced in the early treatment of perityphlitis; an important controversy then began, in which the last word has not yet been said. American medical men (Fitz, R. F. Weir, McBurney, and others) were the pioneers in this, and were followed by medical men in many other countries, especially in Germany, England, France, Switzerland, and Sweden. Since this date—i. e., about fifteen years ago—the amount that has been written is enormous. The chief result of all the work done on this subject is, from the point of view of treatment, the recognition of the advisability of early operation, and, pathologically, the recognition of the importance of inflammation of the vermiform appendix.

Special attention need hardly be drawn to the fact that bacteriologic research has also been directed to perityphlitis; among the number of monographs on this subject the work of Tavel and his pupils deserves special mention.

ETIOLOGY AND PATHOGENESIS.

Disease of the appendix is, in an overwhelming number of cases, the starting-point of inflammation localized in the right iliac fossa. The importance formerly attached to the cecum itself cannot be regarded as justified; the clinical conception of stercoral typhlitis, formerly so often mentioned, must to a great extent be abandoned, and it is only

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very exceptionally that inflammation in the right iliac fossa can be referred to some process originating in the cecum itself.

This important change in our views depends on the results of post-mortem examinations and even more on the extended data obtained of late years by examination of the diseased parts *in vivo aperto*. The results of innumerable operations performed in the last fifteen years have conclusively proved that the vermiform appendix is responsible for the origin of the great majority of cases of perityphlitis. Although some authors still regard the cecum as the starting-point, their views are far from being in accord with the established facts, and at best are based on a few exceptional cases in which the cecum is primarily diseased.

There is thus a return to the views originally put forward (by Wegeler, Louyer-Villermay, Mêlier) as to the starting-point of this disease, and now, as then, the vermiform appendix plays the important part and the cecum a very subordinate rôle in the pathogeny of perityphlitis.

Stress need hardly be laid on the fact that the older writers did not overlook the etiologic relationship between diseases of the appendix and perityphlitis, as is clearly shown by the writings of Grisolle, Rokitansky, and Volz, which appeared half a century ago; at that time, however, the exact pathology of the changes going on in the appendix was not understood, and attention was exclusively directed to one possibility—*i. e.*, perforation of the appendix following the formation of enteroliths within the organ. The following arguments prove that nearly all the inflammatory conditions in the right iliac fossa originate from the appendix.

In the first place, postmortem data show that disease of the appendix, with a few exceptions, which will be referred to later, is the direct cause of the inflammation of the surrounding parts. A few figures only will be quoted in support of this statement: Matterstock, for instance, examined 146 cases of perityphlitis postmortem and found the appendix perforated 132 times; Einhorn found the same 91 times in 100 autopsies. It might be objected that these autopsies were performed only in specially severe cases; many cases of perityphlitis recover, and it might reasonably be urged that under these conditions the older view of a stercoral typhlitis might, after all, hold good.

This argument is, however, refuted by the results of the numerous laparotomies performed in the course of the last fifteen years for perityphlitis, in which the lesions, far from being advanced, were, on the contrary, in the earliest stages of development; the result of all these examinations in the living has been that the vermiform appendix may with absolute certainty be regarded as the primary focus in the great majority of cases of perityphlitis. According to MacMurtry, the cecum was not involved at all in 200 cases; in 130 cases on which he operated Sonnenburg found that in 129 the disease originated from the appendix; in his 74 cases Lennander found the appendix diseased 56 times; in 14 of the cases the appendix was not seen, but Lennander considers it

"highly probable" that in these 14 cases the appendix was also the point of origin of the trouble; it was only in another case where the appendix was completely absent and in 3 other of his cases that the starting-point of the peritonitis was elsewhere than in the appendix; this point will be referred to later on.

[After quoting Einhorn's[1] conclusion, based on 18,000 autopsies, that perityphlitis is of appendicular origin in 91 per cent., and of cecal origin and due to perforation of this viscus in the remaining 9 per cent., Treves[2] says that surgical experience would place the appendicular origin at a much higher percentage, and points out that perforation of the cecum may be secondary and not primary.—Ed.]

Observations made during operations by Roux, Rotter, Kümmel, Volkmann, Czerny, Borchardt (Krönlein), Meusser (Riedel), and by the modern pioneers in this subject—*i. e.*, the American surgeons, Fitz, McBurney, Wier, Monks, Keen, Fowler, Smith, and many others—fully support this view. Special stress should again be laid on the fact that in many of these cases the operation was performed early in the disease —*i. e.*, on the third, fourth, or fifth day after the appearance of the first clinical symptoms of perityphlitis. On these convincing arguments, and further on my own experience, of postmortem examinations and operations, I can fully indorse the opinion, strongly expressed by the majority of modern writers, that in the vast number of cases of perityphlitis—*i. e.*, in 90 per cent.—the clinical syndrome characteristic of this disease is primarily due to the vermiform appendix.

The question naturally arises, How is it that the vermiform appendix specially and more frequently than any other part of the intestine is subject to pathologic changes which in their turn are so apt to produce grave affections of the peritoneum? For the sake of clearness I am not at present referring to diseases like typhoid or tuberculosis, which affect the whole of the intestine and incidentally also, in individual cases, the appendix, but merely to ordinary appendicitis with perityphlitis which is a primary disease of the appendix. Why are these conditions so common? In order to answer this question satisfactorily it is necessary to recall the special anatomic conditions of the appendix. These anatomic facts have only lately been recognized, as the more minute anatomy of the organ has only recently been studied in detail. The important pathogenetic rôle of the appendix in the production of perityphlitis, which has also only recently been recognized, led, of course, to these investigations.

The average length of the appendix is 7 to 9 cm.; but it may vary greatly—in very rare cases the appendix may be completely absent or may consist of a short stump.

[It may be pointed out that cases in which absence of the vermiform appendix[3] has been assumed are capable of another explanation— viz., that it is entirely concealed in either the ileocecal or the subcecal

[1] Einhorn, *Münch. med. Wochenschr.*, 1891, p. 121.
[2] Treves, *Allbutt's System of Medicine*, vol. iii., p. 881.
[3] J. Ferguson, *Amer. Jour. Med. Sci.*, vol. ci., p. 61.

pouches (Lockwood and Rolleston).[1]   Even in a case where the cecum was completely absent, a vermiform appendix was present (Elliot Smith).[2]   Monks and Blake found that there was no apparent relation between the length of the body, the sex, or age of the subject, on the one hand, and the length of the appendix, on the other, except that children are apt to have appendices proportionately longer than adults. —ED.]

In other cases, on the other hand, it may be found to be 14 (Stuparich), 16 (Zuckerkandl), 19 (Sonnenburg), 21 (Ribbert), or even 23 cm. (Luschka, Lennander) long.   [In an examination of 641 cases Monks and Blake[3] found one appendix 24 cm. long; Trevor[4] recorded one 23.75 cm. long.—ED.]   As the appendix is, moreover, only about as thick as a goose-quill, its lumen is proportionately small, and the disproportion between the transverse (4 to 5 mm.) and the longitudinal diameter may be considerable.

[According to Bryant,[5] the lumen of the appendix is larger in the male than in the female.—ED.]

In order to make the subject clear, a few remarks on the position of the appendix will be given here.   This, all writers agree, is by no means constant.   Most frequently the appendix arises from the posterior and median surface of the cecum, and passes freely into the abdominal cavity, being fastened and fixed to a certain degree by a mesenteriolum. A point situated about 6 cm. from the anterior superior spine of the ilium, on a line drawn from this spine to the umbilicus, is said to indicate the position of the appendix (MacBurney's point), or, rather, the spot where the appendix comes off from the cecum.   In order to convey some idea of the great variations that exist in the position of the appendix, the following statements by different authors may be quoted :

Lafforgue, in 200 examinations made indiscriminately, found the appendix pointing downward most frequently (41.5 per cent.), directed in a lateral direction in 26 per cent. of the cases, directed inward in 17 per cent., and upward in 13 per cent.   Bryant, in 144 autopsies, found the appendix directed inward 34 times, behind the cecum 32 times, directed downward and inward 28 times, passing toward the pelvis 21 times, and in other positions 29 times.   According to G. J. Turner, the appendix in 105 autopsies was free in the abdominal cavity 83 times, in the pelvis 51 times ; running transversely across the psoas toward the promontory of the sacrum 20 times, and in the remainder in a variety of other directions.

Sudsuki, in 500 cases taken indiscriminately, found the appendix directed downward in 183, upward in 176, and transversely in 141 ; moreover he found it situated partially or completely in the true pelvis in 168 of the 500 cases.

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[1] C. B. Lockwood and H. D. Rolleston, *Jour. Anat. and Phys.*, vol. xxvi., p. 131.
[2] Elliot Smith, *ibid.*, vol. xxxviii., p. 32.
[3] G. H. Monks and J. B. Blake, *Boston Med. and Surg. Jour.*, vol. cxlvii., p. 581.
[4] R. S. Trevor, *Jour. Anat. and Phys.*, vol. xxxv., *Proc. Anat. Soc.*, vol. xlii.
[5] Bryant, *Annals of Surgery*, 1893.

[In many cases the appendix points toward the spleen and lies under the inferior layer of the mesentery; Treves[1] regarded this as the most usual position. The next commonest position, according to Treves and Hawkins, is subcecal. The pelvic position of the appendix was found in 15 out of 109 consecutive operations (Lockwood),[2] and on 15 of 100 bodies examined by Hawkins.[3] Tubby[4] speaks of the pelvic position as specially common in children. From an examination of 572 cases, Monks and Blake found "downward and inward" the commonest position (179 cases), then behind the cecum (104), downward (79), inward (62), upward (52), upward and inward (39).—ED.]

It would be quite useless to enumerate all the possible variations in the position of the appendix that have been described, but a few general data of some clinical importance must be given. When the appendix passes downward, it usually enters the pelvis; in male subjects it lies in contact with the bladder and rectum, and in women with the uterus and the right ovary. When the appendix passes inward toward the spine, it comes in contact with loops of the small intestine. When it extends outward, it curls around the cecum, either in front or behind and below. When it passes upward, it is found on the posterior surface of the cecum. In addition, there may be abnormalities in the position of the cecum, the colon, the sigmoid flexure (compare p. 335), which determine other anomalies in the position of the appendix. The appendix is occasionally found in hernial sacs.

[The appendix may, in exceptional instances, pass into and become strangulated in retroperitoneal hernia in the peritoneal fossæ around the appendix. Less rare are the cases where the appendix is found in the sac of an external hernia. McAdam Eccles[5] has tabulated cases—(a) where there are other viscera in the sac and the appendix is acutely inflamed; (b) where the appendix is alone in the sac, and (1) is neither acutely inflamed nor strangulated; (2) where it is acutely inflamed; (3) where it is acutely strangulated.—ED.]

It is worth mentioning that when the appendix is particularly long and extends up behind the colon, it may pass up as high as the kidney or liver; a long appendix passing toward the median line may reach as far as the umbilicus, and may even extend upward along the rectus abdominis of the left side, and pass beyond the left margin of this muscle. In one case Lennander found the cecum with an appendix 15 cm. long in the left hypochondrium close to the spleen.

These data, of course, have no bearing on the pathogenesis of appendicitis, and are chiefly of importance in connection with the diagnosis, since an exact knowledge of the position of the appendix makes an accurate localization of perityphlitic or, to be more exact, of periappendicular, abscesses possible, and in this way makes their surgical treatment safer and more exact. In order to avoid a repetition of these

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[1] F. Treves, *Hunterian Lectures, Royal College of Surgeons*, 1885, p. 43.
[2] C. B. Lockwood, *Appendicitis*, p. 9.
[3] H. P. Hawkins, *Diseases of the Vermiform Appendix*, p. 17.
[4] A. H. Tubby, *Clinical Jour.*, vol. xxii., p. 411.
[5] McAdam Eccles, *St. Bartholomew's Hosp. Reps.*, 1896, vol. xxxii., p. 93.

anatomic points in the subsequent paragraphs on diagnosis and treatment, this part of the subject has been dealt with here.

The histologic structure and the nutrition of the appendix are, however, of the greatest importance, for nutritional changes and alterations in its normal histologic structure determine the primary diseases of the appendix.

Generally speaking, the appendix has the same structure as the large intestine, but differs in a few histologic points which are of some practical importance. In the first place, the mucous membrane of the appendix during the first years of life contains an extraordinary number of closely packed lymph-follicles, which persist up to the thirtieth year, when they become smaller and, therefore, appear to be more widely separated (Ribbert). In exceptional cases atrophic changes which are physiologic may develop before the twentieth year, while in other instances they may still be absent after the age of thirty.

From a pathogenetic point of view some importance has been attached to the structure called Gerlach's valve, which is a reduplication of the mucous membrane of the appendix near its origin from the cecum. [Treves [1] says it does not exist.—Ed.] The physiologic rôle of this valve, it is true, has not yet been established, and many authors state that it is not constantly present. At the same time some importance cannot be denied this fold, for it certainly makes it more difficult for the contents of the cecum to enter the appendix, and also renders the exit of any material that may have got into the appendix more complicated.

A large number of investigations have been published on the relation of the peritoneum to the appendix, and this fact alone shows that the relation is not constant, but varies greatly. In the great majority of cases the appendix is completely surrounded by peritoneum, but in a certain proportion of cases it is only partially covered by peritoneum, and in such cases the uncovered portions of the organ are, as a rule, in direct contact with the retroperitoneal connective tissues of the iliac fossa.

The appendix has a mesentery of its own (mesenteriolum, meso-appendix), which is not, by any means, constantly present : at least various authors state that it is sometimes present and sometimes absent ; Kelynack, for instance, found it in all the cases he examined, while it was absent in 100 of 2000 bodies examined by Ferguson.

[In 243 cases the mesentery was completely absent in 16, or 6.5 per cent. ; in half the cases examined the mesentery reached to the extreme tip or close to the end (Monks and Blake [2]). Lockwood says the mesentery is one of the most variable structures in the body.—Ed.]

It rarely reaches right up to the tip of the appendix, and is usually only attached to about one-third or two-thirds of the length of the appendix. It has some influence on the shape of the appendix : thus when it is very short or completely absent, the appendix may stretch

[1] Treves, *Allbutt's System of Medicine*, vol. iii., p. 880.
[2] G. H. Monks and J. B. Blake, *Boston Med. and Surg. Jour.*, vol. cxlvii., p. 581.

out perfectly straight; on the other hand, when the mesenteriolum is relatively too short—*i. e.*, as compared to the length of the appendix—the latter may be bent.

[In a case described by Rolleston,[1] the plica vascularis (Lockwood[2]), or superior fold of the mesorchium, constricted the appendix, which passed through it, and the distal portion showed catarrhal inflammation. The plica vascularis normally runs downward from the appendix or its mesentery to the internal abdominal ring; it carries blood-vessels, and in the female, as the appendicular ovarian ligament of Clado, has been thought to be the means of infection passing between the broad ligament and the appendix (Clado[3]).—Ed.]

The blood-supply of the appendix is important; it is derived from the superior mesenteric artery, an insignificant collateral blood-supply coming from the vessels of the cecum. The main artery of the appendix runs along the free margin of its mesentery and sends branches at right angles into the body of the organ; the mesentery itself is filled with a fine network of small arteries, veins, lymph-vessels, and numerous nerves. When the mesentery is absent, the appendicular artery runs in the peritoneal covering of the appendix. Sometimes, in exceptional cases, the artery runs to the tip of the appendix without sending any branches into the substance of the organ.

[Lockwood[4] has specially studied the arterial supply of the appendix, and shows that it has two sources of arterial vessels which are both derived from the posterior ileocecal branch of the ileocolic artery. The posterior ileocecal artery divides into cecal and appendicular branches. From the cecal artery branches also go to the appendix. One of these, as a rule, runs along the appendix at its junction with the meso-appendix, and anastomoses with the meso-appendicular branches of the appendicular branch of the posterior ileocecal artery. When, however, there is no meso-appendix, the nutrition of the appendix is entirely dependent on the cecal branch, and is, therefore, precarious. As a rule, the appendix receives its most abundant blood-supply from the appendicular branch of the posterior ileocecal artery. The appendicular artery runs into the meso-appendix and divides into three branches: the largest runs along the free border of the meso-appendix; the other two reach the appendix at intervals of half an inch. Owing to this arrangement of the arteries, the portion of the appendix with a meso-appendix has the best blood-supply, while the free end has the least and most precarious.—Ed.]

The nerves supplying the appendix are derived from the superior mesenteric plexus.

These anatomic facts are important in obtaining a proper knowledge of the diseases of the appendix, and are partly responsible for some of the dangers that the appendix is exposed to; since the nutrition and struc-

[1] H. D. Rolleston, *Jour. Anat. and Phys.*, vol. xxxii., p. 64.
[2] C. B. Lockwood, *Medico-Chir. Trans.*, 1886, vol. lxix., p. 502.
[3] Clado, *Compt. Rend. Soc. Biol.*, 1892, vol. iv., p. 133.
[4] C. B. Lockwood, *Appendicitis*, p. 27.

tural arrangement of the appendix and that of the rest of the intestinal tract are different, a number of injurious factors which are counteracted physiologically in the latter cannot be counteracted in the appendix, for its resisting powers are not so great. It follows that the results may be dangerous to the whole organism. In addition, the peculiarities of the appendix give rise to mechanical conditions, which in their turn favor the occurrence of dangerous complications which are impossible in the case of other parts of the intestine. Before describing these various factors, however, I must briefly mention one other quite unique circumstance about the appendix, which must be carefully borne in mind and considered in an attempt to interpret the anatomic changes that may occur in this organ.

The vermiform appendix of man is now regarded as a functionless portion of the intestine, and is considered by most investigators as a rudimentary phylogenetic organ. The fact that the appendix is a rudimentary organ carries with it certain involution processes, the most important of which is obliteration of the vermiform appendix. Wölfler, and particularly Ribbert, E. Zuckerkandl, and Sudsuki, conclude from the histologic conditions that this obliteration, when typical, is not the result of inflammatory, but of involution, changes in an organ that has become functionless. There are no signs of previous inflammatory changes of the organ in these cases, and, on the contrary, all the histologic appearances and other facts clearly show that there are atrophic changes in the appendix. In 400 cases Ribbert found the appendix either totally or, much more frequently, partially closed in 99, or in 25 per cent.; Zuckerkandl found the same 55 times in 232 cases—*i. e.*, in 23.7 per cent.; and Sudsuki 113 times in 500 cases—*i. e.*, 22.6 per cent. Both sexes are affected nearly equally. It is a remarkable fact that this atrophy increases in frequency in proportion to the age of the subjects. In 117 appendices of children under ten, Sudsuki found only 1 case of obliteration, and that only partial. Ribbert found the first signs of the obliteration in the fifth year of life, while in subjects over sixty years of age the change was seen in more than 50 per cent.

The obliterative process is occasionally universal : in about one-half of the cases only one-quarter of the appendix is obliterated, while the remaining cases show an intermediate amount of obliteration. The shorter the organ, the greater the tendency to atrophic obliteration ; the longest appendices are least frequently involved. Partial obliteration occurs most frequently, in fact, in the great majority of the cases, in the distal extremity of the organ. External examination hardly suggests the existence of this condition, and it is impossible to be sure whether the lumen of the appendix is patent or not, except by slitting the appendix up. It is very remarkable that the great majority of appendices undergoing obliterative atrophy do not show peritoneal adhesions.

The following is a summary of Zuckerkandl's careful work on this process : "The mucous membrane undergoes atrophy, the glandular structures desquamate, and the opposing layers of the inner surface of the organ grow together. At the same time, or occasionally before this

time, the submucous coat undergoes thickening and fat accumulates in this portion of the wall. The muscular coat does not necessarily show any changes, but may also reveal an accumulation of fat. When the obliteration is complete, the lymphoid tissue has disappeared, and the connective-tissue framework of the mucosa that remains behind, and the submucosa, from which, in the mean time, the accumulation of fat has disappeared, contracts." Ribbert remarks in this connection that the typical repetition of exactly the same appearances in a number of appendices; the existence of a central uniform strand of connective tissue developed from the mucosa; the absence of all irregularities and of any signs of past inflammation, especially the absence of any cicatricial tissue, clearly prove that the obliterative process is not the result of any inflammatory change in the appendix.

The involution process of obliteration must be regarded as a favorable event—in fact, the most fortunate thing that can happen to a human being so far as this organ is concerned. An appendix, when it is completely obliterated, cannot possibly be affected by the same injurious influences and changes which have so harmful an effect on the " normal " organ, and are fraught with such disastrous results to the whole organism.

In contradistinction to these views, earlier writers attributed the so-called involution changes to pathologic or inflammatory processes; lately, again, several French observers support this view (Pilliet, Monod, Vanviers, etc.), and more particularly Riedel. The latter describes the changes under the term "appendicitis granulosa." Between the tubular glands small-celled and extremely vascular granulation tissue develops, which separates the glands and finally causes them to disappear; in this tissue small hemorrhages are often seen. This form of appendicitis comes on insidiously and slowly, and, as a matter of fact, causes obliteration of the organ without giving rise to any acute attack. Riedel disputes the process of simple involution on the ground that the appendix does not disappear in its entirety, but is replaced by a good deal of granulation tissue, which makes its appearance between the gland tubules. In addition, he urges that the same change is found in quite young children who have repeatedly suffered from slight attacks of appendicitis. These differences of opinion are merely stated here without any attempt to express a dogmatic decision on the point; at the same time, Riedel's conclusions appear too arbitrary in the face of histologic and other factors pointing to non-inflammatory involution changes.

As already mentioned, the predisposing factors that determine the more frequent involvement of the appendix as compared with other portions of the bowel must be sought in the anatomic peculiarities just enumerated; in this way alone it is possible to explain why peculiar primary diseases of the appendix are so common, for it is clear that those agencies that are usually responsible—*i. e.*, bacteria and masses of fecal matter—are as common in other portions of the bowel as in the appendix, and, as a matter of fact, they must enter the appendix from

the bowel. The difference is merely this, that they can readily produce harm in the appendix, whereas in other portions of the bowel this can occur only under special circumstances. In other words, appendicitis (scolecoiditis) and its results are not produced by specific morbific influences, but there are special features in the anatomy and physiology of the appendix which favor the development of serious consequences in the appendix, while other parts of the intestine, although exposed to the same influences, remain immune.

American and English authors were the first to suggest that the large amount of lymphoid tissue in the appendix is responsible for the frequency with which it is attacked by morbid processes ; the appendix, in fact, contains as much lymphoid tissue as the tonsils. The latter are particularly susceptible to disease, owing to the fact that they are specially prone to bacterial infection (for reasons which will not be gone into here). The appendix may similarly be exposed to the same dangers, for it is as close to the contents of the bowel and the numerous bacteria contained therein as the tonsils are to the mouth and its equally profuse bacterial flora. Sahli is, therefore, quite justified in speaking of an "angina of the vermiform appendix." It is certainly remarkable and probably more than a coincidence that the greater number of diseases of the appendix and the majority of cases of perityphlitis occur within the first half of life—*i. e.*, before the thirtieth year ; this observation is interesting because Ribbert (see above) has shown that from this time on the follicles of the appendix begin to atrophy.

Other predisposing factors must be sought in the peculiar formation of the organ ; in the fact that the lumen of the appendix is so narrow that the slightest swelling of its walls leads to obliteration of its lumen and makes the expulsion of any dangerous contents that may be present in the organ (bacteria, secretion, feces) difficult or impossible. Kinking or distortion of the appendix probably has the same effect.

The same cause—viz., a special predisposition on the part of the appendix—explains the family tendency to the disease which seems actually to exist, for it often happens that several members of a family suffer from perityphlitis. In all probability this may be correlated with peculiarities in the anatomic form or situation of the appendix that are common to several members of the same family. [Forchheimer[1] has published the history of a family of 22 members, 7 of whom had appendicitis. Treves[2] quotes a family with 5 cases and says that the association of this disease in families is not so often that of parent and child as of brother and sister.—ED.] This, of course, does not apply to a simultaneous outbreak of appendicitis in several members of the same family, which, according to modern ideas, is probably a kind of epidemic due to bacterial infection.

There also seems to be a special sexual predisposition to appendicitis, since, to judge from the statistics that have been accumulated on this subject, a far greater number of men suffer from the disease than women.

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[1] Forchheimer, *American Medicine*, October 3, 1901, p. 527.
[2] F. Treves, *Allbutt's System of Medicine*, vol. iii., p. 895.

Contributions to the subject agree so uniformly, to cite only a few names,—*e. g.*, Bamberger, Kümmel, Lennander, Matterstock, Maurin, Paulier, Pravaz, Rotter, Sonnenburg, Volz, to which other names might be added, besides one's own personal experience,—that it is quite unnecessary to give statistical tables. To quote an old table of Matterstock's : Of 1030 cases, there were 733 men and 297 women. Particular stress might be laid upon a table of the same observer, in which he gives 51 boys and 21 girls in 72 children from seven months to fifteen years of age.

[Treves put the rates at males 78, females 22, per cent. ; in 224 cases collected by G. N. Pitt[1] from Guy's Hospital there were 160 males and 64 females. It has been shown by Borje[2] that there is no evidence that pregnancy disposes to appendicitis. In women an outbreak of appendicitis is frequently coincident with the menstrual period, and this association is far too common to be merely accidental (Treves[3]). Possibly, in some cases, infection is conveyed from the right ovary to the appendix.—ED.]

Some light might be thrown upon this otherwise incomprehensible want of proportion in the incidence of appendicitis in the two sexes if van Cott's views, which will be considered below, are correct ; he believes that appendicitis is due to disease of the blood-vessels of the organ. He states that these changes partly lead to alterations of the tissues and in part prepare a suitable nidus for the arrest and multiplication of pathogenetic organisms, or prepare the ground in other ways for the action of certain factors that may do harm (fecal concretions, etc.). Lastly, he states that in men this disease of the appendicular vessels is commoner than in women, because the latter have a collateral circulation from the sexual organs, which is absent in men.

The same correspondence exists in regard to the age of the patients. All statistics show that perityphlitis is most frequent between the ages of ten and thirty ; in the first decennium of life the disease is not common, and after the thirtieth year its incidence diminishes, while after forty perityphlitis occurs only in isolated cases. This, of course, refers only to first and not to recurrent attacks. The age incidence of perityphlitis, as estimated by various writers, is as follows :

	Matterstock.	Fitz.	Sonnenburg.	Nothnagel.
	Among 474	228	130[4]	130[5]
0–10 years	46	22	14	1[6]
11–20 "	143	86	33	44
21–30 "	158	65	43	57
31–40 "	72	34	15	14
41–50 "	30	8	11	7
51–60 " . . . . /.	18	11	2	4
61–70 "	5	1	4	2
71–80 "	2	1	1	

[1] G. N. Pitt, quoted in Fagge and Pye Smith's *Text-book of Medicine*, 1902, vol. ii., p. 405, fourth ed.
[2] *Brit. Med. Jour.*, 1903, vol. ii., p. 1355.   [3] F. Treves, *ibid.*, 1902, vol. i., p. 1589.
[4] In seven cases the age is not given.   [5] In one case the age is not given.
[6] Children under ten years are rarely received in my clinic.

[Appendicitis is very rare under two years of age ; Crozer Griffith [1] has collected 15 cases under this age, the youngest being two, Pollard's and Goyens', of six weeks. Cases in infants seven weeks old have been recorded by Demme, Elder,[2] and Blumer and Shaw.[3] More recently, Porak and Durante [4] have recorded fatal appendicitis and peritonitis in an infant three weeks old. The disease may occur at the other extreme of life : Walters [5] successfully removed the appendix from a woman aged seventy-eight.—ED.]

A closer correspondence of figures than this can hardly be imagined, and it is, therefore, unnecessary to do more than to mention that an extensive collective investigation, carried out by Sahli among Swiss medical men, showed a similar preponderance of cases in young subjects. The only statistical data at variance with those quoted above are those of Einhorn, who denies both the greater incidence of the disease in young subjects and in the male sex. In regard to these particular statistics, I agree with others that this single series of 100 cases, on which Einhorn's statistics are based, can have no weight as compared with the much larger figures collected by other writers, especially as to the age of the patients with this particular disease.

It is clear, from the anatomy of the appendix, that differences in the age of the patients with appendicitis must be assumed *a priori* because the development and the atrophy of the lymphoid follicles occur, as we have seen, at certain definite periods of life. The conclusion is almost inevitable that a certain relationship must exist between the amount of the lymphoid tissue of the appendix and the frequency of appendix disease ; exactly what this relation is and the rationale of the connection will be considered later. The chief difficulty in accepting the view is that the disease is relatively so infrequent in the first decade of life, a period in which the lymphoid tissue is fully as well developed as in the second and third decades ; the question arises, therefore, why is perityphlitis not just as common in the first decade ? An answer to this question may in some degree be obtained by the following considerations :

The factors already enumerated partly explain, at any rate, the marked predisposition of the appendix to disease. What are the proper and exciting causes of this disease?

For reasons of historic interest, and not because it is the most important, the rôle of **foreign bodies and of fecal concretions in the appendix** will be described first. The old view that foreign bodies, especially fruit-stones or other articles, such as needles, hairs, pearls, bones, gall-stones, etc., very frequently caused serious changes in the appendix and perityphlitis has now been abandoned. These bodies are only exceptionally found in the appendix. The old idea that fruit-pips frequently entered the appendix is explained by the fact that

[1] J. P. Crozer Griffith, *Univ. of Penn. Med. Bull.*, October, 1901.
[2] J. M. Elder, *Montreal Med. Jour.*, vol. xxx.
[3] G. Blumer and H. L. K. Shaw, *Archives of Pediatrics*, August, 1901.
[4] Porak and Durante, *Compt. Rend. de la Soc. d' Obstet. de Gynéc. et de Pœd.*, Paris, December, 1902.
[5] A. R. Walters, *Brit. Med. Jour.*, 1903, vol. i., p. 1258.

fecal concretions were often taken for these bodies; on superficial examination it is, as a matter of fact, very easy to make this mistake. Even assuming, however, that fruit-pips actually do enter the appendix, they would not necessarily do any harm, for it is now known that foreign bodies of considerable size may remain in the appendix without doing much damage; in a boy, for instance, of seventeen months, who died of phosphorus poisoning, a lead button as large as a "pfenning" [somewhat smaller than a ten-cent piece.—ED.] was found in the vermiform appendix, without having produced any appreciable changes of the mucosa (Matterstock). It is only in a very small minority, indeed, of the cases that foreign bodies produce perityphlitis. In a case published by Siegel the lumen of the appendix of a child who had been treated with phosphate of calcium for rickets was completely filled with concretions of calcium salts.

[In 1400 cases of appendicitis collected from various sources in ten years—1889–1898—by Mitchell,[1] there was 7 per cent. of foreign bodies; in 700 of these cases in which a definite statement as to the nature of the foreign bodies is made, it was a fecal concretion in 45 per cent. He has also collected 33 cases in which pins were found in the appendix, and 2 in which the cecum was perforated by a pin; in no less than 8 of these 35 cases there was secondary suppuration in the liver. It is remarkable that in only one case was there a history that a pin had been swallowed. Among other exceptional bodies found in the appendix and sometimes forming the nucleus of a concretion, shot, a bullet (Mitchell), anthracite coal (Lathrop),[2] a thorn (Rolleston),[3] bristles of a tooth-brush (Wiesswange),[4] glass (Lockwood),[5] and egg-shells (Boldt)[6] may be mentioned.

Thread-worms are common in the vermiform appendix of children, a point especially investigated by Still.[7] Round-worms, segments of tape-worm (Davis,[8] A. Martin,[9] Mitchell), and bilharzia (Cureton and L. Webb[10]) have also been found in the appendix.—ED.]

Fecal concretions, on the other hand, are more important, for they certainly play a definite rôle in the pathogenesis of appendicitis and often lead to dangerous conditions, especially perforation.

The idea that the fecal concretions always entered the appendix preformed from the cecum is being discarded by modern investigators, and only very few writers, among them Talamon, still adhere to this view. Anatomically, the entrance of these concretions would be very difficult during the first years of life, for the orifice of the appendix is narrow, and, further, Gerlach's valve would also prevent the entrance of con-

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[1] J. F. Mitchell, *Johns Hopkins Hosp. Bull.*, Jan.–March, 1899, p. 35.
[2] Lathrop, *Philadelphia Med. Jour.*, July 7, 1900.
[3] Rolleston, *Trans. Path. Soc.*, vol. xliii., p. 71.
[4] Wiesswange, *Centralbl. f. Gynäk.*, May 9, 1903.
[5] C. B. Lockwood, *Pathology and Treatment of Appendicitis*, p. 75.
[6] Boldt, *Amer. Jour. Obstet.*, March, 1903.
[7] G. F. Still, *Brit. Med. Jour.*, 1899, vol. i., p. 898.
[8] G. G. Davis, *Proc. Philadelphia Path. Soc.*, 1900, p. 126.
[9] A. Martin, *Bull. Soc. de Chirurg.*, Paris, 1903.
[10] Cureton and Law Webb, *Lancet*, 1899, vol. i., p. 156.

cretions from the cecum, as has been proved by some experiments on this point. It is, therefore, generally accepted now that the great majority of fecal concretions are formed within the lumen of the appendix.

In a number of examinations made after death without any special selection, the appendix will be found to be either completely empty or, more frequently, to contain small quantities of pultaceous or thin fecal material. Sudsuki, in 500 autopsies, found fecal contents in more than one-half. The actual frequency with which small fecal masses enter the appendix only to be expelled again by the muscular contraction of the appendix is quite an open question; definite factors, such as distortion, twisting, kinking, abnormal length or position of the organ, abnormalities of Gerlach's valve (said by several authors—e. g., Glaeser, Sudsuki—to be functionally useless), or occasionally muscular weakness may all make it difficult for fecal material which has once passed into the appendix to get out. I agree, however, with Ribbert that the presence of fecal matter in the appendix may be a source of danger even in the absence of these special predisposing factors and that the latter are not essential.

The following changes occur in a mass of fecal material which remains in the appendix : It becomes dry and hard from absorption of water through the walls of the appendix, and subsequently may again increase in size; according to Ribbert, this enlargement occurs in the following way : It is only the smaller concretions which consist entirely of feces; the larger ones are composed of a nucleus of fecal matter surrounded by a shell of mucus containing a few cells (leukocytes), the nuclei of which, especially those on the surface, can still be stained. This mucus, derived from the glandular tubules, accounts for enlargement of concretions impacted in the appendix.

The circumference of these calculi varies : the larger ones, especially those which produce perforation of the appendix, are as big as a hazelnut; Volz gave a very good description of these concretions. Their shape also varies : they may be round, oval, or elongated into rod-like structures or perfectly cylindric; their surface may be smooth, nodular, uneven, or angular.

[The concretions contain fecal material largely soluble in ether, phosphate and carbonate of lime, sometimes salts of magnesia, traces of chlorids and sulphates, and very rarely cholesterin, which are all cemented together by mucus. Dieulafoy[1] draws attention to the stratified concentric structure of the concretions, and compares the process, which he calls appendicular lithiasis, to the formation of calculi in the gall-bladder. Lockwood[2] believes that fecal concretions are the result of bacterial growth, which always precedes the formation of the concretion, and that this preliminary stage is associated with ulceration and bacterial invasion of the mucosa. He further shows that the concretions are largely inspissated masses of bacteria (in addition to containing

[1] G. Dieulafoy, *Clinique Médicale de l' Hotel-Dieu*, Paris, 1898, vol. i., p. 332.
[2] C. B. Lockwood, *Appendicitis, Its Pathology and Surgery*, 1901, p. 75.

inorganic salts, etc.). In 65 appendicular concretions examined by Rochaz,[1] only 3 were round, almost all being elongated and cylindric.—Ed.]

They occur in different parts of the appendix, but usually at the distal extremity. When the concretions are fairly large, the appendix may often be seen to bulge before it is opened. There are sometimes two, three, even as many as five, concretions in the appendix [which may then be facetted (Rochaz).—Ed.].

Varying statements are made as to the absolute frequency with which these concretions are found: Ribbert, among 400 appendices taken without any selection, found concretions in 38, or in about 10 per cent.; in some of these cases there were several calculi. Perforation had occurred only in one case. Sex had no marked influence. Influence of age: in the 400 cases there were about 50 children under five years, none of whom had any concretions; between the ages of five and twenty years there were about twice as many calculi as in subjects above twenty years of age.

The great absolute frequency of fecal concretions in the appendix as compared to their occurrence in other portions of the bowel is readily explained by the form of the organ, the narrowness of its lumen, the frequency of strictures in its lumen, and kinks and twists in the appendix; lastly, it must be remembered that the peristalsis available for propelling any abnormal accumulation onward is weaker in the appendix than in any other part of the intestinal tract; in addition, the peristaltic power of the cecal walls is so much stronger than that of the appendix that fecal matter is easily forced into the appendix from the cecum.

Medical men differ greatly in their views as to the real importance of fecal accumulations in the appendix in the production of appendicitis and of perityphlitis. That soft fecal matter may remain in the appendix without doing any harm is clearly shown by its presence after death in cases which have never had any symptoms of disease of the appendix during life. Even hard fecal masses, as long as they are not too big, may remain in the appendix without producing any bad results. Many recent writers go so far as to state that fecal concretions play no rôle whatsoever in the production of diseases of the appendix, and that they exert a determining influence on affections of the appendix only in a secondary way—*i. e.,* by influencing the course of lesions of the organ which are primarily due to some other cause; thus they claim that fecal concretions may act deleteriously on a mucous membrane after it has been modified by the action of bacteria. It is clear that a fecal concretion must exert pressure on the wall of the appendix, especially when the latter is swollen, and may readily occlude the lumen of the organ and thus produce serious results. I cannot, however, indorse the view that fecal concretions only play a secondary part; in fact, the direct observation of a number of cases, one of which will be quoted by way of illustration, proves exactly the opposite. A lapa-

[1] Rochaz, *Thèse,* Lausanne, 1895, quoted by Dieulafoy, *loc. cit.*

rotomy was performed in a man of middle age on account of symptoms produced by a chronic peritoneal adhesion extending from the stomach to the anterior abdominal wall; on examining the abdominal viscera the surgeon found the vermiform appendix free, of medium length, and externally perfectly normal and free from any sign of perityphlitis, but distended at its distal extremity by a resistant body. The appendix after removal was opened while still warm and while the muscular fibers were still contracting; the mucous membrane was pale and perfectly normal in appearance, except at the distal extremity in the immediate vicinity of a hard, round fecal calculus the size of a cherry-stone, where it was injected and light red in color. An observation of this kind conclusively shows, to my mind, that the localized catarrh which was unquestionably due to the presence of the fecal concretion would have ultimately led to other and probably grave results. The anatomic lesions present in cases such as the one just described forces us to the conclusion that hard and large fecal concretions may lead to morbid changes in the appendix. It will be seen below that bacteria produce further consequences and precipitate the development of the characteristic changes; but that is another matter and does not detract from the truth of the view that fecal concretions *per se* are capable of doing harm and of starting pathologic processes. In other words, enteroliths are the primary factor in the pathologic processes, and act mechanically by their pressure effect. In the last few years the importance of fecal concretions has been greatly diminished by modern bacteriologic ideas; lately, however, it has been brought up again, especially by Riedel, who believes that concretions develop in the healthy appendix and secondarily give rise to inflammation, especially of a malignant type. Beck and others state that they nearly always found them in gangrenous appendicitis. [Lockwood's views have already been referred to.—Ed.]

There is no doubt that hard fecal masses can set up changes, such as inflammation and ulceration, in other parts of the bowel, and if this is so, how much more readily must they act in the appendix, in which the mechanical effect of pressure must be so much more marked, both because space is necessarily limited from the anatomic peculiarities of the appendix and because its resistance is diminished.

The statistics quoted above show the great importance of fecal concretions in the pathology of perityphlitis. In a collection of recorded cases Renvers finds that among 459 cases of perityphlitis there were fecal concretions in 179 and foreign bodies in 16; in some of the other cases the presence of these bodies was presumably overlooked, so that they were present in about one-half of the cases; Fitz found them in 47 per cent.; Matterstock found fecal concretions in 63 out of 146 cases examined postmortem.

[Treves [1] considers that concretions are probably present in about 30 per cent. of the cases of perityphlitis. In 216 cases of acute appendicitis analyzed by Deaver,[2] calculi were present in 35, or 16 per cent.,

[1] F. Treves, *Allbutt's System of Medicine,* vol. iii., p. 885.
[2] J. B. Deaver, *A Treatise on Appendicitis.*

being most frequent in the ulcerative ; in 120 cases there were calculi in 28, or 22.3 per cent.—Ed.]

These figures prove, on the one hand, that coproliths play a very important rôle in the pathogenesis of appendicitis, and, on the other hand, that they are absent in about half the cases of appendicitis. It is, therefore, clear that in addition to coproliths other factors must play an important part in the production of appendicitis and perityphlitis.

Bacteria.—The additional factor is bacterial activity, and some authors, in fact, go so far as to claim that bacteria are the sole cause of this disease. This exclusive point of view, as has just been seen, does not agree with the facts of the case, for the demonstration of bacteria in the appendix—*i. e.*, in a portion of the bowel that normally contains the greatest number of micro-organisms—by no means proves that these microbes are concerned in the production of the inflammations of this organ. At the same time, the important part played by bacteria in the genesis of perityphlitis cannot be gainsaid, and this factor, which was first pointed out by Talamon, is now universally recognized.

The majority of bacteriologists agree that no one or specific micro-organism is the cause of ordinary appendicitis—this, of course, does not apply to the rare cases of tuberculous, typhoid, or actinomycotic disease of the organ. The bacteria normally present in the colon, and the micro-organisms which produce peritonitis, are also found in perityphlitis. (For detailed data the reader should refer to the section on the Bacteriologic Etiology of Peritonitis in General.) A brief account of the special bacteriologic pathology of appendicitis and perityphlitis is all that will be attempted here.

The most important micro-organism is the Bacterium coli commune (Escherich), which is almost constantly present. All investigators, the chief of whom are Tavel and Lanz, Hodenpyl, Ekehorn, Barbacci, Fowler-Ezra Wilson, Siegel, agree on this point. Some go so far as to claim that this micro-organism is the only one responsible for this disease, but this is an exaggerated view. It is true that in exceptional cases no other micro-organisms have been found in the appendix, either on direct examination or by culture methods ; as a rule, however, there are many other varieties present, especially diplococci and streptococci. Tavel and Lanz, Barbacci, Ezra Wilson and Siegel state unanimously that although direct examination of non-perforative appendicitis, of the appendix wall, and of perityphlitic pus, shows many different forms present in the mucous and purulent contents of the appendix (in one case as many as 12), yet on cultivation the Bacterium coli is commonly the only form that grows, while other bacteria fail to develop at all.

From a theoretic point of view, the discovery of Tavel and Lanz, since corroborated by various observers, is important—viz., that occasionally a pure coccus perityphlitis may be met with. In a patient who had suffered from three attacks and was operated upon in the quiescent period the mucous contents of the appendix and the fibrinous

peritonitic adhesions around the organ showed nothing but the Staphylococcus pyogenes citreus; in another case they obtained a pure culture of a streptococcus in the pus of a perityphlitic abscess. Then, again, Forster showed recently that the influenza bacillus may cause appendicitis and perityphlitic abscess.

A very brief review will be given of the bacteriologic observations made by Tavel and Lanz in 20 cases which they examined with great care; these cases were all non-specific; the contents of the organ were examined in cases during both the stage of inflammation and the stage of quiescence; the wall of the appendix, the adhesions, pus from perityphlitic abscesses, and exudate from the peritoneal cavity were all examined. The following varieties were found in fresh microscopic specimens: Bacillus coli communis mobilis et immobilis; Bacillus capsulatus; Bacillus fusiformis; Bacillus fœtidus liquefaciens; Bacillus pyocyaneus; the bacillus of glanders; tetanus, diphtheria, and a bacillus resembling the hay bacillus. In addition there were Staphylococcus citreus, streptococcus, pneumococcus, Coccus conglomeratus, Diplococcus intestinalis major et minor, spirilla, and the actinomyces. These bacteria were always found as mixed infections, with the exception of three cases: in one case the staphylococcus was found in pure culture (see above); in another the Staphylococcus citreus; and in one possibly the Bacillus coli communis (the specific forms of inflammation, as already stated, were excluded). In all these cases, however, with the possible exception of two or three, the Bacillus coli communis was present. Is it justifiable to conclude from these data that this micro-organism after all plays the most important part in the etiology of scolecoiditis and perityphlitis? This conclusion is not by any means certain, for Tavel and Lanz found it absent in two cases even when employing culture-media in which the Bacillus coli usually displaces all other micro-organisms that are likely to be present.

A remarkable addition to our knowledge of the bacteriology and etiology of appendicitis was made by the work of Veillon and Zuber, Krogius, and Friedrich. These writers attributed the greatest importance to the pathogenic anaërobic micro-organisms. (To obviate repetition, the reader is referred to the earlier paragraphs on this subject, bearing in mind particularly the work of the four authors named above.) Besides anaërobic organisms and the Bacillus coli communis, Krogius—and earlier still Barbacci—laid stress upon the presence of the Diplococcus pneumoniæ; the streptococcal forms, according to his investigations, playing a less important rôle in the etiology of perityphlitis.

The result of the greater number of bacteriologic investigations reveals, therefore, that in the common forms both of appendicitis itself and of secondary perityphlitis we are dealing, as a rule, not with a pure, but with a mixed, infection, the most important organisms being the Bacillus coli communis, the Streptococcus pyogenes, the Diplococcus pneumoniæ, and the anaërobes. That other forms of bacteria may assume the major rôle will be shown presently.

As most of these bacteria are either constant or at least common

inhabitants of the intestine (p. 35), the questions arise, Why do they not act in the same manner in other portions of the bowel? and what are the causes responsible for their pathogenic activity in the appendix? The reply that the appendix in man is in process of retrograde metamorphosis, and that, consequently, its vital powers of resistance are reduced, is hardly entirely satisfactory. It is possible, however, that a satisfactory answer may be obtained from a consideration of the anatomic and physiologic peculiarities of the appendix already dealt with in preceding paragraphs.

It is now generally recognized that the pathogenic powers of bacteria are not determined exclusively by their individual virulence, but also, and possibly primarily, by the resisting powers of the organism and of the particular tissues invaded by the bacteria. It appears that in the vermiform appendix the various factors which intensify the pathogenic power of the invading bacteria are present ; some of these factors have a tendency to increase the virulence of the bacteria, others to lower the resisting powers of the tissues, while in some instances both of these factors may be at work at the same time.

While difficult to prove, it seems highly probable that the appendix, with its numerous follicles, is a convenient point of entry for micro-organisms, and thus resembles the tonsils, which are also rich in lymphatic tissue.

The superficial layers of the mucous membrane of the appendix can easily be scraped off by hard fecal masses in the narrow lumen of the organ, and these abraded surfaces undoubtedly readily allow bacteria to get in, multiply, and exert their pathogenic action.

The fact that the appendix is so often curved or kinked is also important, for this favors retention of its contents and multiplication of retained bacteria, which find a suitable nidus for their growth in the retained secretions of the wall of the appendix ; in the face of this profuse bacterial growth the resistance and bactericidal powers of the appendix become inadequate in cases of typhoid fever, tuberculosis, and fecal concretions, and ulcers may arise, and in healing and cicatrization may act in the same way—viz., by narrowing the lumen of the organ and interfering with the exit of the appendicular contents (" vase clos " theory of French writers). C. Beck has also shown that a floating kidney on the right side may become an etiologic factor, by pressing the appendix when directed backward against the ilium. As an etiologic curiosity a case may be mentioned in which a small submucous abscess of the size of a pea closed the lumen of the appendix and gave rise to seropurulent fluid in it. This small abscess originated in a chronic catarrh of the colon at the site of the valve of Gerlach.

It is possible that acute or chronic catarrh starting in the cecum may act in the same way ; such a catarrh may produce no serious consequences in the cecum itself, but do great harm by leading to swelling of the mucous membrane of the appendix, either throughout its whole length or merely at the opening into the cecum, and thus causing stricture and retention of its contents, as a result of which otherwise harm-

less microbes develop intense pathogenic powers—*i. e.*, become viru-
lent.

Entozoa.—Recent publications, especially those of Still, Metsch-
nikoff, von Genser, and Schiller, have indicated the rôle played by ento-
zoa pathogenetically, such as Trichocephalus dispar, Oxyuris vermic-
ularis, and Ascaris lumbricoides. Each in its own peculiar way may
cause a catarrh of the appendix, and so favor the deposition and action
of bacteria—Trichocephalus dispar by its mode of attachment to the
mucous membrane, Oxyuris by its irritating movements, and Ascaris
by occlusion of the opening of the appendix into the cecum.

[That Ascaris need not give rise to any morbid change in the
appendix is shown by a number of cases examined by Le Roy des
Barres.[1]—ED.]

In all the above instances—and they represent clinically far and
away the majority—there is a "local" cause for the attacks of appen-
dicitis or perityphlitis. Recent observations have, however, proved
indisputably that in a small percentage of cases acute inflammation of
the appendix may occur through a general infection, such inflammation
being the local expression of conditions of the blood in which bacteria
are circulating freely. Several observers have reported a local out-
break of appendicitis of an almost epidemic character—*e. g.*, Golubow,
who considers the inflammation in most of the cases as an infection *sui
generis,* reported such in Moscow from the autumn to the spring of
1895–96 ; Sonnenburg again a district of Berlin in the spring of 1899
and in Frankfort in the autumn of 1899 ; Cathelin and others made
similar reports. In October and November, 1892, so many severe
cases of appendicitis with anatomic and clinical sequelæ occurred in
Vienna that one may almost speak of it as an epidemic.

In isolated cases, appendicitis and perityphlitis have been described
as sequelæ of scarlet fever, measles, rötheln, small-pox, chicken-pox,
and parotitis. In these cases we must make allowance for coincidences,
but in other cases it is quite certain that an attack of appendicitis is
the localized expression of a general infection.

An extremely acute and usually very severe *inflammation of the ap-
pendix resulting in an abscess* has several times been described in cases
of suppurative tonsillitis (Apolant, Kretz, Sonnenburg, Kelynack,
Schnitzler), and exactly the same streptococcus was found in the ap-
pendix as in the tonsils. Kretz imagines that here the bacteria were
swallowed and so reached the appendix, which some purely local condi-
tion had converted into a "place of least resistance"; other observers,
however, take the view that the vascular system best represents the
means of transport.

The occurrence of appendicitis as a local lesion in **influenza** has
been fairly often reported—*e. g.*, by Faisans, F. Franke, Florand, Mau-
claire. Without a doubt the etiologic connection has been proved by
an observation of Cahn's (reported by Adrian) respecting a patient with
acute influenza and acute perityphlitic abscess. Forster was able to

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[1] Le Roy des Barres, *Gaz. des Hop.*, 1903, p. 1223.

prove the presence of the influenza bacillus in the pus from the abscess both by staining and by culture.

[From a recent statistical investigation Schultes [1] concludes that there is probably no connection between epidemics of influenza and appendicitis.—Ed.]

Finally, appendicitis and perityphlitis have been described in cases of "polyarthritis acuta" by Beverly Robinson, Pribram, G. A. Sutherland, Adrian (from Naunyn's clinic), Finneyand Hamburger, and others. The clinical details varied, but the fact of the etiologic relationship appears no longer improbable in the face of the very considerable number of such cases.

[Probably Sir J. Grant,[2] of Canada, was the first, in 1893, to describe rheumatic appendicitis. Poynton [3] and Edwards,[4] the latter of whom instituted a collective investigation into the subject, come to a conclusion rather opposed to the existence of rheumatic appendicitis. Haig [5] and others have described cases clinically suggesting appendicitis, which have rapidly recovered under salicylates. It is conceivable that some cases of appendicitis begin as rheumatic inflammation and subsequently become infected with other micro-organisms. Haig [6] believes that the action of uric acid on the fibrous tissues of the appendix may give rise to appendicitis, but this view has not been taken very seriously. —Ed.]

Differing entirely from the sequence of events in such general infectious diseases is the case of disease of the appendix in relation to typhoid and tuberculosis. Here the typical picture of perityphlitis, as caused by a local focus of the hemic infection, is wanting; it is merely part and parcel of the general typhoid or tuberculous affection of the cecum.

Another question requires consideration here, namely, how the bacteria supposed to produce appendicitis act. A distinction must be drawn between inflammation of the mucosa and the deeper layers of the appendix, on the one hand, and the secondary changes in the peritoneum due to appendicitis—*i. e.*, perityphlitis and peritonitis with and without perforation. (The reader should refer to the sections on Peritonitis in General for many of the details, such as the questions whether bacteria themselves enter the peritoneal cavity and how they get there; whether they enter the peritoneum alone or mixed with bowel contents; whether the bacterial products alone invade the peritoneum, and, finally, whether this invasion occurs gradually or suddenly; these questions, having already been discussed in detail, will not be considered here.)

Recently **experiments upon animals** have been made with a view of studying more closely the pathogenesis of appendicitis and perityphlitis. Roux, who was first in the field, found the introduction of a foreign body alone insufficient to pro-

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[1] Schultes, *Deutsch. med. Wochenschr.*, 1903, p. 752.
[2] Sir J. Grant, *Medical Record*, New York, 1893, vol. ii., p. 609.
[3] F. J. Poynton, *Trans. Med. Soc.*, vol. xxiv., p. 22.
[4] W. A. Edwards, *American Medicine*, April 12, 1902, p. 595.
[5] A. Haig, *Practitioner*, 1893, vol. l., p. 17.
[6] A. Haig, *Uric Acid*, 1903, p. 466, fourth ed.

duce inflammation. Experiments by Roger and Josué supported the view taken of the importance of narrowing of the appendix in causing its inflammation; their experiments proved that in rabbits a purulent inflammation is set up, perhaps not by narrowing alone, but at all events by complete occlusion. By later experiments Josué succeeded in producing lesions of the appendix in healthy rabbits by intravenous injections of a strepto bacillus taken from the appendix of a rabbit which had succumbed to an epidemic attack in the laboratory.

[Frazier[1] ligated the appendix in rabbits, taking care not to include the vessels, and obtained as a result a mucopurulent condition of the contents, which supports Talamon's and Dieulafoy's view as to the formation of a closed sac favoring microbic activity. He finds that foreign bodies are unimportant, and that imperfect drainage is a most important factor in the production of appendicitis, since the Bacillus coli then becomes virulent. Varying degrees of virulence of micro-organisms account for varying degrees of severity in appendicitis. Interference with the blood-supply of the organ is important in reducing the resistance of the parts and in increasing the virulence of the micro-organisms.—Ed.]

Beaussenat drew the following conclusions from numerous experiments. Appendicitis is almost always the result of infection, chiefly from the intestinal canal, but sometimes from the blood. Generally the Bacillus coli communis is found. Predisposing causes are necessary for the pathogenic action of the bacteria, and of these, enterocolitis is the most important. The experiments of Anghel and de Klecki confirmed these conclusions in the more important points. Mühsam produced experimentally in rabbits disturbances of the circulation in the appendix, in order to arrive at some conclusion as to their importance in producing inflammatory changes. Although gangrene of the organ could be produced by these means, the inflammatory process did not extend to neighboring tissues. These experiments are, therefore, practically unimportant in connection with the pathology of the human subject. Adrian came to the same conclusions with respect to local attacks in the rabbit's appendix. He, however, produced inflammation of the appendix in rabbits by means of the circulation, more particularly by injecting into a vein of the ear streptococci, staphylococci, and pneumococci. Follicular inflammation appeared in the appendix at a time when, in other organs, especially in the rest of the alimentary canal, no such changes were demonstrable.

Reference must be made here to the views of van Cott and his collaborator, Fowler, as to the pathogenesis of appendicitis, which are considerably at variance with those generally accepted. They attach much more importance to factors connected with the circulation in the genesis of inflammations of the appendix than is generally thought to be the case with inflammation of any other part. They answer the question why the appendix is so much more frequently the seat of pathologic changes than any other portion of the intestinal canal as follows:

In no other part, at any rate of the intestine, can vascular and nervous, and consequently nutritional, changes be produced so easily as in the vermiform appendix. In this organ there is an end-artery, a single branch of the mesenteric artery, which supplies the whole organ; in addition, there is a very small collateral circulation through adjacent portions of the cecum. In females a third source of blood supply comes from the appendiculo-ovarian ligament (a fold of the peritoneum first described by Clado, which runs from the meso-appendix to the broad ligament). This ligament is said to contain a small artery that ends in the appendix. The remarkable fact that appendicitis is much less common in women than in men is possibly explained by the existence of this third artery; they also mention another anatomic detail which tends to show why the blood-supply of the appendix is terminal; they believe that sometimes the appendicular artery runs to the end of the appendix, alongside of the organ, and without sending any branches into its substance; when it reaches the distal extremity, it turns upon itself, and as a recurrent artery enters into the substance of the appendix; when the lumen of the artery becomes occluded in any part of its course before it enters the appendix, anemia of the organ would naturally result. [See also Lockwood's observations on p. 823.—Ed.]

[1] C. H. Frazier, *Contributions from the William Pepper Clinical Laboratory*, 1900, p. 395.

The vessels, which, under ordinary circumstances, send only a very small and inadequate quantity of blood into the appendix, are, moreover, according to van Cott's observations, very commonly the seat of pathologic changes. In 14 appendices removed by Fowler he found "some form of vascular obstruction in the meso-appendix—*i. e.*, either paravasculitis, perivasculitis, or endovasculitis, or organized thrombi, a process that must, of course, have preceded by some time the round-cell infiltration and the formation of pus-foci in the walls of the organ." In a number of the cases endoneuritic and perineuritic processes were found in addition to the vascular changes, with extensive atrophy of the nerve-fibers. Van Cott imagines the pathogenetic rôle of these vascular and nervous changes to be the following:

The great motility of the appendix and of its mesentery readily allows torsion to occur, which, there can be little doubt, has a direct causal relationship to the vascular and nervous changes. Another source of trophic disturbances is a progressive hyperplasia of the sheaths of the appendix, a condition that was also discovered by van Cott in the various appendices that he examined. He argues that this condition "is probably the result of repeated hyperemia or chronic venous engorgement due to interference with the return of venous blood in the meso-appendix; and that these primary conditions must eventually lead to the degenerative changes described in the vessels and nerves of the organ." He regards these vascular and nervous changes as the true etiologic factors of appendicitis. They are followed directly by local necrosis or by trophic ulcers, and thus a *locus minoris resistentiæ* is created which favors the invasion of pathogenic bacteria from the bowel contents and inflammation of the appendix. Robert Breuer, at my suggestion, has repeated the observations of van Cott and Fowler on 30 appendices either removed by operation or obtained from autopsies, showing various forms of appendicitis, acute, chronic, recurring, etc.

In addition, a number of normal or practically normal appendices—*i. e.*, appendices adherent at tip or at the free margin of their mesentery to some neighboring part, but that were otherwise normal—were examined by Breuer, whose results were briefly as follows:

The appendix is at a disadvantage as regards its blood-supply, as well as in other respects, when compared with other parts of the intestine, more especially because, being an appendix with a free extremity, it is only in direct continuity with the rest of the bowel at one point; nevertheless, the blood-supply of the organ can hardly be considered to be a terminal one. The conditions are somewhat different here than in the end-arteries of the brain, the spleen, and kidneys, inasmuch as a few, though not entirely insignificant, arterial branches enter the appendix from neighboring parts (the cecum); these anastomotic branches, as was demonstrated in specimens injected immediately after removal, are partly in the mucous membrane, partly in the muscular coat, and partly immediately under the peritoneum; their lumen is much smaller than that of the appendicular artery, but the total amount of blood they convey must be of distinct importance in the nutrition of the organ. The vascular supply of the appendix may, therefore, rightly be regarded as different from that of other parts of the bowel, but it cannot be described as a terminal blood-supply composed of end-arteries.

Breuer's observations did not confirm the presence of the little artery which van Cott states runs in the "appendiculo-ovarian ligament," and considers as responsible for the relative infrequency of appendicitis in the female; in some of the female appendices this vessel could not be found at all on careful microscopic examination; and in one appendix that was carefully injected, the vessel was definitely absent.

Changes in the vessels were not present so constantly or to such an extent as van Cott claimed. In cases of chronic appendicitis the large vessels running in the mesenteriolum were invariably found to be intact even when they were surrounded by connective tissue in a state of cicatricial contraction. The smaller arteries and veins in the various layers of the wall of the organ were only rarely altered, and then only to a very slight extent; thus in places where the whole of the mucous membrane was converted into cicatricial tissue there was slight thickening of the adventitia or slight proliferation of the endothelium of the intima, changes which would presumably be found in any obliterated tissue and were, moreover, also found in normal subinvolution of the vermiform appendix.

In acute purulent appendicitis changes in the vessels were more common; but they were limited to the area acutely inflamed and to the tissues in its immediate neighborhood. Thus, for instance, in cases in which the mucous membrane was necrotic throughout its whole extent and covered with a fibrinous exudate the blood-vessels in this area were also necrotic and had lost their typical structure; in the vicinity of a miliary abscess a vein was occasionally found full of pus-cells; in one case of acute appendicitis a suppurating thrombus was found in one of the larger venous branches in the meso-appendix. In all these instances, however, the vascular changes were not more marked than in any other case of acute inflammation, and there was certainly nothing to suggest that the changes in the blood-vessels preceded the other inflammatory changes; in other words, these observations, taken as a whole, did not corroborate van Cott's theory that the vascular changes were the cause of the inflammation of the appendix. Breuer's investigations, therefore, show that, as regards their number, arrangement, and powers of resistance, the blood-vessels of the appendix are inferior to the blood-vessels of other parts of the intestine; that the blood-supply of the appendix is inferior to that of other parts of the alimentary canal, and, therefore, less able to protect against infection. It is also clear that embolism of the appendicular artery near its origin must be fraught with most serious consequences; this, however, is a very rare event, and cannot be regarded as playing a part of any importance in the production of such a common disease as appendicitis.

From a practical point of view, therefore, it may be concluded that changes in the blood-vessels play no more important rôle in the acute and the chronic inflammations of the appendix than in the causation of similar processes in other portions of the intestinal tract.

A careful and extended search was made in all the cases, a number of specimens being examined by Marchi's method, so as not to miss any of the finer changes, but, as in the case of the blood-vessels, no changes were found which could be regarded as playing a causal part in the production of appendicitis as suggested by van Cott. Hemmeter's experiments do not definitely support van Cott's or Breuer's views. Kelly came to the same conclusion as Breuer with regard to the nerves, but he attributed some importance in the pathogeny to partial or complete obstruction of the blood-vessels.

The essential cause of appendicitis and of the resulting perityphlitis must, therefore, be sought exclusively in the two factors already mentioned—*i. e.*, fecal concretions and bacteria—which are responsible for the overwhelming majority of the cases of inflammation in the right iliac fossa, seen in every-day practice as perityphlitis. All the other possible causal factors, which will be very briefly referred to, are relatively quite insignificant and of very subordinate importance.

Tuberculosis occasionally produces ulceration of the appendix; how frequently this occurs cannot be stated, for no large statistics exist on this subject. There is almost invariably, in addition, more or less general tuberculosis of other parts of the bowel. At all events, as Kelynack says, primary and isolated tuberculosis of the appendix is exceedingly rare, if it ever occurs. Clinically, tuberculosis of the appendix occupies an exceptional position, since, although it may occasionally produce perforation and diffuse peritonitis, it only exceptionally produces the syndrome of an inflammatory process strictly localized in the right iliac fossa. As a rule, the signs of perityphlitis are merged and obscured by those of chronic diffuse inflammation of the peritoneum.

[Crowder has published a detailed account of a case of hyperplastic tuberculosis limited to the appendix. In the published cases of hyperplastic tuberculosis of the intestine, of which there are about a hundred, the appendix was only affected very rarely, and then in common with

the cecum. Crowder's [1] case, which was operated upon for appendicitis, appears to be the only one in which the process was confined to the appendix.—Ed.]

Typhoid ulcers may also occasionally occur in the appendix and may sometimes lead to perforation and acute peritonitis. The statements of different writers vary greatly as to the frequency of this accident; Heschl, for instance, states that it occurs in 14 per cent. of the cases of typhoid perforation; Fitz in only 3 per cent.; and Curschmann, of 64 perforations in autopsies made by himself, found one only originating in the appendix.

[Finney,[2] from various statistics, estimates that perforation of the appendix occurs in 5 per cent. of the cases of enteric perforation. In 133 cases of perforation in typhoid collected by Nacke[3] the appendix was involved in 15, or 11 per cent. In 18 cases of perforation in typhoid fever at St. George's Hospital the appendix was involved in 2, or 11 per cent. (Rolleston[4]).—Ed.]

The ordinary picture of perityphlitis is hardly ever seen in direct connection with typhoid; at the most, perityphlitis occasionally develops indirectly as the result of cicatrization of a typhoid ulcer in the appendix, which ultimately leads to the development of the changes in the appendix (superinduced by non-specific bacteria and stereoliths) which have already been described in detail.

[Dieulafoy[5] describes two forms of typhoid appendicitis as capable of producing peritonitis: (*a*) "Peritonitis by propagation," in which typhoid ulceration occurs in the appendix with perforation and peritonitis, the symptoms resembling those of perforation of the intestine in typhoid fever; (*b*) "para-appendicitis," or true appendicitis of the ordinary form, with its usual symptoms.—Ed.]

In the last few years a number of cases have been reported in which **actinomycosis** produced a disease of the appendix that exactly simulated perityphlitis, and was, in fact, diagnosed as such by the experienced physicians who studied these cases. It is, of course, impossible to determine whether or not this inflammation actually started from the appendix in all the reported cases, but there are a few on record in which an autopsy was performed and in which the possibility of this occurrence was established beyond a doubt; such a case is, for instance, reported by Ransome (quoted in Kelynack's work).

[As a curiosity, reference may be made to an appendix twice its natural size and gritty from the presence of nodules composed of dense fibrous tissue surrounding groups of bilharzia parasites (Cureton and Law Webb[6]).—Ed.]

A number of factors may be briefly mentioned which were formerly thought to play an important part in the pathogenesis of appendicitis

[1] T. R. Crowder, *Amer. Jour. Med. Sci.*, vol. cxxiv., p. 236.
[2] Finney, *Johns Hopkins Hosp. Bull.*, May, 1897.
[3] Nacke, quoted by Monod and Vanverte, *Rev. de Chir.*, March, 1897.
[4] Rolleston, *Trans. Clin. Soc.*, vol. xxxi., p. 234.
[5] G. Dieulafoy, *La Presse Méd.*, October 28, 1896.   [6] *Lancet*, 1899, vol. i., p. 156.

and perityphlitis, and are still regarded in this light by some writers, but in my opinion possess no direct relation to this characteristic disease.

That a cold may cause perityphlitis is often stated by the patients themselves; but accurate proof of this assertion has so far never been forthcoming.

Appendicitis is sometimes stated to be caused by injuries or traumatic factors, such as violent muscular effort, concussion of the body, or a blow directly over the ileocecal region. It is quite conceivable— and cases have actually been recorded to the effect—that rupture or perforation of an appendix which had previously been diseased for a long time may be precipitated by injury, but in such cases it can hardly be said that injury caused the disease. It is also conceivable that local injury in the region of the appendix may occasionally produce a disease-picture similar to that of appendicitis or perityphlitis, but such a disease-picture has in reality nothing to do with appendicitis, for it is due to the presence of a circumscribed peritoneal abscess which, as far as its causation is concerned, might just as well have developed in any other part of the peritoneum; pathogenetically, an abscess of this kind has nothing whatever to do with the special anatomy of this region, and, above all, with the vermiform appendix.

In the last few years traumatic perityphlitis, apart from casual communications, has been repeatedly described (Pribram, Epstein, Stern, Schottmüller, Fürbringer). I do not find, however, that the view expressed in the above paragraph in the first edition needs any considerable reconsideration on account of these communications. Some of the observations cannot be considered in any way as a basis for scientific discussion of the pathologic questions concerned; others confirm the view that trauma takes effect in obviously diseased appendices—*e. g.*, in all three cases of Schottmüller's fecal concretions were present; others again have nothing to do with appendicitis proper, but are really a circumscribed traumatic peritonitis in the right iliac fossa. It still remains, in my opinion, to be proved that inflammation, eventually leading to perityphlitis, can arise in a perfectly healthy and empty appendix, as Fürbringer believes. The medicolegal aspect and the question of accident and insurance are, of course, quite distinct and fall into quite another category.

[Appendicitis coming on after a sudden strain or effort has been usually attributed to the action of the psoas muscle, the appendix being supposed to be adherent to the sheath of this muscle as the result of a slight former attack of appendicitis. On a very vigorous contraction of the muscle the adhesions are torn and the appendix ruptured. Barnard[1] believes that in some cases the laceration of the adherent appendix is due, not to contraction of the psoas, but to a powerful elevation of all the abdominal viscera. This elevation is attributed by A. Keith to the action of the transversalis muscle, which surrounds the abdomen like a belt.—Ed.]

In the past, and even at the present day, a great deal of importance

[1] H. L. Barnard, *Clinical Jour.*, September 16, 1903, vol. xxii., p. 340.

was attributed to the functional activity of the bowels in the genesis of the diseases of the appendix and the surrounding parts. Constipation in particular was credited with an important rôle; and, conversely, although to a slighter degree, diarrhea. In the same way the régime and the general mode of nutrition of the patient were occasionally thought to be of importance in the causation of the disease. In fact, the relative frequency of disease of the appendix in English and American subjects has been attributed to racial peculiarities in their mode of life and food.

[The increased incidence of appendicitis in the last twenty years, about which there can be no doubt, even after making full allowance for the wider recognition and the influence of abdominal operations, has been thought to be due to various factors, such as influenza, greater consumption of frozen meat,[1] and other conditions, some of which cannot be taken seriously.—ED.]

I confess that, so far as I have been able to study the literature on this particular subject, I am very far from being convinced of this relative frequency of the disease in these nationalities; the statements that have so far been made are too indefinite to justify this assumption.

In discussing the subject of stercoral typhlitis the action of the bowels will be referred to again, and I shall not do more here than state that I do not recognize any clear relation between the condition of the bowels and appendicitis and perityphlitis, a view which is accepted by the majority of clinicians who have written on this subject. It is possible that slight perversions of the normal functional activity of the bowel may occasionally favor the entrance of small masses of fecal material into the appendix, and may thus indirectly constitute an etiologic factor in the genesis of disease of this organ, but on the grounds of my own observations, which are in accord with those of Reginald Fitz, Fowler, and others, I believe that in only a small proportion of my cases was the onset of the first symptoms preceded by constipation, and still less frequently by diarrhea. The majority of the patients state that their bowels have acted perfectly regularly. Constipation and diarrhea must, therefore, not only be eliminated from the list of factors directly causing disease of the appendix, but they cannot even be regarded as indirect accessory factors in its production.

[The view that appendicitis is due to the spread of infection and inflammation from the cecum is widely held. Treves[2] says that a large proportion of patients suffering from perityphlitis are chronic dyspeptics, and gives examples of methods in which an attack of appendicitis may be precipitated by unsuitable diet. Bad teeth may favor the production of appendicitis, either mechanically, by preventing proper mastication, or through oral sepsis and infection of the whole of the alimentary canal. Reclus[3] argued in favor of the view that intestinal catarrh precedes appendicitis, and Robin[4] brought forward statistics to

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[1] K. Smith, *Brit. Med. Jour.*, 1903, vol. i., p. 1527.
[2] Treves, *Allbutt's System of Medicine*, vol. iii., p. 897; *Brit. Med. Jour.*, 1902, vol. i., p. 1590.   [3] Reclus, *Acad. de Méd.*, Paris, April, 1897.   [4] Robin, *ibid.*

show that intestinal catarrh and appendicitis have a common origin in a peculiar form of dyspepsia associated with "arthritism" and largely due to constipation. Dieulafoy,[1] who strongly opposed Reclus' views, has since suggested that inflammation of the gall-bladder may form the primary focus from which micro-organisms descend *via* the intestine to the appendix.—Ed.]

One class of cases remains to be discussed, namely, those with the well-known clinical picture of perityphlitis in which there is an inflammatory process in the right iliac fossa, but of appendicular origin. In contrast to the views formerly held, it is now recognized that this occurrence is exceedingly rare—so rare, in fact, that many authors are skeptical whether such a pathologic condition ever occurs. They explain the cases in which perforation of the cecum with perityphlitis actually occurs by the supposition that it is just as conceivable that the perforation of the cecum took place from without—*i. e.*, by apposition of the appendix to the cecum and subsequent rupture of the abscess into the cecum ; in other words, that the ulceration of the wall of the cecum began on the peritoneal surface and extended inward to the mucous membrane. That such a thing does occur is undoubtedly true, but it is hardly justifiable to deny the possibility of a typhlitic perityphlitis or of perityphlitis of cecal origin. It is well to keep an open mind and adopt a cautious attitude, particularly at the present time, where the more modern ideas on this disease are universally dominant. Unbiased observation proves beyond a doubt that there are cases, although their number is infinitely small as compared with those in which the appendix is the primary cause of the disease, in which the anatomic and the clinical picture of perityphlitis develops because the typhlon (cecum) itself is primarily diseased, while the appendix is perfectly normal.

Opinion on this subject has changed to the extent that at the present day the possibility of a primary affection of the cecum must be admitted in isolated cases. As this change of opinion has, however, occurred only within the last few years, and as many prominent physicians still believe that primary disease of the cecum is a common occurrence, and in *many* cases predisposes to the development of perityphlitis, it is necessary, in order to clear up this matter, to discuss it in some detail.

It has just been pointed out that there are exceptional cases in which disease of the cecum leads to perityphlitis while the appendix is normal. As postmortem examinations show, nearly all these cases are due to ulceration of the cecum, sometimes of specific origin (tuberculosis, rarely typhoid), but in the vast majority of the cases decubital ulcers, either with stricture of the intestine or with obstinate constipation and without any stenosis. These cases, however, present nothing peculiar or characteristic of disease of the cecum. They present the same manifestations of peritonitic inflammations as are seen in other portions of the gastro-intestinal tract as the result of stenosis and long-continued fecal accumulation, with the resulting formation of decubital ulcers. It is, therefore, merely a matter of

[1] Dieulafoy, *Acad. de Méd.*, Paris, June 16, 1903 ; *La Presse Méd.*, 1903, p. 445.

chance due to the localization of the inflammation in the right iliac fossa that the same clinical picture is produced as in disease of the appendix.

The real question to be decided is whether simple inflammation of the cecum without ulceration and perforation can produce the syndrome of perityphlitis, as was advocated by Puchelt and Albers. This form of inflammation was regarded as being chiefly due to fecal accumulation, and the condition has, therefore, been known under the name of "typhlitis stercoralis." The discussion as to the genesis of stercoral typhlitis is by no means an idle one, nor purely theoretic in scope, but is, on the contrary, of the greatest practical importance, for the view that fecal accumulation was the determining factor in the genesis of the disease has exerted a great influence on the treatment of this condition.

In the first paragraphs of this section the reasons were given for the conclusion that in the great majority of cases the appendix, and not the cecum, is the starting-point of the inflammatory process ; this knowledge is based on numerous autopsies and biopsies performed since the advent of improved surgical technic and operative treatment of these cases. The objection that only the severe cases were caused by disease of the appendix, while the milder cases that recovered without an operation were due to disease of the cecum, has been refuted by the lesions found in many cases in which an early operation was performed in mild cases. The assumption that there is a primary stercoral typhlitis is based on speculation and on conclusions drawn from ambiguous clinical symptoms ; the existence of primary appendicitis, on the other hand, is demonstrated by facts.

This controversy might, therefore, well be abandoned and the question regarded as settled, were it not that the theory of a primary typhlitis stercoralis is so persistently supported. On these grounds no apology is necessary for adducing further arguments against this obstinately maintained view. These arguments, which are independent of the actual conditions made postmortem and *in vivo*, have been clearly put forward by several authors, especially by Sahli and Talamon.

If fecal accumulation can really produce typhlitis and perityphlitis, why do similar inflammatory conditions never occur in the neighborhood of the sigmoid flexure, where fecal accumulation is both more frequent and more marked? Why is there a history of habitual constipation in only a small proportion of the cases of perityphlitis, and an absence of this condition in the majority of the cases? for the constipation which accompanies the onset of perityphlitis cannot, of course, be adduced as evidence of the causative rôle of constipation in perityphlitis, since it is the result and in no way the cause of the disease. Why is perityphlitis more common in men than in women, whereas constipation is much more common in the latter? The answers to these questions are a strong argument against the supposition that the disease originates primarily in the cecum.

A number of plausible arguments drawn from the clinical history

of a number of cases have contributed much to the popularity of the ancient view we are attempting to refute. A series of cases with a definite clinical picture and ending in recovery were attributed to a primary stercoral typhlitis; another series of more severe cases was attributed to disease of the appendix (perforation of the appendix). There has been no proof that this arbitrary method of subdivision and classification is in any way justified. The exploratory abdominal operations which have been so frequently carried out on these cases during the last fifteen years have clearly shown that the mild cases that were formerly attributed to stercoral typhlitis, are produced by disease of the appendix, as well as the severe cases that were always regarded as due to changes in the appendix.

The "fecal plug" was also formerly considered a proof of the existence of stercoral typhlitis. A tumor-like swelling was found in the ileocecal region, and a history of constipation was at the same time given by the patient—*i. e.*, during the time when the inflammatory process was developing; it was, furthermore, noted that this tumor grew smaller when the patient improved spontaneously, or even sooner if a purgative was given; hence the conclusion that the swelling was a fecal tumor, and that this accumulation of feces, by irritating the wall of the cecum, had produced inflammation of this part of the bowel, and subsequently of the surrounding tissues. It must be admitted that occasionally the swelling consists in part, or even entirely, of feces, and that it may sometimes be present before the development of the inflammatory symptoms. This, however, does not prove that the tumor was necessarily the cause of the inflammation. It is even more difficult to prove that the fecal accumulation does not occur during the inflammatory stage, as will be shown below; and it is, finally, still more difficult to prove that the swelling was really composed of fecal contents and constituted a "fecal plug." In fact, it may be considered as practically certain now that in the great majority of the cases the swelling consists of inflammatory products and is, therefore, the result and the anatomic expression of the perityphlitic inflammation, and not by any means its cause. It appears, therefore, that all the evidence tends to show that the conception of simple ulcerative stercoral typhlitis as a cause of perityphlitis is erroneous and should be abandoned.

Very few cases, indeed, are on record in which the course of events corresponds with the description given by the earlier writers on the subject; and careful criticism reduces their number to a minimum. As an illustration, the carefully observed cases recorded by Lennander may be referred to; in 74 cases diagnosed and operated upon as perityphlitis, only 4 were not appendicular in origin. In 2 of these 4 cases, moreover, although the manifestations were those of perityphlitis, there was no discoverable inflammation in the cecal region; in one of the cases there was only acute diffuse peritonitis, probably, as shown later, due to typhoid; in the other case there was a small swelling in the right iliac fossa, consisting of a mass of omentum surrounding a small ovarian cyst of the right ovary. In the third case, in a young

girl of fifteen, diagnosed as "perityphlitis," the appendix appeared perfectly normal at the operation, and no other primary source for the disease was found ; as a matter of fact, the condition found differed in several respects from the typical picture of perityphlitis, since there were adhesions extending upward as far as the hepatic flexure of the colon. The possibility of tuberculosis and of actinomycosis was thought of, but at all events there was no definite proof of the existence of simple stercoral typhlitis. The fourth case is not more conclusive than the other three, although it can be more correctly interpreted as simple stercoral typhlitis. The patient, a young man the subject for six years of chronic constipation, had for the last year and three-quarters had repeated attacks of "inflammation of the appendix." At the operation the appendix was completely absent, and there were none of the typical morbid lesions of perityphlitis, but there was a band 4 cm. broad extending from the anterior surface of the intestine at the junction of the ascending colon and the cecum to the abdominal wall. This was divided and the patient made an uneventful recovery. It is possible that this band was due to fecel accumulation, but even this does not prove that stercoral typhlitis ever existed (compare p. 472, where Virchow's view on this subject is given).

I cannot point to any published cases in which the clinical picture of perityphlitis could definitely and without any question be attributed to non-ulcerative stercoral typhlitis ; the majority of the cases recorded under this heading do not stand rigid criticism. To quote one more example : Sonnenburg reports a case that he declares with some reserve to be a "circumscribed inflammation of a portion of the wall of the cecum" ; but, according to the author himself, the case is so obscure that it can hardly be used to prove the existence of this condition. Lop has also recently reported a case of "typhlite suppurée sans lésion de l'appendice," in which laparotomy showed gangrenous perforation of the cecum of obscure origin, for after the death of the patient no autopsy was performed. Meusser has quite recently reported two cases from Riedel's clinic, which also differ so markedly from the ordinary picture of perityphlitis that they can hardly be taken to prove anything. We, therefore, arrive at the conclusion that, although the possibility of simple stercoral typhlitis as a primary cause of perityphlitis cannot be absolutely denied, it is exceedingly rare.

The following statistics appear to me to be a reliable basis for merely forming a sound opinion as to the etiology and the prognosis of this disease.

Being taken from the reports of the Pathologic Institute of the General Hospital in Vienna, they are, of course, compiled without any prejudice whatever ; they are drawn from the years 1870–1896, and include 44,940 autopsies. I reproduce the figures without comment ; the data given can be discussed without difficulty in the appropriate places in the text :

Total number of autopsies in the years 1870–1896 . . . . . 44,940.
Number of autopsies of appendicitis, perityphlitis, and
  paratyphlitis . . . . 148 (0.3 per cent.).
Men . . . . 107 (73.3 " ").
Women . . . . 41 (27.7 " ").

In the decade— 1– 9 years, 2 cases.   In the decade—40–50 years, 18 cases.
  "   "   10–19 "   43 "   "   "   50–59 "   4 "
  "   "   20–29 "   49 "   "   "   60–69 "   5 "
  "   "   30–39 "   24 "   "   "   70–79 "   1 case.
     No age given, 2 cases.

Changes were present in the vermiform appendix in 129 cases:
  With perforation . . . . 124 cases.
  Without perforation . . . . 5 "
  No statement as to the condition of the vermiform appendix . in 19 cases.
  Fecal concretions present . . . . in 42 " [1]
  Diffuse peritonitis present . . . . in 107 "
  Combined with disease of the genital organs . . . . . . in 6 "
  Disease of the genital organs alone (clinically perityphlitis) . in 3 "

Cases in which a clinical diagnosis of perityphlitis was made, but after death were
shown to be—
  Tuberculous stricture of the cecum . . . . in 2 cases.
  Pyosalpinx . . . . in 1 case.
  Metrolymphangitis . . . . in 1 "
  Thrombosis of the portal vein . . . . in 1 "
  Typhoid fever . . . . in 2 cases.
  Caries of the vertebræ with abscess . . . . in 1 case.
  Septicæmia ex tonsillitide abscedente . . . . in 1 "

Perityphlitis found after death where the clinical diagnosis was:
  Incarceratio interna . . . . in 3 cases.
  Ileus . . . . in 1 case.
  Tumor adnexorum . . . . in 1 "
  Perforation of a gastric ulcer . . . . in 1 "
  Tuberculosis of the peritoneum and lungs . . . . in 1 "

Summary of the cases according to years:

1870 . . . . 1 case.	1884 . . . . 2 cases.
1871 . . . . 1 "	1885 . . . . 6 "
1872 . . . . 7 cases.	1886 . . . . 6 "
1873 . . . . 4 "	1887 . . . . 4 "
1874 . . . . 6 "	1888 . . . . 14 "
1875 . . . . 1 case.	1889 . . . . 11 "
1876 . . . . 3 cases.	1890 . . . . 8 "
1877 . . . . 2 "	1891 . . . . 8 "
1878 . . . . 1 case.	1892 . . . . 10 "
1879 . . . . 3 cases.	1893 . . . . 8 "
1880 . . . . 2 "	1894 . . . . 10 "
1881 . . . . 1 case.	1895 . . . . 10 "
1882 . . . . 3 cases.	1896 . . . . 14 "
1883 . . . . 2 "	

PATHOLOGIC ANATOMY.

A number of morbid changes underlie the clinical manifestations of inflammation in the right iliac fossa, and consequently various anatomic divisions and so-called forms of the disease have been described according to the different standpoint taken by the various writers on the subject. A simple description of the actual morbid lesions seems the best way of considering the subject, and will give a more accurate account of the disease than any systematic or formal classification, such as some authors have attempted.

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[1] Other foreign bodies in two cases.

[Lockwood [1] classifies cases of appendicitis according to their pathology into the following groups :
  I. Appendicitis with ulceration of the mucosa.
  II. Appendicitis with ulceration of the mucosa and bacterial invasion.
  III. Appendicitis with ulceration of the mucosa and with fecal concretions—foreign bodies—and their complications.
  IV. Appendicitis with stenosis and its complications—cysts, mucocele, empyema, ulcerations, and bacterial invasion.
  V. Appendicitis with stenosis and obliteration of the lumen.
  VI. Appendicitis with lymphangitis and lymphadenitis.
  VII. Tuberculous appendicitis—actinomycotic appendicitis.
  VIII. Appendicitis complicating malignant and other diseases.—Ed.]

At the outset it must be expressly stated again that so-called obliteration of the appendix, at least in the majority of cases, should not be described as pathologic. Ribbert's, Zuckerkandl's, and Sudsuki's exhaustive observations, already described, clearly prove that it is not inflammatory, and the final result of appendicitis, but is merely the last stage of a physiologic subinvolution. Neglect of this fact accounts for statements in many works as to the great frequency of pathologic changes in the appendix in postmortem examinations. Thus Toft (quoted by Matterstock) found pathologic conditions in the appendix in nearly every third body between the ages of twenty and seventy years in 300 unselected cases examined postmortem, and when there was no evidence of actual disease, he found evidence of past inflammation. Tüngel (also quoted by Matterstock) found the appendix affected in the same proportion ; Kraussold even goes so far as to say that Toft's estimate is, if anything, too low, while other observers, such as Ranschopf, Kelynack, and Sonnenburg regard these figures as exaggerated.

In order to make the subject as clear as possible, the anatomic changes and the pathologic processes going on in the appendix itself, apart from the peritoneum, will be first described ; then the morbid processes and changes in the peritoneum of the appendix and in its immediate vicinity, and, lastly, all the other pathologic results.

Changes in the Appendix Itself.—Early surgical interference has brought to light a fact which was unknown when information was solely derived from the data found postmortem in the most severe cases —viz., that there is such a thing as a simple inflammation of the appendix—appendicitis simplex. This condition may occasionally occur alone without any change in the neighboring parts, so that the lesion of the appendix is the only cause for the clinical symptoms present. In the great majority of cases, moreover, this simple inflammation of the organ is the starting-point of the further changes which are summarized under the name of " perityphlitis."

In simple appendicitis the morbid changes are entirely confined to the walls of the appendix, and may not extend to its peritoneal coat, and in some cases, as shown by examination of the appendix, in cases

[1] C. B. Lockwood, *Appendicitis, Its Pathology and Surgery*, p. 5.

where the abdomen has been opened for some other condition, the inflammation is limited to the mucous membrane. Cases of this kind are rightly spoken of as *catarrhal appendicitis* ("endoappendicitis" of some authors, "appendicitis granulosa" of Riedel). In acute cases the mucosa of the appendix is swollen and reddened, and there is an excessive amount of secretion in its lumen. The submucosa is also injected and infiltrated with round-cells, and the follicles are distinctly swollen. I do not agree with Talamon, who believes that the whole wall in all its layers, including the muscular coat, is always affected in appendicitis. Siegel reports cases in which the appendix showed nothing either macroscopically or microscopically except the ordinary signs of catarrh, but in which, as a result of dilatation, the appendix had for years been the seat of very violent pains. On the other hand, it is true that in some cases of simple appendicitis there is marked hypertrophy of the muscular coat. As this hypertrophy is by no means constant in ordinary chronic catarrh of the bowel, it must be assumed that special conditions are present in the case of the appendix. This, I believe, is explained by the fact that in chronic catarrh of the appendix the lumen is often narrowed, and, as a result, its contents are retained in the appendix; when this occurs, hypertrophy of the muscular coat may be expected, as in other forms of stricture of the intestine, from the increased work involved in the efforts of the muscular fibers to force the retained contents through a stricture.

As a result of the inflammatory hyperemia and swelling, the appendix becomes stiff, thicker, and more rigid. The lumen contains thick grayish or yellowish material, mucus, or secretion from the wall of the appendix, and occasionally fecal material, which may be either liquid and soft or sufficiently solid to form small fecal concretions, rupture of some of the blood-vessels often occurs, and leads to ecchymoses in the mucous membrane and to a reddish and hemorrhagic color of the contents, while superficial ulceration of the mucous membrane is comparatively common (erosion ulcers).

In cases that run a rapid course these changes may all pass away, for its clinical course shows that appendicitis may end in perfect recovery. This is probably due to the exit of bacteria or other inflammatory agents through a patent lumen into the cecum; this can happen only when the organ is free from kinks and obstruction of its lumen by swelling of the mucosa.

Very often, however, probably in the majority of cases, complete resolution does not occur, and the process runs a different course and gives rise to the following morbid changes:

Occasionally the appendix merely shows the changes of chronic catarrh—viz., a slate-gray color of the mucous membrane. The muscular and submucous coat may be normal microscopically, while small-cell infiltration or areas of fibrous hyperplasia and sometimes masses of blood-pigment are found in the mucosa. These signs of chronic catarrh have often been found accidentally in persons who, at least as far as their statements go, had had no definite symptoms of appendicitis

or of perityphlitis during life. In these cases the peritoneum and the mesentery of the appendix may be perfectly normal, there may be no fecal concretions, no kinking, stricture, or adhesions of the organ. In such cases it is difficult to decide whether the condition is one of simple independent appendicitis which began acutely and then ran a chronic course, or the catarrh was chronic from the start and was not primary and confined to the appendix, but merely an extension of a general catarrhal condition of the cecum. In the majority of cases I believe that the latter explanation is the correct one. Riedel has recently drawn special attention to these extremely insidious and clinically latent forms of appendicitis; in 1 case, in operating for an acute attack, and that the first, of appendicitis, he found complete stenosis, although the patient, a boy of twelve years, had not up to that time had any symptoms pointing to such changes in the appendix.

In other cases appendicitis, which has become chronic and led to ulceration of the mucous membrane (catarrhal or erosive ulcers), may eventually lead to a very favorable termination—viz., obliteration.

[Lockwood,[1] who has made a special study of the morbid changes in the appendix, states that the stricture is in most instances due to organization and cicatrization of inflammatory exudation in the submucous and subperitoneal tissue, and is not so often due to cicatrization of an ulcer as is sometimes stated.—Ed.]

This is exactly the same result as that described above in physiologic subinvolution of the organ. In such cases of obliteration of the lumen the mucosa and the follicles disappear, and the whole appendix is converted into a solid, hard, cord-like structure that may either be uniformly thick throughout or may show small enlargements here and there.

[In a case of chronic catarrhal appendicitis with marked narrowing there were two lumina visible to the naked eye, and each with its own set of tubular glands, follicles, and muscularis mucosæ (Rosenberger[2]).—Ed.]

In such cases the appendix is generally found imbedded in a mass of thick, chronic, peritoneal adhesions. It is hardly necessary to call attention to the importance of differentiating this inflammatory obliteration and degeneration of the appendix from the physiologic obliteration already mentioned.

In a minority of the cases the process terminates in a different condition, which is known as *hydrops of the vermiform appendix.* In these cases the lumen is locally obliterated, presumably as the result of ulcers due to catarrhal ulcerative appendicitis (with or without stercoliths), or occasionally by kinking or constriction of the appendix by external factors, chiefly chronic peritoneal adhesions. These internal cicatrices are comparatively common near the cecal orifice, but they may be found in any part of the organ; this also applies to constrictions due to external causes. The cicatricial stenosis is not always complete, and there is sometimes a minute communication between the lumen of the appendix

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[1] C. B. Lockwood, *Appendicitis, its Pathology and Surgery*, p. 103.
[2] R. C. Rosenberger, *American Medicine*, July 18, 1903, vol. vi., 93.

and the cecum. When obliteration of the lumen occurs in the appendix, especially when it contains no virulent bacteria, no suppuration follows. Cystic dilatation of the distal part of the organ does not necessarily follow, and does not occur when the secretion present in the organ is absorbed or when the mucous membrane is destroyed, and, therefore, unable to manufacture any secretion. Occasionally, however, secretion is more rapid than absorption ; and accumulation of the secretion and cystic dilatation of the organ naturally and inevitably result. The pressure within the organ produces gradual atrophy of the mucosa, while the connective tissue surrounding the appendix, which is now converted into a retention-cyst, increases.

[According to Lockwood, the submucous coat may be edematous, the walls show inflammation, and, though the mucous membrane is thinned and may disappear, it is probable that a mucocele cannot develop unless the epithelium persists. The contents may be sterile.—ED.]

The size of these cysts may vary from that of a cherry to an adult's fist. Guttmann describes a cyst 14 cm. in length and 21 cm. in its largest circumference. The contents may be tough and gelatinous or white and mucous.

[Latham [1] described and figured a dilated appendix obliterated at its cecal end which was filled with small, round, white, somewhat transparent bodies, the largest of which was about the size of a dried pea. The wall of the appendix was thickened, the mucous membrane had almost disappeared, and it contained no lymphoid tissue. The bodies did not give the reactions for mucin or for fibrin.—ED.]

In some cases the contents are watery, serous, and of a whitish or yellow color; this condition has give rise to the term hydrops of the vermiform appendix. The cysts may be pedunculated, sessile, and attached to the cecum, or have an obliterated pointed tag at their distal extremity, according to the point where the lumen has been obliterated.

[Treves [2] says these cysts may rupture and set up perityphlitis, and that fibrous cords found in the place of the appendix may be thus explained. Lockwood,[3] however, is unable to confirm the statement that the cysts burst.—ED.]

It has already been said that complete resolution in simple appendicitis is very often prevented when the secretion is retained and the exit of the inflammatory factors and their products into the cecum is difficult or impossible.

A number of factors may lead to retention of its secretion in an inflamed appendix. When the muscular coat is inflamed and its functional activity thus impaired, the expulsive powers of the appendix become insufficient. In other cases external mechanical conditions, such as twists, kinks, abnormal position or fixation of the organ, give rise to retention. The swelling of the mucosa itself and Gerlach's valve may also prevent the exit of the contents of the appendix. The secretion

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[1] A. Latham, *Trans. Path. Soc.*, vol. xlviii., p. 86.
[2] F. Treves, *Allbutt's System of Medicine*, vol. iii., p. 609.
[3] C. B. Lockwood, *Appendicitis, Its Pathology and Surgery*, p. 186.

itself, after it has once begun to accumulate, favors further retention in two ways—*i. e.*, in the first place, by exerting mechanical pressure on the wall of the organ; in the second place, by serving as a good culture-medium for the growth of bacteria.

The anatomic conditions seen under these conditions are the following ones :

The appendix, especially when the disease is chronic or when there are many relapses, eventually becomes greatly thickened, and may become as big as the little finger or even larger. The organ rarely remains straight, and as a rule there are bends and kinks at one place or another ; or there may be narrowings or constrictions in some part of the appendix. There are almost always some changes, though of very varying degrees, in the peritoneal coat, which will be described below. The muscular coat may remain unchanged, but is as a rule thickened, either from inflammatory infiltration or from true hypertrophy, due to overactivity of the muscle-fibers which is the natural result of the attempt of the organ to expel its contents (compare the section on Intestinal Stenosis). The submucosa is thickened and occasionally fibrosed and traversed by numerous lymph- and blood-vessels, with thick walls and a narrow lumen, which may be completely occluded by thrombi ; the mucosa is reddened, swollen, loose, and frequently covered with small hemorrhages ; sometimes the mucosa is completely absent in parts even when there are no fecal concretions ; in other words, there may be ulceration of the mucosa in one, two, or even more places. The organ contains mucopus or sanious or dark-colored stinking material. This constitutes *suppurative appendicitis.*

When the organ is kinked or its lumen is narrowed in some other way, the mucous membrane beyond the stricture shows a number of serious changes. When, as sometimes occurs, there are two strictures, the mucous membrane of the proximal part of the appendix close to the cecum may remain comparatively normal, while the middle portion shows the changes just described, and the peripheral segment may be simply dilated with catarrhal changes in its mucous membrane, which is fairly pale ; the latter portion contains nothing but simple secretion, and, as a whole, resembles the condition of hydrops of the vermiform appendix.

When pus collects in the appendix, the condition is spoken of as *empyema of the vermiform appendix*, but this condition is rarely seen, since the appendix either ruptures or is removed by operation before suppuration has led to a collection of pus. As in hydrops, there is a closed sac formed by the wall of the appendix, which contains pus instead of mucous or serous fluid. These sacs vary in size, but never become so large as in hydrops, since an empyema either ruptures spontaneously or is operated upon. The whole length, or only part, of the appendix may be involved. In rare cases, when the empyema is not operated upon, ulceration and perforation may occur, even in the absence of a fecal concretion. It is impossible to say what interval must elapse before the empyema develops ; but speaking generally, the process is

sometimes fulminating and sometimes exceedingly slow; this probably depends on the nature and virulence of the micro-organisms present. Empyema is comparatively common in tuberculous appendicitis, and usually runs a chronic course.

Lastly, another form of appendicitis must be mentioned, which is clinically the most important of all, since it is the most dangerous and the most rapidly fatal. In this form there are ulceration and gangrenous destruction of the wall of the appendix—*phlegmonous, " diphtheric,"* or *ulcerative* and *gangrenous appendicitis.*

In the preceding paragraphs attention has repeatedly been called to the occurrence of ulceration in the appendix, and it has been shown that ulceration, particularly when merely catarrhal, and even occasionally when due to impaction of a fecal concretion, may either heal completely or only produce local changes limited to the appendix (obliteration, cicatrization, with secondary hydrops or empyema). In some cases, however,—and these are clinically the most important ones,—ulceration or gangrene in the wall of the appendix leads to complete destruction, and, except in very rare cases, to perforation of the appendix; this well-known condition is described as *perforative appendicitis.* The morbid changes in the appendix vary according to the exact mode of perforation; the most characteristic appearances occur in cases of acute perforation due to necrobiosis or gangrene. The organ in these cases is, as a rule, rigid and considerably, sometimes enormously, thickened. The external surface—the condition of the peritoneal coat will be dealt with later—is intensely but not uniformly red. Either the tip of the appendix, the adjacent part, the middle, the proximal extremity, or as much as a third or even a half of the organ may be discolored, either in one or in several places, and appear black, greenish black, or yellowish in color, or show a combination of several of these tints. As the bulk of the organ is usually red, the contrast of colors thus produced is very striking in appendices removed during operation and examined fresh. There is a perforation in some part of the appendix passing into its cavity; it may be very small, and sometimes is barely visible; it may, however, measure from one-half to one centimeter in diameter, and in rare instances even more. It may be round or irregular in shape, and sometimes there are two or more perforations. Immediately around the perforation the wall of the organ is not red, but discolored, black, or yellowish, and gangrenous. When due to a fecal concretion, the latter may sometimes still be felt in the opening, or it may still be inside the appendix, or, lastly, it may have worked its way out of the perforation into the pus around it. The situation of the perforation and that of the concretion inside the appendix do not, however, always correspond; in fact, not uncommonly the two are far apart and the concretion may be above or below the perforation. When opened, the appendix is found to be swollen, softened, and sometimes infiltrated with minute abscesses. The mucous membrane shows the same changes in color as the external surface of the organ; occasionally the yellow color and the necrotic character of the mucous mem-

brane are so marked that it was formerly spoken of as diphtheria of the appendix; the contents of the appendix may have completely escaped through the perforation or may still be found inside the organ, and usually consist of thick, offensive pus.

The whole of the appendix may become necrotic within twenty-four to forty-eight hours, the course of the disease being extraordinarily rapid and acute. In these cases the whole appendix becomes gangrenous, sloughs off, and is found as a dirty-green or black slough in the abscess that forms. In circular necrosis the line of demarcation may also form in any part of the organ, and, as a result, the lower third or half of the appendix alone sloughs off. Fowler reports a very remarkable case in a young man of eighteen who developed general peritonitis seven days after the onset of an attack of ordinary appendicitis; at the laparotomy all that remained of the appendix was a worm-like tube of the mucous membrane, the other coats having completely sloughed off; this tube of mucosa was not perforated, although it contained a fecal concretion about half way down. The peritoneal cavity contained an abundant quantity of seropurulent exudate.

Rose raises the question, whether possibly some of the most virulent cases of appendicitis may not follow on complete (hernial) protrusion of the organ into the ileocecal pouch of peritoneum (recessus retrocæcalis, ileocæcalis inferior, ileo-appendicularis).

Various other peculiar conditions may also be met with. Frankfurter calls attention to the fact that occasionally in circumscribed gangrene there may be a connecting bridge between the distal and the proximal parts of the appendix. Roux reported a remarkable case (*loc. cit.*, Case XLIX.) in which the appendix was divided into two halves imbedded in peritoneal adhesions; the two parts were supplied by two different arteries that ran in opposite directions, one of them being the normal artery of its mesentery, the other apparently a newly formed vessel running from the region of Poupart's ligament into the mass of adhesions.

Very peculiar conditions are also seen when perforation or gangrene occurs in an organ that is narrowed or occluded in some part of its course. Here the distal or the proximal extremity may be perforated and show all the typical changes of this accident, while the other portion of the organ may be perfectly normal. The morbid change may thus occasionally be limited to the base or to the tip of the appendix. Sonnenburg has described a very instructive case of this kind in which the first 6 cm. of the appendix were perfectly normal and extended in an arch free through the peritoneal cavity, while the tip, which measured 3.5 centimeters, was in an abscess behind the cecum, which was perfectly normal. In the thickened termination of this appendix there were a perforation no larger than a pin-head and a fecal concretion. There is considerable variation in the direction of the perforation in different cases and in the part of the abdomen into which the contents of the appendix, with or without a fecal concretion, pass. Usually the contents of the appendix pass either into a preëxisting pouch formed by

peritoneal adhesions, or into a perityphlitic tumor of the kind which will be described later on. When the perforation occurs very rapidly, and before a protective wall of peritoneal adhesions can form, the contents pass into the general peritoneal cavity. When the appendix becomes adherent to some other organ, special forms of perforation may occur. That perforation may occur into the cecum is not surprising after what has been said above; but Fürbringer reports a case in which an exceptionally long appendix perforated into the duodenum; in a case of Favre's (quoted by Sonnenburg) the tip of the appendix was found floating in the intestine, and the contents of the appendix passed into the bowel. Bossard described a case in which the appendix perforated into the urinary bladder, and a fecal concretion which passed into the bladder subsequently formed the nucleus of a large vesical calculus.

How does perforation occur? In the great majority of cases—and there can be no doubt about this—it depends on the presence of fecal concretions in the appendix. Less frequently the perforation is due to other foreign bodies in the organ. Necrosis from pressure and the action of bacteria combined produce perforation which may occur either rapidly or, more commonly, slowly. Hard fecal masses and typhoid, tuberculous, or dysenteric ulcers are responsible for the majority of perforations of the cecum itself. Whereas formerly fecal concretions were always considered to be the cause of perforation of the appendix even though they were not found at the necropsy (for it was then assumed that they had either become absorbed by the perityphlitic pus or had been overlooked), it is now so well known that they are absent in many cases that it is unnecessary to support this statement by statistics. The "reliable observations" that Volz required as proof of the assertion that "perforation of the appendix occurs in the absence of concretions" (with the exception of typhoid, tuberculous, or dysenteric ulcers) are very numerous.

The course of the disease, the sequence of events, and the change in the appendix which leads to perforation in the absence of concretions and of the specific forms of ulceration cannot always be made out from a study of the pathologic anatomy.

In exceptional cases empyema of the appendix may perforate and rupture in the same way as in any other form of abscess. As in perforation from fecal concretions, the process takes some time and is usually chronic. In some recorded cases, which are clinically the most important, the whole disease runs a very rapid course from the first onset of symptoms to the occurrence of perforation. In these cases there may have been a previous attack of appendicitis, and it is generally impossible to decide whether this was simply catarrhal or a chronic purulent process. At any rate, from a clinical point of view, when a recurrence occurs in cases which appear to have been cured, the disease runs an acute course and rapidly ends in perforation. Occasionally, however, the very first attack is perforative. This result, of course, requires special conditions, the most important of which is probably in most cases intensified microbic virulence. This presumably

damages the blood-vessels, especially those in the submucous coat, and sets up arteritis, phlebitis, and thrombosis, or produces a most extensive small-cell infiltration of the tissues which compresses the blood-vessels. The vascular disturbances then produce further nutritional changes, such as softening, gangrene, and eventually perforation. The process may be compared with cutaneous erysipelas, in which variations in the virulence of the streptococci determine whether a simple inflammation or gangrenous destruction of the skin results. It appears, however, that gangrenous inflammation is more frequent in the appendix than anywhere else in the body.

Gangrene and perforation are sometimes favored by certain anatomic peculiarities of the appendix; thus there may only be one artery running along the whole length of the appendix, which does not send off any branches until it gets to the tip of the organ, where a few arterioles pass to the submucosa. Fowler reports a case in which the lumen of the vessel was obstructed at a point about one-third from its origin at the base of the appendix; there were appendicitis, obstruction of the blood-vessels supplying the mucous membrane, capillary stasis, necrotic changes in the submucous coat, infection of the submucous coat, endarteritis, and thrombosis, all these changes appearing within ten hours after the onset of the first symptoms of the disease.

Lastly, to avoid any false impression, it must be stated once more that deep ulcerations and extensive gangrene may occur in parts of the appendix without necessarily or always leading to complete perforation or separation of the organ; or, to put it more correctly, it may be said that severe local symptoms or evidence of sepsis and diffuse peritonitis may necessitate operation or kill the patient before perforation has had time to occur. Thus, to quote one out of numberless examples, Beck found, eleven hours only after the first sudden onset of clinical symptoms, a condition of purulent ulceration extending to the serous covering, and Pozzi, at an operation performed on the second day of an acute attack of appendicitis, found gangrenous areas in the appendix without any fecal concretions.

These morbid conditions of the appendix are clinically important mainly, if not entirely, from the spread of inflammation to the peritoneum of the appendix, of its immediate vicinity, or even of the whole abdominal cavity. Changes in the peritoneum cannot be separated from those in the appendix, and it is only for the sake of clearness that the latter have been described separately; in the following paragraphs the peritoneal changes accompanying appendicitis will be described. The description of peritonitis in connection with appendicitis—the most common cause of peritonitis—applies, anatomically speaking, with equal force to that rare condition—peritonitis undoubtedly due to ulceration of the cecum.

There are, it is true, some exceptional cases in which peritonitis is completely absent even on the surface of the appendix. This phenomenon cannot always be explained in the same way, but it is possible to suggest a reason, and in some cases to prove why the peritoneum re-

mains free. In order to avoid repetition this particular point will be dealt with here. Complete absence of peritonitis is most commonly seen in cases of the simple superficial catarrh of the appendix described above. This chronic superficial catarrh resembles ordinary intestinal catarrh, and, like it, does not produce any change in the peritoneum.

In cases again in which perforation or gangrene runs a fulminating course and an operation is performed or death supervenes from septic infection so early that peritoneal changes have really no time to develop, and the peritoneum surrounding the appendix appears normal, the absence of peritonitis is apparently due to the rapid course of the disease.

Again, several cases of chronic ulcerative appendicitis with or without enteroliths have been reported in which the serosa was quite smooth and there was a complete absence of adhesions in the appendiceal region. The explanation of this peculiar phenomenon is not at present clear, and will probably be obtained only by the most careful histologic and bacteriologic investigations of each individual case of this kind.

Apart from these exceptional cases, changes in the peritoneum surrounding the appendix are the rule in all forms of appendicitis; and in rare cases there may be diffuse peritonitis.

The anatomic forms of peritonitis seen in appendicitis are exceedingly varied and numerous.

In a recent and first attack of simple appendicitis there should *a priori* be only recent inflammation of the peritoneum covering the appendix, without any adhesions in the neighborhood. Such cases do actually occur; Rose reports one [1] with sudden onset on December 14th, midday; operation followed on the morning of the 16th. The thickened appendix, the mucous membrane of which was completely covered with a diphtheric-like deposit, was covered externally at the end with a layer of recent fibrin, but was quite free from adhesions.

The peritoneal changes are often comparatively slight in cases of recurrent appendicitis which have been operated upon. It is true that neighboring parts are frequently involved in the process, but in some cases the changes are confined to the peritoneal covering of the appendix and its mesentery. Beck reports a case in which, after two attacks in one year, he found at the operation, eleven hours after the onset of the first, but unimportant clinical symptoms, a fine layer of fibrin covering the appendix and adherent to the cecum and the peritoneum of the mesentery. The appendix contained pus, necrotic tissue, and blood-clot. There is no marked difference in the anatomic conditions in cases that are examined postmortem and during life, or even between cases operated upon during the interval between two attacks or at the so-called recurrence.

The peritoneum of the appendix and, as a rule, its mesentery are red, show numerous dilated vessels, and become thickened in proportion to the duration, so that eventually these two structures may appear as tough cicatricial bands. Cord-like thickenings in its mesentery may cause kinking of the appendix. The mesentery may, however, be

[1] *Loc. cit.*, pt. ii., case 19.

unaffected, the process being limited to the peritoneum of the appendix, but the reverse is rare. Not uncommonly the inflammatory changes are limited to half or a third of the peritoneal surface of the appendix. At the same time the appendix may be perfectly free or only slightly restricted in its movements, and there may be no adhesions whatever between the appendix and neighboring parts, though this is exceptional.

As a rule, the inflammation spreads from the peritoneum covering the appendix to the adjacent peritoneum, and leads to the formation of thick adhesions which unite the whole or considerable portions of the organ to other organs (chronic adhesive peritonitis). The appendix may thus become imbedded in cicatricial tissue; in other cases the process is limited to the formation of thin cords and bands which attach the appendix to other parts and fix it in certain positions.

Moszkowicz doubts this view of the origin of adhesions, which, in case of extreme inflammation or perforation of the appendix, encapsulate the inflammatory swelling (the perityphlitic tumor) and form a protective wall for the general cavity of the peritoneum. His descriptions touch upon the pathogenesis of circumscribed intraperitoneal abscess as well as that of diffuse peritonitis. He bases his opinions upon the following observations—viz., that at an operation during the first two or three days of an attack of appendicitis there are not only advanced changes in the appendix, but pus and turbid serum in the peritoneal cavity between the coils of the intestine; in these cases encapsulation around the appendix is entirely absent. In connection with such cases Moszkowicz criticizes the above generally accepted view of the origin of circumscribed perityphlitis. According to him, early operations prove that perityphlitic inflammation is not at the commencement, or at least not always, circumscribed. "Infection of the appendix brings about in a short time, with or without perforation, an exudation in the neighborhood of the appendix, which at first is not localized, but in many cases probably affects sympathetically the greater part of the abdominal cavity. A similar condition is seen in severe septic infection of an extremity. Starting from a small focus, in a very short time edema of the whole limb follows. Corresponding to edema, there is in the abdominal cavity a free serous exudate, rapidly followed by a fibrinous one. After a time the process becomes localized, the exciting inflammation being most vigorous in the region of the appendix, where, under its influence, firm adhesions form and the infective mass is shut off. The rest of the peritoneum slowly quiets down."

From the different positions that the appendix may normally occupy it is clear that it may become fixed by peritoneal adhesions in a number of different parts of the peritoneal cavity. It is most frequently adherent to the cecum, next to the right iliac fossa, loops of the small intestine, and rarely the colon. In women it is frequently united to the right broad ligament (the converse, of course, may occur, and disease of the Fallopian tube, etc., on the right side may lead to adhesions with the appendix when the latter organ is perfectly sound—Foges).

The peritonitic process as it extends may then further lead to the formation of adhesions between other organs, such as the coils of the small intestine, the cecum, the colon, and the broad ligament. The mesentery is often adherent either to the appendix itself or to the mass of adhesions surrounding it. Rare cases are on record in which the appendix has been found adherent to the rectum, the bladder, and the duodenum.

The importance of these adhesions varies : sometimes they cause further complications and are consequently dangerous ; thus they may favor kinking, constriction, and strangulation of loops of intestine, interfere with the onward passage of the intestinal contents, narrow and even completely obliterate the lumen of the bowel ; they may also give rise to the clinical manifestations imitating recurrences of perityphlitis. On the other hand, these adhesions may serve a distinctly useful end by forming a protective barrier in cases of perforation and thus preventing the entrance of the contents of the appendix into the general peritoneal cavity.

A description will now be given of the anatomic changes implied by this term perityphlitis, or the inflammatory swelling which develops acutely around the appendix, and, as a rule, involves the cecum as well ; this is the iliac phlegmon of ancient writers, the perityphlitic tumor and abscess. It is generally preceded by a number of attacks of simple appendicitis, but occasionally the appendix and the cecum during the first attack become surrounded by a large or small and usually firm, solid swelling. What are the anatomic characteristics of this swelling and what is its origin ?

Postmortem and clinical observations have shown that in many cases these swellings have been due to suppuration or a true intraperitoneal abscess. It is, however, exceptional to come across abscesses of such a size as to give rise to these large swellings ; and this probably only occurs in advanced cases. As a rule, the swelling subsides without any operation, and thus shows that it was not an abscess. The moot points about the significance and origin of these tumors cannot be settled by postmortem examinations alone, since only the serious and advanced cases are thus available. The main question is what factors determine the development of these swellings in the early stages of the disease ?

The majority of observers agree that the infiltration that will be described below plays an important part in the formation of perityphlitic swellings ; discussion largely turns on the question, as Sahli has pointed out, whether these tumors contain a " pus nucleus," which must be considered the prime cause of the swelling, or whether these swellings (that are not simulated by masses of feces) ever develop in the absence of suppuration. In addition, fecal plugs play an important rôle ; before entering into a discussion on these points the actual anatomic changes in these cases, and all the factors which conceivably are concerned in the formation of these perityphlitic swellings will be dealt with.

The appendix, the wall of the intestine, the cecum, and certain loops of the small intestine may all become swollen, and the peritoneum of all these parts may become thickened by the formation of fibrinous de-

posits. In some cases there may be phlegmonous infiltration of the
fasciæ and of the abdominal muscles. Part of the omentum may
become thickened and add to the bulk of the tumor, as pointed out by
Roux and von Recklinghausen (seé Frankfurter).

Sonnenburg, from observations made during operations, calls atten-
tion to the fact that the size of the swelling often depends in part on
the presence of serous or serofibrinous exudates, but, according to him,
this is not so frequent in perityphlitic tumor proper as in simple appen-
dicitis, in which condition "the extended dulness which in many cases
appears and disappears so rapidly may be explained by the formation of
serofibrinous exudates." Renvers, Kümmel, and others also assume
that encysted serous exudates are concerned in the formation of the
swelling, and it appears to me that observations made during operations
prove that this is often the case.

Sahli vigorously opposes this view, and believes that exudation does
not, or only in very exceptional instances, play a part of any importance
in the formation of the swellings. But his position is greatly weak-
ened. Lennander,[1] in a patient suffering from a first attack of acute
perityphlitis with fever, found on the fifth day an area of resistance as
large as an adult's hand. At the operation, on opening the peritoneum,
which was greatly thickened, a cavity lined by fibrin and containing
serum and the appendix was found; the distal part of the appendix
was gangrenous, but there was no perforation or fecal concretion; the
patient recovered.

Lastly, the tumor often appears to be larger from fecal accumulation
in the cecum, but this factor does not often account for the whole of
the swelling. It may, of course, happen that a case of simple appendi-
citis, with pain and fever, but without any serious affection of neigh-
boring parts, may be accompanied by an accumulation of feces in the
colon; in such cases, however, it cannot be said that the latter consti-
tutes the inflammatory tumor, for such a tumor does not exist; the
fecal tumor can at best simulate an inflammatory tumor of the peri-
typhlitic region.

Krafft was the first to enunciate the dictum that "stercoral appen-
dicular perityphlitis is always accompanied by the formation of an
abscess; that the circumscribed peritonitis that always develops around
a perforated appendix is always purulent is now universally accepted.
Sahli, however, as has been seen, advocates the view that the perityph-
litic tumor proper, even in the absence of perforation and of fecal con-
cretions in the appendix, always contains a purulent focus. This view
must be understood as follows: the swelling is probably formed by ad-
herent loops of intestine, fibrinous exudate, etc., but the cause of all
these changes is suppuration, which need not be extensive. It is possi-
ble, of course, for the area of suppuration to extend rapidly and so to pro-
duce a large and distinctly fluctuating abscess, but, according to Sahli,
the perityphlitic tumor in its early stages often contains a very minute
quantity of pus. Aufrecht and Sahli have drawn a very apt comparison

[1] *Loc. cit.*, p. 54, observation 8.

between the tiny pocket of pus and the large amount of swelling and infiltration in perityphlitis and in furuncle. The purulent focus in these cases is either inside the unperforated appendix or in the perityphlitic swelling.

At the same time I am willing to admit that a perityphlitic tumor may be formed by inflammatory thickening of the tissues without the presence of any pus, resembling in this respect erysipelas, where the skin may be enormously swollen from inflammatory exudation without a trace of suppuration. The conditions, however, found in early operations indorse Sahli's view, though there are not sufficient grounds to regard this as absolutely constant. This idea is somewhat startling, and has met with much opposition. The chief argument against it has already been mentioned, namely, that many large perityphlitic tumors pass away without operative interference and without any proof of perforation, whereas it is usually believed that spontaneous recovery is out of the question when pus has once formed. How, then, is Sahli's view, which I indorse, to be substantiated? how is the assumption of a purulent focus to be justified, and what becomes of the pus present in the majority of the cases of perityphlitic tumor?

The chief proof is that in the great majority of cases of perityphlitic tumor, even in those operated upon early, pus is present either in the appendix itself or in the inflammatory swelling. Contemporary literature shows that many cases are now operated which would not have been treated in this way in the past. In these cases the possibility of a spontaneous recovery cannot be denied with certainty, and, therefore, according to the general view held as to the absorption of pus, no pus would be expected. These operations, however, show the presence of pus, and this justifies the conclusion from analogy that pus is also present in other and similar cases, which, though not treated surgically, recovered spontaneously. This view is shared by other modern authors; thus Rotter, from a statistical investigation, arrives at the conclusion: " I must consider Sahli's view correct if the words ' during an acute attack ' are added."

The question what becomes of the pus must be answered differently in the different cases. The way in which abscesses are naturally got rid of is by discharge of pus into the intestine or elsewhere. This, however, applies to definite abscesses of some standing, comparatively rare, easily recognized, and different from the solid, hard, perityphlitic swellings, which, as we know, may disappear in an astonishingly short time, and are, as a rule, purulent in origin. What becomes of the pus in these cases? According to Sahli, in these cases it usually passes into the intestine through the appendix, which acts like a drainage-tube. It must be admitted that it is quite possible for such small quantities of pus as these to escape detection in the stools, so that a failure to find pus in the feces would not militate very strongly against this supposition, and, on general principles, no other objection can be raised against this idea. On the other hand, it is very difficult or impossible to bring any proof that this does occur, and I, therefore, agree with writers, such

as Rotter, that there is some other method in most cases, and that the pus is usually absorbed by the peritoneum ; Sahli also mentions this method of resolution, but considers it subordinate in importance and in frequency to resolution by direct drainage.

It has been proved by animal experiments (see section on Peritonitis) that the peritoneum possesses the power of absorbing small quantities of pus when the bacteria producing it are not too virulent, and that it may even absorb larger quantities of sterile pus. Actual experience in perityphlitis, moreover, shows the correctness of this assertion. Thus a number of observations made postmortem and during operations show that small abscesses have been absorbed, for the appendix or fecal concretions are found embedded in masses of calcareous or fibrous tissue. This view as to the absorption of pus is also supported by the observation (Renvers and others) that removal of an extremely small quantity of pus by aspiration (thus Rotter, *e. g.*, removed 1 c.c. from an abscess that was as large as a hen's egg—*experimenti causa*) may be followed by recovery and complete absorption. Sonnenburg's view that in these cases the pus was accidentally aspirated from an empyema of the appendix, and that this produced resolution of periappendicular edema appears improbable from the relative rarity of empyema of the vermiform appendix. Lastly, consideration of the very small quantities of pus often removed by an operation—1 c.c., 10 c.c., 1 dram, half an ounce, or an ounce—shows that the possibility of spontaneous absorption of pus cannot be denied, assuming, of course—and this seems justifiable—that the virulence of the bacteria in the pus is only slight. In this way, then, an acute perityphlitic tumor may disappear and recovery take place. To what extent, if at all, this can be regarded as permanent will be considered below.

Perityphlitic tumors may only partially disappear and leave an indurated mass of cicatricial tissue behind. Occasionally the thick fibrous capsule formed in these cases is subsequently found, sometimes after an interval of years, to contain a thick, pasty, or even fluid pus, absorption and entrance of pus into the circulation, and all harmful results to the body generally, having been prevented by the barrier of dense cicatricial tissue.

This description has so far been confined to large swellings composed chiefly of edematous infiltration around a suppurating appendix, and containing only a small quantity of pus. In a number of cases, however, as already pointed out, the condition is very different and there are abscesses of considerable size, which may develop slowly or rapidly. These abscesses in the neighborhood of the cecum may be large or small, unilocular or multilocular, and may have a number of fistulous passages which extend far into the surrounding tissues ; their walls consist of more or less firm and dense connective tissue or of discolored membranous adhesions, and the abscesses are either strictly localized or with indefinite walls. The pus is rarely pure, and, as a rule, is more or less putrid, grayish or yellowish-brown in color, not unlike diarrheic mucopurulent dejecta ; sometimes it is sanious, occasionally hemorrhagic and

red, or even black from the presence of decomposed blood.   Sometimes
the abscess contains gas, either formed *in situ* or derived from the bowel.
The gangrenous appendix is sometimes found loose in the abscess as a
discolored slough ; in other cases the appendix, either perforated or gan-
grenous, is still in connection with the cecum or adherent in some other
part.   Fecal concretions are also quite frequently found in the pus.   The
adjacent coils of intestine are adherent and covered with adhesions or
with purulent, dirty-looking masses ; between the coils there are collec-
tions of turbid yellowish pus.   The mesentery is, as a rule, adherent,
and the purulent and gangrenous process may involve the abdominal
walls.

These morbid appearances were formerly by no means rare, but they
are not so often seen now and only in neglected or in exceptionally acute
cases ; as a rule, cases are operated upon before these changes have
had time to develop.

It is also noticeable that in cases which run a somewhat slow course
there are often not one, but several, abscesses.   Thus there may be one
in the ordinary position and another behind or in front of the bladder,
near the kidney or elsewhere.   This condition merges into progressive
purulent peritonitis, which is described more fully elsewhere.

The position of the perityphlitic tumors is constant, in the majority
of cases, being generally in the right iliac fossa.   But there is consid-
erable variation in the position of the tumor in this region, correspond-
ing to the position of the appendix and its origin from the cecum.
Abnormalities in the position of the cecum and the appendix, though
not very common, are clinically extremely important, since they may ac-
count for the presence of the perityphlitic tumor at a considerable distance
from the right iliac fossa, and thus give rise to very puzzling conditions ;
lastly, the spread of the suppurative inflammation has a great deal to
do with the position of the abscess.

There are several positions which the perityphlitic swelling may
occupy, although strict differentiations cannot be made.   It appears to
me, if it be desirable to make a list of these positions,—and from a
clinical point of view this is not without its advantages,—that the
position of the swelling can best be tabulated from its relative position
to the cecum, as Sonnenburg has done in the following scheme : when
the swelling is (*a*) antero-exterior, (*b*) posterior, (*c*) interior, (*d*) in the
pelvis.   In group (*a*) the coils of intestine situated in front of the cecum
are displaced, and the tumor touches the anterior abdominal wall, the
cecum forms the posterior and inner, and the iliac fossa the outer, wall
of the abscess.   In group (*b*) the abscess is bounded in front by the
posterior internal wall of the cecum, posteriorly by the back of the
abdomen ; in this group the swelling is often fairly high up and in the
region of the kidney, where it may produce bulging of the lumbar
region.   In group (*c*) the colon and the cecum form the lateral wall, the
mesentery and adherent coils of intestine and occasionally the bladder,
the inner and the lower wall of the abscess.   In group (*d*) the abscess
is in the pelvis, usually on the right side, but occasionally it extends to

the left or downward, and may lie between the rectum and the bladder or uterus. [According to Tubby,[1] pelvic abscesses are frequent in children from the fact that the appendix is often in the pelvis.—ED.]

Abnormalities in the position of the bowel, especially of the cecum and consequently of the appendix, may lead to unusual situations of the swelling. Curschmann, who has carefully investigated all the various conditions which can occur, describes a case of perityphlitis in which a painful tumor about as large as a man's fist was adherent to the abdominal wall below the right costal arch. At the autopsy this was shown to be an abscess cavity full of discolored and offensive pus, and shut off from the rest of the abdominal cavity by adhesions. The appendix, which was swollen, perforated, and contained a fecal concretion, was adherent to the floor of the abscess, and its tip reached up to the level of the costal arch. The cecum was folded over, so that it lay in front of the ascending colon, and touched the lower margin of the liver. The appendix may actually be found behind the liver when the colon is displaced, and if it becomes inflamed, may imitate a subphrenic abscess. The perityphlitic tumor may also be found in the center of the abdomen in the region of the umbilicus, and sometimes in the lower part of the abdomen on the left side, external to the margin of the left rectus muscle and below the umbilicus.

The length, direction, and a fixed position of the appendix, from adhesions due to past inflammation, are also important factors in determining the position of the swelling, which may be found in the umbilical region, in the pelvis, or even in the lower half of the abdomen on the left side, or in the left hypochondrium. Perityphlitic swellings are sometimes found in hernial sacs. The cecum with the appendix occasionally passes into inguinal hernias, even on the left side; and in rare instances into femoral hernias; if appendicitis supervenes, with or without perforation, an inflammatory tumor or abscess is found in the hernial sac. Sonnenburg believes that this is the same as the so-called "strangulation of the appendix"; Rotter reports three cases of true strangulation of the appendix in right-sided inguinal hernias.

[Eccles[2] has collected 9 cases of acute inflammation of an appendix in a hernial sac; in 7 of these cases it was the only organ in the sac. It has been suggested that the pressure of a truss on the appendix may dispose to inflammation; he refers to a case where a pin was found in an appendix in a hernial sac, and describes 2 cases where pieces of bone were present. He quotes 16 cases of acute strangulation of the appendix in a hernial sac, but considers that it is much less frequent than is usually thought.—ED.]

Lastly, the spread of the inflammatory process in various directions is of great importance in the position of the abscesses, which may be found at a considerable distance from its cause,—the appendix,—as the pus tracks to another part of the abdomen and forms an abscess there. The pus usually tracks along the retrocecal connective tissues, and here

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[1] Tubby, *Clinical Jour.*, vol. xxii., p. 411.
[2] W. McAdam Eccles, *St. Bartholomew's Hosp. Reps.*, vol. xxxii., p. 93.

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the question must be decided whether there is such a condition as a true paratyphlitis—*i. e.*, extraperitoneal inflammation originating from the appendix and confined to the retrocecal connective tissues—in other words, a true "phlegmon" of the retrocecal tissues in which the peritoneum is unaffected. This question is more theoretic than practical, particularly from the surgical standpoint, but even theoretically, the probability of a primary paratyphlitis is very small. For, according to the investigations of Bardeleben, Luschka, Maurin, and others, the cecum is, as a rule, almost completely covered by peritoneum, and the few cases that really start from the cecum and involve the neighborhood of this section of the bowel must almost invariably also involve the peritoneum, while, further, the appendix itself is rarely completely behind the peritoneum.

[The appendix, especially when it lies in a deep subcecal pouch or in a groove to the outer side of the ascending colon, may, as the result of inflammation and subsequent adhesions, become practically converted into an extraperitoneal organ.—ED.]

The anatomic conditions, therefore, almost exclude the possibility of an extraperitoneal inflammation. Körte, however, has shown by experiments that even when the appendix is completely surrounded by peritoneum, the retrocecal tissues can easily become infected. Having perforated the walls of the appendix exactly between the two layers of its mesentery, he injected a colored solution into its lumen and found that it spread into the connective tissue behind the peritoneum in the right iliac fossa, and from there into the perirenal connective tissues.

[The following observation illustrates this view to a certain extent : an appendicular abscess situated between the layers of the mesentery of the small intestine was apparently due to a perforation between the base of the appendix and the cecum by a round-worm (Bloodgood [1]).—ED.]

Aufrecht, indeed, states that perityphlitis is not an inflammation of the serosa, but genuine paratyphlitis, or inflammation of the retroperitoneal tissues, and that this inflammation is transmitted through the mesentery of the appendix.

When suppuration extends to the retrocecal tissues, the process takes on specially dangerous characters, for while the ordinary perityphlitic or intraperitoneal abscesses are cut off from the general peritoneal cavity by adhesions, and in this sense are clinically extraperitoneal, the retroperitoneal paratyphlitic suppurations have a malignant tendency to extend and often reach an enormous size.

The process easily spreads behind the colon by a broad track, and may thus give rise to lumbar or perirenal abscesses ; many so-called paranephritic abscesses are undoubtedly the result of appendicitis. Subdiaphragmatic abscesses and suppuration around the liver also readily result.

[Elsburg [2] collected 73 cases of subdiaphragmatic abscess due to

[1] J. C. Bloodgood, *Amer. Jour. Med. Sci.*, vol. cxxvi., p. 602.
[2] C. A. Elsburg, *Annals of Surgery*, 1901, vol. xxxiv., p. 729.

appendicitis, of which 35, or 48 per cent., were intraperitoneal, 20, or 27 per cent., extraperitoneal, and 18, or 25 per cent., doubtful. In 86 fatal cases of acute appendicitis there was suppuration in the subphrenic region in 7, or 8.13 per cent. (Christian and Lehr [1]). Barnard [2] has described the way in which a pelvic appendicular abscess may spread up behind the small intestine into both iliac fossæ, both loins, and eventually produce bilateral subphrenic abscesses. In 1 case he opened a pelvic, 2 iliac, 2 lumbar, and 2 subphrenic abscesses.—Ed.]

The process may further pass through the diaphragm and cause purulent or sanious pleurisy, or, when pleuritic adhesions are present, may perforate into the lung and bronchi; in the latter event spontaneous cure has been known to follow.

[Dieulafoy [3] has described " appendicular pleurisy " secondary to a subphrenic abscess, which has a special tendency to be putrid or to be accompanied by the rapid development of gas (pyopneumothorax) due to bacterial activity. It is remarkable that though the accompanying peritoneal inflammation may be fetid, it is not putrid or a mixture of pus and gas. Cultures from the peritoneum are not capable of producing gas, or when inoculated into a guinea-pig, a gaseous abscess, while pus from a putrid appendicular pleurisy is. Appendicular pleurisy is always (except for one—Doyon's—case) on the right side. Dieulafoy also describes a case of an enormous subphrenic abscess of appendicular origin which led to gangrene in the lower lobe of the right lung.—Ed.]

Aufrecht described a remarkable case in which the pus burrowed behind the spleen and collected below and behind the stomach.

On the other hand, the pus may pass downward and point above Poupart's ligament or on the inner side of the thigh, the hip-joint, or may track in front of the psoas or backward toward the spine.

[Suppuration may spread through the fascia into the substance of the psoas muscle, and may imitate hip-joint disease by producing flexion of the hip. Seventy per cent. of the cases of psoitis occur on the right side, and probably it has a close relation to appendicitis (J. Roger [4]). Delbet [5] and Barnard have described cases of suppurative psoitis of appendicular origin.—Ed.]

When the abscesses appear underneath the skin, areas of severe sanious suppuration, sometimes of enormous size, may develop in the cutaneous and the adjacent muscular tissues; they may extend as far as the scapula or the axilla, and destroy the muscles of the abdomen, the back, the chest, or the thigh; this has been seen in a number of cases, especially in former times.

What eventually becomes of these abscesses when not operated upon?

Attention has already been called to the formation of extensive adhesions which effectively isolate the organ and render any inflammatory changes in the appendix harmless to the general organism; this ter-

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[1] H. A. Christian and L. C. Lehr, *Medical News*, January 24, 1903, p. 149.
[2] H. L. Barnard, *Clinical Jour.*, vol. xxii., p. 364.
[3] G. Dieulafoy, *Cliniques Médicales de l'Hotel-Dieu de Paris*, 1901–02, vol. iv., p. 105.
[4] Jean Roger, *La Presse Médicale*, September 15, 1900.
[5] Delbet, *Progrès Médical*, December 13, 1902.

mination cannot, however, be relied upon to occur. At any time the process may be renewed, quite apart from the possibility of other unfavorable sequelæ, such as kinking, adhesion, and constriction of adjacent portions of the bowel. The formation of adhesions, moreover, only occurs when the amount of pus is small and probably never in larger abscesses, which, if they do not cause death by general pyemia, fever, and exhaustion, usually terminate differently—*i. e.*, by perforation ; the pus ulcerates through its capsule of peritoneal adhesions or into adjacent organs, and thus escapes from the abscess.

The description given above of the various possible positions of these abscesses makes it clear that perforation may occur in various directions and discharge into various viscera.

The pus is most often discharged into the intestine, and gives rise to the same clinical manifestations as in any other form of perforation of the bowel from without inward. The ulceration is more extensive on the outer than on the inner surface of the bowel. Perforation occurs most frequently into the cecum, and naturally, since the abscess is generally in close contact with this part of the bowel ; there can be no doubt that perforation of the cecum due to this cause was formerly often regarded as a perforation from within outward, and was used as an argument to prove that perforation of the cecum from within outward set up perityphlitis, and thus supported the old doctrine of primary perityphlitis of cecal origin. Perforation may also occur into the ascending colon, the ileum, the rectum, and even into the duodenum. Perforation of a perityphlitic abscess into various parts of the bowel must be clearly differentiated from those rare cases in which the appendix itself perforates into these organs. In order to illustrate the number and variety of the results which may occur, the following interesting case of Grawitz's may be quoted : Fecal concretion ; purulent gangrenous destruction of the appendix ; an abscess internal to and behind the cecum ; tracking of the pus up to the diaphragm ; perforation as large as a dime into the right pleural cavity ; purulent pleurisy ; in addition to the perityphlitic abscess, adhesions between many of the coils of the ileum and the cecum ; in the cecum and in the coils of the small intestine, numerous funnel-shaped perforations which were smaller on the mucous surface ; lastly, in the horizontal part of the duodenum, there were three perforations as large as beans communicating with the retroperitoneal abscess.

Perforation into the bladder is rare, and into the vagina still rarer ; perforation into the uterine cavity (Langheld) or into the gall-bladder (Sonnenburg) are pathologic curiosities ; perforation into the pleura, the lungs, and the bronchi has already been mentioned.

Perforation through the skin is seldom seen now, but was not so rare formerly and occurred either in the region of the iliac fossa, above the crest of the ilium, or in more distant parts of the abdomen, in the back, the lumbar region, or the thigh. The cutaneous fistulas, particularly when there are several, and they have formed spontaneously, usually have an angry look, being surrounded by suppurating tissues and communicate by sinuses with hollow organs or cavities inside the abdomen ;

further, from the shape of these sinuses, the exit of the pus is usually somewhat difficult.

Occasionally, these fistulas, especially the intestinal and cutaneous, may tend to produce a spontaneous cure of the abscess. On the other hand, the possibility must always be borne in mind that fecal matter may enter the abscess from the bowel, and that the character of the pus will thus become more virulent; the fact that the fistulous opening into the bowel is usually funnel-shaped, with the small end directed toward the bowel, probably explains why contamination with the contents of the bowel is not commoner. The chances of spontaneous cure are less favorable in cutaneous fistulas, and, fortunately, this accident is less frequent now than formerly. This can readily be understood, from consideration of the conditions described by Salzer, as follows : the fistulous tracks ramify and form spaces and pockets close to the intestine, become dilated, and form fresh abscess cavities ; while, again, the orifice opening into the intestine may be very narrow and may end in a number of narrow tracks, undermining the mucous membrane of the intestine.

[Adami[1] has given a good account of this process of exogenous ulceration of the intestine, in which there is incomplete destruction of the mucous membrane, so that pieces are left, bridging over fistulous tracks underneath.—ED.]

In general, treatment should be directed toward preventing the formation of these fistulas, but it is well to remember that if they do occur, the possibility of spontaneous cure cannot be excluded. Another group of perforations,—viz., those into the free peritoneal cavity,—however, always terminates unfavorably. This accident sometimes occurs while the abscess is quite small, from the adhesions which shut in and surround the abscess, giving way readily and early in the course of the process. In other cases perforation does not occur until the pus in the retroperitoneal spaces has collected so as to form a large abscess, which may, *e. g.*, extend as far up as the kidney. The result is always septic, purulent, or sanious peritonitis, and death almost always inevitably follows.

Finally, the process may involve the blood-vessels, arteries, and veins, causing erosion of their walls, inflammation, and its sequelæ. In this connection simple compression of the iliac vein by an abscess may be mentioned; it is followed by the usual results (edema of the legs, pulmonary embolism, etc.).

Ulceration of arteries—usually of the internal iliac—with fatal hemorrhage is rare, and only a few isolated cases have been recorded.

The veins, on the other hand, are comparatively often affected, and all observers of experience have seen such cases. In exceptional cases hemorrhage from ulceration occurs. Fowler has described a case. The usual change is phlebitis and thrombosis, generally of a septic, purulent character. As a rule, the process begins in one of the radicles of the portal vein, in the mesentery or wall of the appendix, and seldom starts in larger branches of the mesenteric vein. The purulent or septic phle-

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[1] J. G. Adami, *Montreal Med. Jour.*, vol. xxxii., p. 401.

bitis then runs its usual course, and extends by contiguity into the portal vein, and gives rise to multiple abscesses in the liver. Hepatic abscesses may develop before the thrombosis extends into the portal vein, and be due to emboli carried to the liver in the portal blood ; in fact, the thrombosed vein in the region of the appendix may be so small that it escapes detection at the autopsy. Thierfelder and Schüppel consider perityphlitis to be the most common cause of pylephlebitis and of embolic hepatic abscesses.

[In 64 cases of suppurative pylephlebitis collected by Langdon Brown,[1] lesions of the appendix and cecum were responsible for far more cases than any other cause,—viz., for 27,—morbid changes in parts of the gastro-intestinal tract being responsible for 18 other cases. —Ed.]

Embolic hepatic abscess may occur in cases in which the perityphlitic abscess is old and almost completely cured, or is, at all events, quite latent, and when the local manifestations of perityphlitis have completely disappeared.

I saw a case in point in a young man of thirty-five who suffered from " inflammation of the cecum " in 1890, but recovered from this in two weeks, and was well until March, 1894, when septic fever of obscure origin suddenly developed, and proved fatal in June. Phlebitis starting from a small, almost cured perityphlitic abscess with secondary abscesses of the liver and embolism of the lungs was found at the necropsy.

Other observers, for instance, Hermes, have reported similar cases. [Dieulafoy[2] has described these multiple abscesses in the liver, which may sometimes be 150 or 200 in number, under the name of the " appendicular liver." In addition to this purulent or infective hepatitis the liver may show degenerative changes or toxic hepatitis analogous to the toxic appendicular nephritis (Dieulafoy). Thus, in a girl who died the same day that she was operated upon for appendicitis, and in whom there had been no signs or symptoms pointing to the liver, there was complete fatty degeneration of the liver (Ménétrier).[4]—Ed.]

In exceptional cases where infection spreads to one of the systemic veins and phlebitis develops, pulmonary embolism, hemoptysis, and abscess of the lung may be produced.

In the preceding description two ways in which the peritoneum may become involved in appendicitis have been mentioned : one local, more or less limited to the peritoneum covering the organ and usually adhesive ; the other forming a perityphlitic tumor in the neighborhood of the appendix. There is, in addition, a third, which is most dangerous —viz., diffuse peritonitis involving the greater part or even the whole of the abdominal cavity.

The enormous importance of this form, which proves fatal in the vast majority of cases,—since surgical methods have been in vogue and have reduced the mortality of all the other forms,—necessarily requires

[1] W. Langdon Brown, *St. Bartholomew's Hosp. Reps.*, vol. xxxvii., p. 97.
[2] G. Dieulafoy, *Cliniques Médicales de L' Hotel-Dieu de Paris*, vol. ii., p. 167.
[3] G. Dieulafoy, *La Sem. Méd.*, 1903, p. 341.   [4] Ménétrier, *ibid.*, November 4, 1903.

detailed description. But as it has already been dealt with in the section on Diffuse Peritonitis (to which the reader should refer), a brief summary of the most important points which have a direct bearing on perityphlitis will only be given here.

According to the character of the exudate, four types of diffuse peritonitis due to perityphlitis can be distinguished :

1. In the first place, acute peritoneal infection in which the intestine may be distended and the serosa injected, but in which there may be a complete absence of exudate. Cases of this kind are very rare and are probably seen only when death occurs very rapidly and before there is time for the formation of any exudation.

2. Related to this form, or possibly identical with it, is that in which there is either a serous or a serosanguineous exudate, usually small in amount ; the course of the disease is so rapid that pus is not formed, but, as a result of the hyperemia, there may be rupture of blood-vessels.

3. In a third form there is diffuse peritonitis with more or less copious purulent, feculent, or sanious-purulent fluid, without much fibrin, and consequently without many adhesions between the intestines.

4. Lastly, the fourth form, which runs a slow or even a chronic course, and may undergo spontaneous resolution, is the progressive fibrinopurulent form. There are numerous adhesions between the coils of intestine, which inclose larger or smaller collections of pus, producing the well-known appearances described in the section on Peritonitis. In this form the peritoneal cavity may be so cut up by adhesions that some parts are healthy while others show the characteristic changes of progressive fibrinopurulent peritonitis. (The reader should refer to the section on this form of peritonitis for a detailed description.) Nothing further need be added here than to quote Rotter's statement, with which I agree, that the anatomic changes found after death are not alone enough to settle the nature and character of the peritonitis, and that for this purpose the additional information provided by clinical observation is required.

After this description of the morbid anatomy, a few remarks may be made about the genesis of these different forms of peritonitis ; we must study by what paths (tissue-spaces, lymph- or blood-vessels) the inflammation travels, to what extent toxins or bacteria are concerned in the process, and what the rôle of the contents of the appendix is in cases of perforation. These points have all been dealt with in the section on Peritonitis, and will not be repeated here : since that description applies equally to peritonitis from appendicitis, which is merely a special variety of the disease, only a few special points will be referred to here.

The inflammatory agent that comes in contact with the serous layer of the appendix in simple or recurrent appendicitis causes chronic inflammatory thickening of the external surface of this membrane ; the resulting adhesions form a protective barrier which is of great importance in perforation of the appendix, for in this way the purulent or sanious contents of the appendix are prevented from entering the gen-

eral peritoneal cavity. Several different results may be met with under these conditions. The whole peritonitic process may either remain limited to the immediate neighborhood of the appendix, or it may extend slowly along the retrocecal connective tissue; or the protective wall of peritoneal adhesions may become infected and the disease slowly extend to larger areas (progressive fibrinopurulent peritonitis); in these cases veins may become infected and pylephlebitis and suppurative hepatitis result; or, in rare cases, there may be serous peritonitis in addition to the encysted perityphlitic abscess; the serous inflammation of the peritoneum may either undergo spontaneous resolution or may at any time become purulent.

When perforation occurs acutely before the formation of adhesions in sufficient quantity and strength or even before there are any, diffuse septic or sanious peritonitis results.

Lastly, another important event must be considered. It was formerly generally believed that a perityphlitic abscess was always due to perforation of the appendix, and that, as a rule, this was caused by fecal concretions. There can be no doubt, however, that a perityphlitic swelling or abscess, as well as diffuse peritonitis, may occur in the absence of fecal concretions or even without any perforation of the appendix; but to allow of this, the appendicitis must be either purulent or gangrenous, for there is no evidence to show that simple catarrhal appendicitis ever produces severe perityphlitic inflammation with swelling, abscess, or acute diffuse peritonitis. The factor that determines the character and the severity of the peritoneal affection in cases in which the appendix remains unperforated is the virulence of the infectious agent and the rapidity with which it invades the peritoneum.

Other forms of inflammation of the appendix are all of subordinate practical importance in comparison with the idiopathic varieties just described; they are, moreover, very rare, and the anatomic changes as well as the clinical manifestations are so similar throughout that the differential diagnosis, which can hardly ever be made at the bedside, is possible only when the organ is examined after removal or at a necropsy. The most common form is tuberculous appendicitis; Fenwick and Dodwell's statistics show the rarity of this affection, for in an examination of 2000 bodies they found tuberculous ulceration of the appendix only in 17. Kelynack claims that the appendix is rarely, if ever, primarily affected. An anatomic peculiarity of tuberculous perityphlitis is that this chronic disease tends to give rise to fistulous sinuses.

[Bouglé[1] considers primary tuberculous appendicitis less rare than it is usually thought to be, and believes that it is often overlooked. It is usually near the cecal end of the appendix. Clinically, it tends to recur more frequently than non-tuberculous appendicitis. Crowder's[2] unique case of hyperplastic tuberculosis limited to the appendix has already been mentioned.—ED.]

The anatomy of the perityphlitic tumor and abscess is the same in

[1] J. Bouglé, *Arch. Gén. de Méd.*, 1903, vol. i., p. 263.
[2] T. R. Crowder, *Amer. Jour. Med. Sci.*, vol. cxxiv., p. 236.

cases that really originate from the cecum as in the appendicular cases, and it is therefore unnecessary to describe this form further. The only point which must be reiterated is that simple typhlitis never produces perityphlitis, and that there must always be some ulceration of the cecum—as a rule, stercoral ulcers from pressure or in some instances from tuberculous ulcers. Occasionally malignant disease of the cecum may produce perityphlitic and paratyphlitic abscesses; I have seen these complications follow growths in the wall of the cecum, the ileocecal valve, and the appendix. A case was mentioned on page 423 in which the picture of a paratyphlitic abscess developed from perforation of the lower portion of the ileum in a case of decubital ulcer and stenosis of the cecum.

Within the last few years a number of cases (so many, in fact, that it is unnecessary to quote them) have been recorded in which actinomycosis imitated acute, or more often chronic, perityphlitic abscess. Postmortem examination of these cases shows that the disease usually originates from the cecum, though in some cases it starts from the appendix. There is no anatomic difference between these cases and ordinary paratyphlitic and perityphlitic abscesses. The only way in which the true nature of the lesion can be made out is by examination of pus let out at an operation or obtained at an autopsy, and the discovery of the typical fungus of actinomycosis.

CLINICAL FEATURES.

The clinical and anatomic history of the various forms considered above has been dealt with of late years in numerous publications, and the descriptions vary and depend on whether the author relies entirely or chiefly upon his personal observation, whether he obtains his material from the extensive literature of monographs, or whether he confines himself to the more condensed text-books. The vivid dramatic nature of the one, the epical profusion of the other, are necessarily replaced in the last by almost dogmatic but comprehensive statements. There is no reason, however, why the latter should sink to mere diagrammatic reproduction, as the natural sequence of events can be accurately and clearly set forth. But it must be borne in mind that the most comprehensive work can never entirely exhaust the actual facts and events.

A good clinical description should be given in connection with the actual anatomic changes known to exist in these different conditions. Adopting this plan, three main groups of clinical pictures can be distinguished: (*a*) The first comprises the symptoms that appear in inflammations limited to the appendix and its immediate neighborhood; (*b*) the second is concerned with perityphlitic inflammation proper, and includes the tumor and abscess that are so common in this disease; (*c*) the third deals with diffuse peritonitis and its sequelæ and with the other possible terminations of perityphlitis. In reality there are many transitional forms between these different syndromes.

These three main groups of clinical pictures, however, are clearly defined and will form the basis of the descriptions in the following sections.

Clinical Picture Presented in Affections Limited to the Appendix Itself and its Serous Covering Alone.—Simple appendicitis may run its course without producing any symptoms, so that the morbid process is discovered only accidentally postmortem without any history to indicate that the appendix was diseased during life. Comparatively frequently, however, symptoms are present which enable the condition to be diagnosed not only with a certain degree of probability, but with absolute certainty. The symptoms vary somewhat in the acute and chronic forms, being transient in one and continuous and persistent in the other.

To begin with a picture of a typical acute attack : in this the more important and characteristic clinical traits appear over and over again,

FIG. 7.
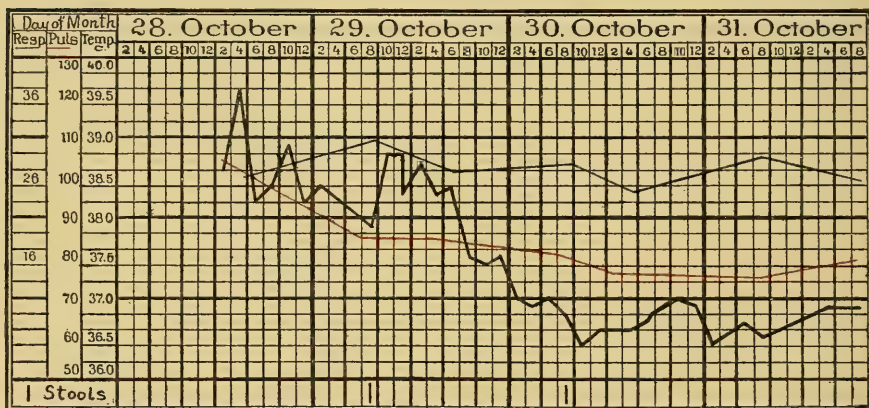
independent of anatomic and etiologic conditions. As prototype a sketch may be given of a case of so-called acute inflammation of the appendix.

The patient was a woman twenty-three years old who had had two children ; after the birth of the first child, three and three-quarter years ago, she had suffered for several weeks from inflammation of the peritoneum accompanied by fever, and since that time from frequently recurring attacks of constipation. On the evening of the 27th of October, 1896, there was a sudden onset of severe abdominal pain, more marked on the right side and radiating into the region of the stomach and into the back, accompanied by vomiting, chills, and febrile sensations. As these symptoms continued, the patient was admitted into the clinic on the 28th.

Condition on the 29th : Poorly nourished woman ; no symptoms of collapse ; complains of pain in the abdomen to the right and below, and radiating inward and backward ; the physical signs of fully compensated mitral insufficiency are found. The patient has vomited once ; the vomit contains lactic and hydrochloric acids ; the urine shows traces of indican and of nucleo-albumin, but otherwise nothing abnormal. All the other organs are normal. The temperature is raised ; the pulse is normal. The chart that is appended (Fig. 7) shows the course of the fever, pulse, and respiration for the three days of the disease. The bowels were open naturally every day.

The condition of the abdomen was specially favorable for examination, as it

was retracted, free from subcutaneous fat, and allowed the aorta, the spine, and the iliac arteries to be distinctly felt. No tumor or abnormal resistance could be felt anywhere; there was no tenderness on pressure; but in the right iliac region a round cord could be felt, starting from McBurney's point and extending for about five centimeters downward and inward, which was about as thick as a small little finger, with a smooth surface, soft walls without any hard or uneven spots, and readily movable. It was exquisitely painful even on slight pressure, but immediately to the right and the left of the cord it was possible to press deeply without producing any pain. From time to time, and even when the patient was lying quite quiet in bed, fairly active pains were felt in the right iliac region which radiated over the whole abdomen and into the sacral region. In a few days the pain and tenderness on pressure disappeared, the cord got smaller during this time, and the patient was discharged on the 16th of November.

Pain is the chief symptom. It is always present in acute attacks, and when the acute attack spontaneously subsides or when some harmless termination (inflammatory obliteration) occurs, the pain may disappear forever. The pain is rarely excruciating, but is often very considerable. It is in the right iliac fossa, and may remain localized there or may spread to distant parts—the epigastrium, the whole abdomen, the back, the right side of the chest, the groin, and the right leg. It is stated (Fowler and Foges especially) that the pain sometimes begins on the left side of the abdomen or in the epigastrium, and subsequently becomes localized in the right iliac region. Other observers confirm this, and from my personal experience I can state that occasionally pressure in the region of the appendix (both in acute and in subacute cases) produces less pain than in the epigastrium and toward the left hypochondrium.

[Lockwood [1] says that at the commencement of the attack the patient seldom locates the pain in the iliac fossa, and that, as a rule, it is felt all over the abdomen, especially about the umbilicus; in a few hours perhaps the most acute pain is felt in the region of the appendix. When the inflamed appendix is in the pelvis, the pain is usually referred to the umbilical region.—ED.]

The pain is diminished by rest and increased by movement or pressure in the ileocecal region, while the rest of the abdomen is insensitive to pressure.

The pain, which, as a rule, is continuous in acute attacks, may further show colicky exacerbations, which are probably due to true spastic colicky contractions of the muscular coat of the intestine produced by the morbid changes in the appendix.

[Cutaneous hyperesthesia in appendicitis has been carefully worked out by Sherren,[2] whose results are as follows: Cutaneous hyperesthesia varies from a complete band extending on the right side of the abdomen from the middle line below the umbilicus in front to the lumbar spines behind, down to a small circular spot a little above the middle line, between the umbilicus and the anterior superior spine, corresponding to the eleventh dorsal area of Head. According to Mackenzie,[3] the

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[1] C. B. Lockwood, *Appendicitis, Its Pathology and Surgery*, p. 193.
[2] J. Sherren, *Lancet*, 1903, vol. ii., p. 816.
[3] J. Mackenzie, *Brit. Med. Jour.*, 1903, vol. ii., p. 66.

area is supplied by the eleventh and twelfth dorsal and the first and second lumbar nerves. Small filaments of the last two dorsal nerves piercing the rectus muscle are responsible for "McBurney's point." Very often the tenderness extends somewhat into the tenth dorsal area, and occasionally, but not often, into the twelfth dorsal area, sending a tongue-shaped process over the gluteal region. The width of this band in the adult is about three inches. This area of cutaneous hyperesthesia is probably present at some time during all first attacks, except perhaps in the fulminating type, and depends on tension within the appendix. It may be absent in subsequent attacks if the first attack was sufficiently severe to destroy the nerves in the appendix. It gradually disappears in convalescence, but its disappearance without improvement in the general condition of the patient points to perforation or gangrene of the appendix. It may, however, persist when there is general peritonitis. It is, as a rule, absent in cases of abscess of the appendix. The editor has not found this cutaneous hyperesthesia constant in first attacks, and, on the other hand, has quite recently had a case under his care where this characteristic hyperesthesia was very well marked and in which operation showed that the appendix was healthy ; the symptoms suggesting appendicitis were due to an inflamed and softening gland near the cecum.—Ed.]

A crucial point in the diagnosis is a palpable appendix. Occasionally the appendix cannot be felt, because there is extreme pain, excessive reflex rigidity of the abdominal muscles, overloading of the abdominal walls with fat, or because the appendix is hidden in some inaccessible position in the abdomen. In many cases, however, the organ can be clearly felt as a rounded, cylindric structure of varying length ; it is usually as thick as the little finger, but may be thicker or thinner— usually thicker ; as a rule, its size is uniform throughout, but its circumference may be larger in some parts than in others ; sometimes the organ is freely movable, while in other cases it is fixed. It is this structure which is tender on pressure, and it is characteristic of appendicitis that the pain on pressure is often strictly localized here, while close to the appendix the patient can stand a great deal of pressure without any pain whatsoever. When the abdominal walls are moderately thick, an indistinct resistance may be felt in the painful point, though it may be impossible to make out clearly the outlines of the appendix.

The author himself has often been unable to convince himself that McBurney's point is particularly sensitive. Lenzmann finds the most tender spot 2 cm. below McBurney's, in the interspinal line (drawn from one anterior spine to the other), about 5 or 6 cm. from the right anterior-superior spine.

Lennander gives the following interesting explanation of the tenderness on pressure at the so-called McBurney's point: he believes that this spot represents the place where, in most people, the lymphatics of the appendix pass into the posterior wall of the abdomen ; in other words, the place on the posterior abdominal wall which is first attacked by a lymphangitis and lymphadenitis originating from the appendix. The peritoneal covering of the posterior wall of the abdomen, covered as it is by numerous cerebrospinal nerves, then becomes inflamed.

[McBurney's [1] description is as follows : " In every case of appendicitis the seat of greatest pain, determined by the pressure of one finger, has been very exactly between an inch and a half and two inches from the anterior-superior spinous process of the ilium, in a straight line drawn from that process to the umbilicus. This point indicates the situation of the base of the appendix where it arises from the cecum, but does not by any means demonstrate, as one might conclude, that the chief seat of the disease is there." He considers that tenderness there in the early stages of an attack is evidence of appendicitis and occurs in no other acute disease. Treves [2] states that tenderness is commonly present at this spot in healthy subjects, and is due to pressure on the ileocecal valve. Munro's point is situated on this spino-umbilical line (running from the anterior-superior spine of the ilium to the umbilicus), at the point where the outer border of the rectus abdominis muscle is crossed. Munro's point nearly corresponds to McBurney's point, but has the advantage of being more precisely localized. There may be marked tenderness at McBurney's point in colitis, especially when right-sided and chronic. The nerve-supply of the ileocecal valve is from the eleventh or twelfth dorsal nerves or from both, and the skin between the iliac spine and Munro's point is supplied mainly by the lateral cutaneous nerve of the eleventh dorsal (Treves).—ED.]

When the organ can be felt at all, it is difficult to confound it with other organs in the vicinity, such as a contracted loop of small intestine or part of the colon. The adnexa of the uterus may occasionally be misleading ; the exact determination of the palpable cord, however, especially the fact that it reaches far up above the pelvic brim, is, as a rule, sufficient to clinch the diagnosis.

[Some operating surgeons are extremely cautious about the correct palpation of the appendix. Lockwood [3] says : " After many mistakes I am inclined to think that the appendix itself is seldom felt." Treves [4] points out that a roll of adherent omentum, enlarged glands, or a mass of feces in the cecum may simulate an enlarged appendix or one supposed to contain a concretion. A condition which is often regarded as the appendix is muscular contraction ; this " phantom appendix " (Treves [5]) is an elongated, pipe-like body, either vertically or obliquely placed, the obliquely placed one being external to the vertical. It is due to contraction of a few muscular fibers either in the outer edge of the rectus abdominis muscle or in the internal oblique or transversalis muscle. Thus, since the abdominal muscles are supplied by several nerve-roots, the persistence of this band of contracted muscular fibers would seem to show that this visceromuscular reflex was due to stimulation passing out of the spinal cord by only one nerve-root (MacKenzie [6]). —ED.]

[1] McBurney, *New York Med. Jour.*, December 21, 1889, p. 678.
[2] F. Treves, *Brit. Med. Jour.*, 1902, vol. i., p. 1589.
[3] C. B. Lockwood, *Appendicitis, Its Pathology and Treatment*, p. 199.
[4] F. Treves, *Allbutt's System of Medicine*, vol. iii., p. 920.
[5] F. Treves, *Brit. Med. Jour.*, 1902, vol. i., p. 1589.
[6] J. MacKenzie, *ibid.*, 1903, vol. ii., p. 66.

A vaginal examination should also be made, but may not throw any light on the case, especially when there is some salpingitis; another available plan is to inflate the colon with air, which may bring the appendix nearer to the anterior abdominal wall and so make it more accessible to palpation; this is an uncertain method, and would hardly be available in acute cases.

The pain in the right iliac region and the palpation of the swollen appendix are the main symptoms on which the diagnosis depends, and when they are present, certain other signs acquire significance.

As a rule, particularly in the severe cases, there is reflex vomiting, which is occasionally very severe. The bowels are, as a rule, constipated, but they may act regularly or there may be diarrhea; sometimes in cases, which there is no reason to believe are purulent, there may be slight fever, which may be ushered in with an initial chill or with a general feeling of chilliness, and, as rule, is not above 100° to 102° F.; the fever is, moreover, of short duration, rarely lasting more than a few days. Sometimes, however, the rise of temperature may be considerable, even in mild cases—*e. g.*, in the case on p. 874 it went up to 103.2° F.; on the other hand, there may be no fever at all. The general health of the patient is rarely affected, as in a case reported by Lennander,[1] in which a patient with simple catarrhal appendicitis suddenly developed symptoms of peritonitis and collapse; the appendix in this case was afterward removed and examined microscopically. There are no other remarkable or at all constant signs in the abdomen.

When these symptoms appear for the first time in a subject, all that can be said is that there is some morbid change limited to the appendix, but it is impossible to determine what form of the disease is present— *i. e.*, whether there is appendicitis with purulent or mucous contents, or whether there is a fecal concretion. To judge from analogy, it may be said that in recurrent cases any one of these alternatives is possible, and it is of the greatest importance to note that even the most severe forms of appendicitis—the diphtheric, phlegmonous, ulcerative, and gangrenous—may present the features of this simple clinical picture only. This will be referred to again later.

An acute and even a febrile attack may completely subside; the fever drops, the pain disappears—first the spontaneous pain and later the tenderness. Vomiting and constipation pass off, and the patient recovers. It is probably true that recovery is possible, although this cannot be absolutely proved when the appendix contained pus, provided this pus can escape from the appendix into the colon, either as the result of energetic contractions of the appendicular wall or as the result of diminution in the swelling of the mucous membrane of the appendix.

Unfortunately, this favorable termination is not the rule; the appendix may get rid of its contents, but the primary conditions which favored the occurrence of the first attack persist, such as abnormal position of the organ, kinking, fecal concretions, etc.; or these factors may be-

[1] *Loc. cit.*, No. 54.

come exaggerated by the first attack, and fresh conditions may arise which also favor the development of appendicitis, such as chronic swelling of the mucous membrane of the appendix, and ulceration, followed by stricture of the lumen of the appendix, inflammation of the mesentery of the appendix, and consequent stretching of the appendix. Again, the same conditions may exist in "angina of the vermiform appendix" (see above) as in angina tonsillaris—that is, one attack may dispose to subsequent attacks.

It is, therefore, clear that a patient may recover completely from an acute attack of appendicitis and may remain perfectly well afterward, but it must also be recognized that in many cases the disease takes an insidious chronic course, with more or less frequent intercurrent acute attacks. Cases of this kind have an enormous practical importance.

The most common occurrence is that a patient who has once suffered an attack of appendicitis has repeated attacks of exactly the same character—viz., pain, constipation with or without vomiting, and fever. The pain in such cases may appear suddenly while the patient is apparently quite well, and without any known cause; some authors claim that occasionally trauma, some error in diet, constipation, or diarrhea may be the determining factor.

[Lockwood[1] speaks of a railway journey as a common exciting cause of appendicitis.—Ed.]

The attack lasts for a few days, occasionally for two or three weeks, and then the symptoms all disappear and the patient is practically well; in some cases, however, there are disagreeable sensations or even pain in the ileocecal region, and I have been able to determine that in the interval between attacks, when the patients were apparently quite well, the vermiform appendix throughout its whole extent or at McBurney's point was distinctly tender on pressure.

Recurrences may occur after a few weeks or months, so that the patients suffer three or four attacks a year; this may continue for a number of years. The severe impairment of health, loss of appetite, pallor, loss of weight, highly nervous symptoms of different kinds, which appear, even in these mild but recurrent forms, can readily be explained from the frequency of the attacks; it is also obvious why these patients are never in possession of their full working powers. In addition, their is, of course, always the danger of more serious consequences.

Such clinical pictures, giving rise, only a few years ago, frequently to the most curious and unfortunate errors in diagnosis, are explicable as acute attacks of an unhealed, subacutely inflamed appendix. The anatomic anomalies that I have described are in these cases seen in the appendix—most commonly knuckling or stenosis. The contents of the organ, if removed by operation, are found to be either simply mucous or mucopurulent-hemorrhagic. Fecal concretions may be present or absent. At the beginning of an acute attack—and this is particularly noteworthy—it is impossible to decide, from the slightness or the severity

[1] C. B. Lockwood, *Appendicitis, Its Pathology and Treatment,* p. 141.

of the attack, whether we have to deal with a purulent or a non-purulent appendicitis.

Operative treatment has provided so many opportunities of examining appendices diseased as described above that the correctness of these views cannot be doubted ; these clinical manifestations are without doubt due to the anatomic changes.

In other cases the patients do not recover at all after the first or after repeated attacks, and there is not even any apparent improvement. The disease at once runs a subacute course, with persistence of all the symptoms. In such cases there are, as a rule, more advanced·changes in the appendix—either great thickening of its walls with inflammation of the peritoneum, and inside the organ ulceration, fecal concretions, stenosis from kinking, or swelling of the mucous membrane, the lumen usually containing pus. These cases often develop into empyema of the appendix.

Sonnenburg has described a mechanism which may lead to the continuous distress in appendicitis. The appendicitis may lead to chronic adhesive peritonitis, and, by inclosing the cecum and other neighboring parts of the bowel in a ring of adhesions, may narrow the affected portion of the bowel as soon as there is the slightest inflammation or swelling ; in this way stagnation of the contents of the bowel is produced, and a tumor can be felt which disappears after a copious evacuation of the bowels. A number of clinical observations made during operation bear out this sequence of events, so that this is apparently a definite condition simulating perityphlitis with recurrences.

Talamon has introduced the term *appendicular colic* (colica processus vermiformis—Breuer) to describe the following clinical manifestations : The sudden onset of pain in the appendix or around the umbilicus, radiating into the epigastrium or even to the left side of the abdomen, and often accompanied with vomiting and collapse ; at the expiration of from twelve to thirty-six hours all symptoms promptly disappear after an injection of morphin, and even tenderness over McBurney's point may entirely pass away. Sometimes, however, perityphlitis with fever develops in these cases. There can be no doubt but that attacks of this kind do occur, and Talamon is unquestionably correct in his view that they are produced by energetic muscular contractions and consequently represent a true colic of the appendix. I cannot, however, accept his view that these attacks are produced by the sudden passage of fecal concretions from the appendix into the cecum, for it has never been proved that this occurs. I agree with Breuer (quoted from Hochstetter) that material in the appendix (accumulations of mucus or larger masses of fecal matter) precipitates these attacks when the cecal orifice of the appendix becomes stenosed or occluded ; this, moreover, seems to occur only in appendices that are already abnormal in some way (kinking, catarrh, swelling of the mucous membrane, etc.), and never in a perfectly healthy appendix. The observations of Riedel, to be given presently, teach us that intra-appendicular hemorrhages may simulate simple appendicular colic.

Of the greatest clinical importance are those cases in which the appendix is the seat of severe phlegmonous, ulcerative, or gangrenous changes, for in these cases the most dangerous complications appear, especially in relation to the peritoneum. They run an extremely rapid course, and lead, with or without perforation, to diffuse septic peritonitis; a criterion of the severity is the excessive virulence of the bacteria. It is important to note here that in such cases, so long as the disease is limited to the appendix, the clinical aspect does not necessarily differ in any way from a slight or even the slightest forms of purulent or non-purulent appendicitis; no single symptom may point to immediate danger. A gangrenous or severe phlegmonous case may present an apparently quite harmless aspect, with a moderate amount of pain, no great rise of temperature, some vomiting, an uncertain result from palpation in the right iliac fossa, and no grave constitutional symptoms, and yet an operation thirty-six, twenty-four, or even twelve hours after the first appearance of clinical symptoms may show that the appendix is totally destroyed by inflammation. Every practitioner of experience has made such observations, and Beck is right when he says: "In hardly half the cases is the clinical aspect in the early stages so clearly defined that a definite conclusion as to the pathologic changes in the appendix can be made."

The above sketch of the clinical aspects has been broadened by the numerous operations of late years, resulting in careful and methodic anatomic research. The literature is enormous, but attention may be drawn to the communication of Riedel, based upon roughly 300 cases. He supplemented his clinical and surgical observations by microscopic examinations, undertaken by himself, of appendices which he had removed. He has tried more particularly to ascertain the determining conditions and ultimate cause of a sudden attack of appendicitis. From his instructive studies the following paragraphs are particularly worthy of note:

"A thoroughly healthy appendix, free from foreign bodies, is practically never attacked alone by the disease which we term appendicitis." Riedel himself attributes perforation to pointed foreign bodies. Perhaps the acute cases having a hematogenous origin are an exception to this rule.

"Appendicitis is almost always a preëxisting disease of old standing which develops insidiously and almost without symptoms. It appears precisely as does cholecystitis, through the lighting up of an acute inflammatory process in an appendix primarily the seat of chronic disease.

"The appendix is predisposed to the acute attack by two completely different causes: (a) Either a fecal concretion forms in a perfectly healthy appendix which causes disease of a more or less limited area, or (b) the appendix is primarily the seat of disease in a characteristic manner, fecal concretions being absent. This typical disease of the appendix is perhaps best named appendicitis granulosa.

"An acute inflammatory attack of non-purulent appendicitis is

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exceptionally caused by a fecal concretion in an almost normal appendix ; generally it is the result of appendicitis granulosa, stricture, or stenosis. Vice versâ, the acute suppurative or gangrenous attack follows more often on a fecal concretion or round foreign body than on soil predisposed through appendicitis granulosa or stricture or stenosis.

"Probably the acute inflammatory attack is often preceded by a hemorrhage from the granulation tissue. The injury to the epithelium is followed by the entrance of infective agents into the lymph-channels of the chronically diseased appendix." Riedel considers hemorrhage as the primary factor, infection as secondary, and brings forward interesting observations bearing on this conception. In one case attacks accompanied by violent pain and rise of temperature lasted for sixty-two hours ; then clots of blood were passed per anum. Riedel thinks that in this case the appendix contracted violently upon the blood-clots in its interior and so gave rise to the clinical features of the attack. The hemorrhage itself occurs very easily from the granulations with their delicate vessels in the diseased wall of the appendix.

One thing only must be added to Riedel's observations : How does the insidious form of "appendicitis granulosa" arise? To explain this the reader must refer to the section on the Etiology and Pathogenesis.

Clinical Picture of Perityphlitic Inflammation with Formation of Abscess and Tumor.—The characteristic features of the clinical picture can be stated in a few words : pain in the right iliac fossa, a tumor, or at least resistance, in the same region ; fever, as a rule ; usually vomiting, and frequently constipation ; in addition, a few other inconstant features, such as meteorism, painful micturition, changes in the composition of the urine, and accentuation of the second pulmonary sound of the heart.

Deviations from this picture, which may, it is true, often be so considerable that the real disease can be recognized only with difficulty, are largely due to the characteristics of the tumor—*i. e.*, in its position, size, and, most important of all, the virulence of the inflammatory agent that produces the inflammation of the appendix. Before describing these variations in the course and the general characters of the clinical picture, the individual symptoms enumerated above, which are commonly seen in perityphlitis when localized in the usual position of the appendix, will be described seriatim.

The *pain* is the most regular symptom : in cases that run a protracted course the pain, it is true, may be very slight, but it is always present at the beginning and I have never seen or heard of a case in which it was completely absent. Sometimes the pain appears while the patient is in perfect health and without any known cause ; it may even appear suddenly during sleep. In other cases certain injurious factors are apparently responsible, such as an attack of indigestion, a hearty meal, violent exertion. Many of the patients state that they suffered for some time from pain in the iliac region, or that they have had one or more previous attacks of perityphlitis. Occasionally, too,

the pain gradually increases in severity and only slowly reaches its maximum intensity.

The seat of the pain is in the right iliac region, and in the overwhelming majority of cases is there from the very beginning. In order to avoid mistakes, however, it is well to remember that the patients occasionally localize it in other regions of the abdomen. In some instances it is first felt in the region of the stomach; in others, in the right hypochondriac region, around the navel, or on the left side of the abdomen; in some cases the pain is first felt over the whole abdomen, and subsequently becomes confined to the right lower half of the abdomen. From this region it may then radiate into any one of the regions mentioned or into the lumbar region, the testicle, the sacral region, the bladder, and especially down the right leg.

Special attention should be directed to the fact that the pain may often be felt diffusely over the whole abdomen, and later be limited in the region of the appendix; this sign probably favors the view, which will be discussed below, that the infectious material is sometimes, in the first instance, disseminated over the whole peritoneal cavity, from which it is subsequently absorbed, as it is so dilute, except from the neighborhood of the appendix, where it is more concentrated and consequently produces a violent reaction. Moskowicz is inclined to the opinion, based upon the experience of early operations, that, as a rule, the perityphlitic inflammation is at its commencement not circumscribed, and that the infection of the appendix in a short time, either with or without perforation, gives rise to an exudation in its immediate neighborhood, at first not shut off, but affecting in many cases later a greater part of the peritoneal cavity.

The intensity of the pain varies, and is probably dependent on the individual reaction of the patient; in some cases it is excruciatingly severe; in looking through the notes of all my cases, I cannot find any constant relation between the intensity of the pain, the height of the fever, and the size of the tumor; the pain, for instance, may be very severe when there is a small lump and very little fever; movements of all kinds, in particular walking, seem to increase the pain; lifting or stretching the leg in bed is often impossible; micturition, too, may often be difficult. In some cases the patients remain perfectly quiet on their backs. In all cases pressure over the right iliac fossa is painful—sometimes so unbearable that the slightest touch is feared and avoided, and there is great hyperalgesia of the skin in many cases. The area of tenderness on pressure, as a rule, corresponds with the extent of the palpable resistance or the tumor.

Usually the pain is continuous at the onset, even when the patient keeps perfectly quiet, and there are frequently, in addition, exacerbations of the pain not only on movement, coughing, micturition, and deep inspiration, but also spontaneously. Many patients describe this pain as tearing, while others speak of colicky, cutting exacerbations.

The pain is due to involvement of the peritoneum and is peritonitic. Occasionally the position of the pain and the character of the concomi-

tant symptoms make it possible to localize the exact position in the abdomen of the perityphlitic process; thus, when the patients keep the hip in a flexed position, there is definite evidence that the inflammation involves the psoas muscle, while if the pain is confined to or is most marked in the right lumbar region, there are definite grounds for diagnosing retrocecal inflammation.

When the process runs a favorable course, the spontaneous pain and tenderness on pressure may disappear after a few days or may be greatly diminished by the administration of opiates. In other cases the pain persists in spite of the use of opiates, cold, and local depletion. Under these conditions spontaneous resolution can hardly be expected, and there is, on the contrary, every reason to fear the formation of an abscess or even more serious consequences.

When pain is present, the correct diagnosis is strengthened by the second important sign—namely, palpation of a definite resistance or of a *tumor*. Sometimes the swelling cannot be felt at the outset; this may be due to a variety of causes : it may not be palpable at all in the first few hours of the disease because it is not yet sufficiently developed; or, again, the abdomen may be so tender that a thorough examination is impossible; or, again, the abdominal muscles contract and become so rigid that when the abdomen is palpated nothing whatever can be felt. Inexperienced practitioners occasionally mistake the muscles that are in a state of tetanic contraction for the tumor; this error can, however, easily be avoided by carefully palpating the contour of the rigid muscles; in this way it can readily be shown that the resistance felt is not a circumscribed swelling.

Inspection, palpation, and percussion must be employed in the examination.

On inspection a projection in the right iliac region is sometimes seen, especially when the two sides are compared. According to its size and position, the swelling is more or less distinct, and is round, flat, or diffuse; sometimes it fills the iliac fossa more or less completely or it is situated above or below it; these points, however, are all made out much better by palpation than by inspection, and often when the swelling is very small nothing abnormal may be seen.

On palpation the swelling appears either as a diffuse tumor or as a circumscribed and hard resistance, and occupies the right iliac region. Its size varies within considerable limits : it may be no larger than a small egg, or it may occupy the whole space between the crest of the ilium, Poupart's ligament, the median line, and the line running from the anterior-superior spine of the ilium to the umbilicus; it is only in exceptional cases that the tumor extends beyond the median line to the left; in fact, tumors of small and medium size are by far the most common. The form of the tumor may be round, oval, or elongated, the swelling usually extending in a direction parallel to Poupart's ligament; they are usually about two finger-breadths below the iliac crest or below a line that would run through the spine of the ilium. Larger perityphlitic tumors may extend upward to the bone. Tumors that are large or of

medium size can often be felt per vaginam; only very large tumors can be felt per rectum.

The tumor may have sharp distinct margins, or may gradually merge into the surrounding tissues. The posterior surface of the tumor can hardly ever be palpated, and there is only one position in which it is sometimes possible to grasp the tumor from behind.

As a rule, the tumor cannot be displaced by external manipulation; this is a point of some diagnostic value, and may sometimes enable a differential diagnosis to be made from a cecal neoplasm and a fecal tumor. It is true that in exceptional cases a small perityphlitic tumor may to some extent be displaced, but in my experience these swellings are never so easily displaced as carcinomata of the cecum.

The surface is almost always smooth and even. Sometimes slight nodosities may be felt, but there is never the nodular character of malignant growths. The tumor is hard, firm, and feels like a solid mass—this is a striking and important peculiarity. The tumor never feels soft, and never appears to fluctuate unless there is a large quantity of pus. (For the more or less severe tenderness on pressure that is seen in all acute cases the reader should refer to previous paragraphs.)

The tumor cannot always be felt as distinctly or as clearly as has just been stated. Sometimes there is only a diffuse resistance in the right lower half of the abdomen or exactly in the right iliac fossa, which cannot be accurately mapped out, and cannot be said to fluctuate, though it may slightly suggest it. Sometimes, too, the resistance is as hard as a board, without, however, possessing the configuration of a tumor.

The anatomic changes that lead to the formation of the perityphlitic tumor have already been described. When nothing more than a resistance is palpable, one of two alternatives may be present—either a serofibrinous exudate or inflammatory edema of the abdominal wall. In the latter case there may be, in addition, an inflammatory tumor further down; this is particularly the case when the inflammatory tumor lies behind the cecum.

Percussion, in my opinion, is of subordinate importance, and as it may lead to numerous errors in diagnosis, should never be relied upon alone; it need hardly be mentioned that the percussion-note is dull over a large perityphlitic tumor. Fecal tumors, however, may also produce the same phenomenon with a slight admixture of tympanitic note; on the other hand, too, tumors of moderate size may give a note that is slightly tympanitic and dull at the same time; and large tumors may do the same when the intestine in front of them contains much gas.

The above are the chief abnormalities observed. The abdomen is not tender on pressure, except in the right iliac fossa, and it is soft and compressible everywhere else. There may be some meteorism, but this sign is so inconstant and usually so slight that it is of little diagnostic importance. This statement is based on a large number of my own cases which were carefully studied; most of these showed no sign of

meteorism, a few showed moderate or slight signs, and in a very few there was marked meteorism.

In a small proportion of the cases the patients complain of bladder symptoms—pain in the region of this organ, either spontaneous or on urination, difficulty in micturition, or retention.

Vomiting is common; in my cases it was present in three-quarters of the patients, and this agrees with general experience. It is one of the first symptoms appearing with the pain, and is, of course, reflex in character. Occasionally there is only initial vomiting; in others the symptom persists for a number of days. Vomiting coming on later points to a marked exacerbation of the attack (E. Rose). The vomit consists of the contents of the stomach, of mucus, and of bile. In severe cases there may occasionally be feculent vomiting, and in some cases there is hiccup.

[Black vomiting ("vomito negro appendiculaire") is due to toxins absorbed from the appendix inducing hemorrhagic necrosis of the mucous membrane of the stomach and hematemesis. It is one of the manifestations of toxemia due to appendicitis, and may be classed with an icteric tint of the skin, urobilinuria, albuminuria, diminution or suppression of urine, and even uremic symptoms. The prognosis of these cases is very grave (Dieulafoy [1]). Box and Wallace [2] have recorded a fatal case of appendicitis in which there was profuse intestinal hemorrhage imitating typhoid fever.—Ed.]

Since some writers still adhere to the idea of stercoral typhlitis, it calls for discussion. In the paragraphs on etiology the theory that constipation causes the disease was controverted. In the majority of my hospital and private cases the bowels were habitually regular, but after the onset of the disease constipation is the rule, even without opium, and is a symptom of the disease and not one of the causes. It is due to the peritonitic paresis of the intestinal musculature or occasionally to reflex paralysis.

Constipation is by no means a constant symptom, and in many cases there is a natural daily evacuation. There may even be diarrhea, caused presumably by some intestinal complication, or there may be a regular action of the bowels for the first few days, and later constipation. Sometimes, indeed, the patient claims that there is not even flatus.

When the course of the disease is febrile,—and this is the rule,—there are loss of appetite, a coated tongue, and eructations.

The *fever* is important from the point of view of prognosis and of treatment, and many investigators have recently paid considerable attention to this symptom. My experience corresponds with that of Rotter and Lennander, that there is nothing typical about the fever and that many variations occur.

In the first place, some cases run an afebrile course in which the temperature never rises above 100.4° F. This applies to cases with

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[1] Dieulafoy, *La Presse Médicale*, February 13, 1901; *Clinique Médicale de l' Hotel Dieu*, 1901–02, vol. iv., p. 194.   [2] Box and Wallace, *Lancet*, 1903, vol. i., p. 1588.

both small and large exudates. In larger exudates accompanied by fever, however, it is always well to be on watch for an unfavorable turn in the disease, unless an operation is performed early. For example, I saw a case in a man of forty-nine who stated that he had had three attacks within the last five years which were identical with the present and fourth attack, and were accompanied by a large ileocecal tumor, pain, etc., but not by fever. He entered the clinic two weeks after the onset of the attack, and remained there for two weeks more. This attack also began without fever, and there was no nausea or vomiting; the bowels acted naturally three times a day at first, and subsequently became constipated. There was a large, hard, and painful tumor in the right iliac region, which did not fluctuate and extended from Poupart's ligament above the iliac spine, and as far as the median line to the left. The highest temperature recorded was 100.2° F.; on the fourteenth day it rose to 102° F., and an operation was performed at once and a large abscess containing large quantities of offensive pus was evacuated; recovery in about four weeks.

Complete absence of fever is, however, the exception. According to Rotter, this occurs in about 10 per cent., which is about the same proportion as in my cases. As a rule, there is fever, with many variations in the height, duration, and curve.

In some cases there is slight fever or a considerable rise—over 102° F.—for two or three days only, or the patients state that they felt feverish only when the pain appeared, and that this sensation disappeared within twenty-four hours.

In other cases the fever persists for from four to seven days, rising as high as 102° F. and less often to 104° F. or higher. There is, as a rule, continual fever, with slight or marked morning remissions. The drop is usually by lysis, and only exceptionally by crisis. I have seen critical defervescence from 103° to 97° F. on the eighth day within sixteen hours with disappearance of all subjective and objective phenomena. This is probably usually due to discharge of an abscess, although in this particular case this could not be proved.

In other cases there is a rise of temperature for a week. Rotter distinguishes two groups among these cases, lasting more than five days. In the first group the fever is not above 102° F. after the fifth day, and a spontaneous cure may take place although the fever lasts for a period of about five to seven days, but the prognosis becomes worse each day that the fever persists; in the second group, in which the fever after the fifth day is above 102° F., the prognosis is grave, although here, too, spontaneous resolution may occur in exceptional cases. The persistence of high fever, therefore, after the first week is a bad sign. My own observations agree with these statements.

In another group of cases there is a slight degree of fever for a few days, followed on the third, fourth, or fifth day by a normal temperature for several days, and then, without any known cause, a second rise of temperature, which may be sudden or gradual.

This is the common course of the fever in uncomplicated perityphlitis;

when complications occur and there is no longer an encapsulated abscess, matters become different; in progressive suppuration, paratyphlitic abscesses, in pylephlebitis, in progressive purulent peritonitis, in acute perforative peritonitis, or in septic peritonitis, there may be either a high continuous fever or irregular fever with chills, or the temperature of collapse with excessively rapid pulse; all this will be discussed below.

The onset of the fever may vary: sometimes it is gradual, with only a slight rise, or there may be a chill or several chills, with a rapid rise. It is impossible to forecast the future course of the disease from these data, for the case may run a mild course after severe initial fever and vice versâ. The subsequent course of the fever later in the course of the disease is of great importance, and it is, therefore, essential that the temperature should be carefully watched by all medical men who do not at once proceed to operate as soon as the diagnosis of perityphlitis or appendicitis is made. The temperature is of more importance as regards the prognosis and treatment than the tumor. Occasionally it is necessarily the only guide—namely, when no tumor can be distinctly felt or when the development of the swelling cannot be clearly followed.

[A leukocytosis which progressively increases from hour to hour is of value in these cases as showing that suppuration is present and that operative interference is indicated. A leukocytosis of 20,000 or over indicates pus or gangrene (DaCosta[1]). Absence of leukocytosis may, however, occur in the most severe fulminating forms of appendicitis. —Ed.]

The fever and the presence of a tumor, the rapidity with which the swelling forms or disappears, and the formation of an abscess all depend on the virulence of the invading bacteria, and, of course, also on the power of reaction and resistance of the individual. The local changes in the tumor and the fever, as a matter of fact, usually run parallel, consequently one is justified in drawing conclusions about the former from the latter. When the fever is of short duration, there is, as a rule, little or no suppuration and a rapid cure follows. The more marked the fever, the more severe, as a rule, the suppuration, and the more serious the consequences.

The *pulse* offers nothing characteristic; its characters depend on the general conditions in each case. I have never seen any typical pulse changes in perityphlitis so long as the process remained localized. [Many writers lay great stress on a rapid pulse as evidence of suppuration, perforation, or gangrene; thus a sudden quickening of the pulse to 110 or 120, which is sustained, points to one of these events (Lockwood).—Ed.]

The same applies to the *respiration*. In acute peritonitis the respiration becomes rapid, but this is common to all forms of acute inflammation of the peritoneum. Dyspnea, of course, also appears when there is much meteorism.

An observation of Mannaberg's may be mentioned here, which was made in my clinic and which I have had occasion to verify, namely,

[1] DaCosta, *Amer. Jour. Med. Sci.*, November, 1901.

that in simple peritonitis the second pulmonary sound is often accentuated. At present it is impossible to explain this phenomenon.

[Accentuation of the second pulmonary sound in biliary colic has been explained on the hypothesis that there is reflex constriction of the pulmonary capillaries.—ED.]

There is nothing characteristic about the patient's general condition so long as the process remains local. Sometimes symptoms of collapse appear in cases that begin very violently or in cases that lead to abscess formation with pyemia or diffuse peritonitis.

In the majority of cases, however, the local symptoms dominate the picture and the general health is only slightly involved.

The *urinary symptoms* are important chiefly from a negative point of view. The urine has been constantly tested in all cases in my clinic and nothing characteristic of perityphlitis has ever been found. Serum- and nucleo-albumin are generally absent, but may appear when there is high fever. The same applies to acetone; diacetic acid has been found in some cases. Besides high fever, the state of the bowels influences the appearance of acetone. The presence of indican (Jaffé's and Obermayer's reaction) was specially investigated, but no constancy in its excretion could be made out; large or small quantities or mere traces might be present. The character and the severity of the local inflammation do not influence indicanuria, which depends on conditions inside the alimentary canal and on the incidence of general peritonitis.

[Brewer,[1] described a case of fatal acetonemia following operation for acute perforative appendicitis in which the urine contained albumin, casts, and much acetone and diacetic acid. Sepsis was considered to be entirely excluded by the author, though this view was not taken by Deaver in the discussion on the case.—ED.]

These are the symptoms of perityphlitis when it goes no further than tumor formation in the right iliac region and runs a course of medium severity. Changes in the position of the tumor do not have much influence on the clinical picture, but are important in performing the examination of the patient. (The reader should refer to the anatomic paragraphs above, and it need only be pointed out that examinations made after death and at operations show that there are many more possibilities than can be made out before the operation. At the same time, some points can be made out by physical examination and others by symptoms which definitely show that the tumor is in an unusual position.)

In some cases there are symptoms connected with the right thigh and leg, such as violent pain, both in the sciatic and in the crural nerves; any kind of movement is painful, and the limb is kept flexed and often adducted. These symptoms point to inflammation extending backward and flexion of the thigh; in particular, irritation of the psoas.

When the abscess develops in the retrocecal region, there is pain in the back and the lumbar region, which become tender on pressure.

[1] G. A. Brewer, *Annals of Surgery*, October, 1902, p. 481.

The right flank protrudes, and these changes may sometimes be seen to extend to the renal region if the swelling spreads in this direction. These cases then appear as lumbar, perinephritic, or subphrenic abscesses.

When the process extends upward in front of the cecum and colon, the whole side of the abdomen may become tender on pressure, and pain and swelling may extend even above the umbilicus.

The perityphlitic inflammation rarely passes inward beyond the middle line to the left; in many cases, however, the tumor is concealed by coils of intestine in front of it.

The extension of the perityphlitic process toward the pelvis is important, and the diagnosis may become still more difficult when it starts in the tip of an appendix which hangs down into the pelvis from its abnormal length. In such cases the tumor is found on the right side of Douglas' pouch, and in males between the bladder and the rectum, sometimes in front of the os sacrum; in women, usually between the rectum and the internal genitals. When the tumor primarily starts in the pelvis, there may be no tumor palpable through the anterior abdominal wall. Sciatic and crural pain, violent pain in the small of the back, dysuria, tenesmus, and occasionally mucous catarrh of the rectum may all be due to this condition. On vaginal or rectal examination a painful resistance or a tumor is palpable on the right side. In women it is often impossible to distinguish between lesions of the uterine appendages on the right side and perityphlitis and appendicitis. Comparatively often the two occur together, since the annexa become infected from the appendicitis.

In very rare cases the inflammation extends to the left across the sacrum, and cases have been recorded in which it went even further and crept up along the descending colon. Symptoms analogous to those on the right side then appear on the left.

Stress need hardly be laid on the fact that the swelling can be found in the most unusual positions when the appendix is abnormally placed and its distal extremity becomes involved in a perityphlitic process; this abnormal position may be due to abnormalities in the course of the colon, unusual length of the appendix, etc. In these cases the tumor is in peculiar locations from the very onset of the disease—as, for instance, at the liver margin, behind the liver, or at the umbilicus. In such cases a number of various conditions must be considered in arriving at a diagnosis, but the possibility of perityphlitis in an abnormal position should never be forgotten under these circumstances. The clinical picture of subphrenic abscess will be described later.

Lastly, a perityphlitic tumor may develop in a hernial sac, and may, of course, simulate inflammation or strangulation of the hernia. The swelling may appear in the left, as well as in the right, inguinal region. Lastly, in rare instances, the appendix is in the right femoral canal, and if inflamed, may simulate inflammation or strangulation of a femoral hernia.

Among the clinical manifestations of local inflammation of the appen-

dix quite independent of diffuse peritonitis, attention must be drawn to an important, although not very common, group of symptoms which simulates occlusion or stenosis of the bowel—viz., vomiting, complete constipation, even without flatus, meteorism, collapse, feculent vomiting, and increased peristalsis. This syndrome may appear at different stages of the disease, and may be due to a variety of causes. We are now, however, not concerned with this group of symptoms when caused by diffuse secondary peritonitis, but only when due to local perityphlitis.

The acutely inflamed swelling may compress a loop of the bowel and in this way produce occlusion. I have seen this repeatedly, and other observers have published similar cases. Leyden has reported such a case that was not well developed. I have seen symptoms of stenosis with greatly exaggerated local peristalsis in a case where the tumor was quite small; there was acute perityphlitis, and it is probable that adhesions played a part in producing the occlusion (no operation was performed in this case). The compression may involve the small or the large intestine, or even the rectum. In other cases the symptoms of intestinal obstruction may appear several weeks after the onset of the disease, when the acute symptoms have all disappeared and the absorption of the exudate is already taking place, and be due to knuckling from peritoneal adhesions.

Lastly, in some, and they are clinically the most important, because they are difficult to diagnose, the signs of obstruction appear in the earliest stages as a result of reflex paresis of the bowel. Lennander says that the picture of "ileus" may be caused by severe appendicitis without peritonitis, or that it may be due to a slight local peritonitis. Other observers—Ranschoff, Hartley, Peyrot, Angerer—express similar views. A correct differential diagnosis can be made only after a very careful examination of the case, and not always then.

Some remarkable cases described by Ewald also belong to this category, in which symptoms of occlusion appear at once, and lavage of the stomach brings up feculent material, with death on the third day with severe cerebral symptoms, somnolence, coma, and restlessness. The onset of the disease was the same as in perityphlitis, and at the autopsy "there were very slight inflammation of the cecum and phlegmonous inflammation in the posterior part of the psoas muscle, and nothing else." (For an interpretation of these cases the reader should refer to the section on the so-called Nervous Symptoms of Acute Intestinal Obstruction.)

So far the symptoms of perityphlitis have been described without any reference to the underlying anatomic changes and their etiology, but the following two questions of great practical importance must now be answered—viz.: What symptoms show whether the perityphlitis is due to perforation of the appendix? When and under what circumstances can it be assumed that the perityphlitic tumor is an abscess?

Sonnenburg, in answering both these questions, says: When the disease begins suddenly with fever, an initial chill, and severe symptoms, acute pain, resistance, and tenderness in the right iliac fossa, usually

without a trace of fluctuation, vomiting, diarrhea or constipation, and serious constitutional disturbance, there is perforative appendicitis with the formation of pus around a perforated appendix.

Sahli, leaving out the question of perforation, says that in the cases described by Sonnenburg pus is always present, and that this statement is well borne out by the results of operations. But the discovery of pus at operation is not, he says, of much importance as regards the diagnosis of perityphlitis, for perityphlitis with the formation of a tumor and these symptoms is always phlegmonous and purulent, or, at any rate, is always accompanied by a " pus nucleus " somewhere—in other words, the symptoms described are not specially characteristic of perityphlitis, but occur in any form of severe acute peritonitis. I shall attempt to answer the two questions separately :

Perforation in general has just been referred to, but without any reference to fecal concretions, and from what was stated in the section on the Anatomy, this is justified, for perforation may occur in their absence. It is impossible to determine whether they are present or absent, and we must content ourselves with making out whether there is perforation. But I must admit that I consider even this impossible, for analysis of my cases shows that the syndrome which Sonnenburg regards as characteristic of perforation may be present when the appendix is not perforated. The essential point is the rapid spread of a very virulent infection in the neighborhood of the appendix, and it is immaterial, so far as the clinical picture is concerned, whether this infection occurs through a hole in the appendix or whether, in suppurative and gangrenous appendicitis without destruction of the wall of the organ, it passes through the lymph-channels or the tissues of the wall and in this way spreads in the neighborhood of the appendix. The fact that in the majority of cases the appendix is found perforated is due to the relative frequency of this accident.

The question whether pus is present in the tumor is still more important, for the treatment largely depends on this point.

Attention has already been called to the possible anatomic characteristics of the tumor. In some cases, which are now fortunately rare, there is an abscess full of pus ; there are also distinct fluctuation and possibly some cutaneous edema, so that the diagnosis is easy. A large abscess may, however, be present without any fluctuation. This is the case in deeply seated swellings or in more superficial swellings when there is much pain with rigidity of the abdominal walls (these difficulties can, of course, be overcome by an anesthetic); in cases, further, in which there is much thickening of the peritoneum or of the fasciæ and muscles, or in which there is much fat, fluctuation may not be detected. In such cases the diagnosis of large abscesses depends on the same signs as in smaller accumulations.

In another and smaller group of cases operation shows the existence of a circumscribed fibrinous or serofibrinous exudate during the first period of the disease. In these cases there is only a certain amount of resistance, behind which, however, a large abscess may be hidden.

I do not think that in such cases a decision as to the nature of the swelling can be arrived at from external examination alone. It is only from the course of the swelling that definite conclusions as to its character can be drawn; thus, if a swelling in the right iliac fossa which does not really possess the conformation of a tumor rapidly disappears, the existence of a large abscess may be excluded, and a serous exudate may be diagnosed with fair certainty.

The most important question is, after all, whether the swelling contains pus, even though only a small quantity, or whether it is due to inflammatory swelling of the tissues, a fibrinous exudation, or an accumulation of feces. Let us consider the various signs that aid us in deciding this matter.

It is clear that the hardness of the tumor does not militate against the presence of a small amount of pus. The whole question, already referred to above, simply turns on the one point whether it is justifiable to assume the presence of pus when the tumor is hard and solid. The adherents of the theory of a minute central collection of pus claim that the acute course of the disease and rapid development of symptoms definitely point to perforation, and this always leads to suppuration; the reverse—*i. e.*, slow insidious course—is against suppuration. Experience shows, however, that both conclusions are incorrect, for pus may be found in chronic cases and may be absent in cases that run a rapid course.

The temperature is more important, for it gives valuable information in large and small abscesses even when the former are hidden. If the fever continues after the first few days or after the first week, and when there are considerable elevations, the presence of pus is probable. The type of the fever, whether continuous, intermittent, or remittent, is unimportant; on the other hand, it remains to be seen whether different temperature-curves, especially the absence of fever, are against pus. The temperature may fall to normal after ten, seven, or five days, and yet pus may be found on operation; this may also be the case when there has been very little fever during the first few days, or even no fever at all. Or, again, the fever may disappear after a few days, and a period of apyrexia supervene lasting for several days or weeks, and a favorable, though false, prognosis be naturally given, when suddenly the fever lights up again and a definite abscess appears or diffuse peritonitis develops.

This variable character of the temperature shows that when the fever continues or reappears after apyrexia, pus may almost certainly be suspected, but that, on the other hand, pus may be present when the fever runs almost any other course. Thus the temperature is not without value in recognizing pus, but its value is limited and relative.

Roux has described a peculiarity in the wall of the cecum as characteristic of pus: on palpation the wall is said to feel like "paste rendered soft with water"; this is said to be due to edema of the wall in cases of perityphlitic suppuration. I cannot criticize this phenomenon, as I have had no experience of it.

K. Lewin has advised as a help to diagnosis the application of a hot fomentation. When an acute purulent attack due to infection is in question, the pain is said to be increased by the application of heat, in contradistinction to non-purulent cases, where it is diminished or entirely ceases (with chronic cold abscesses this is not the case). Unfortunately, from own experience, I cannot recommend this simple procedure as trustworthy. Further aids to diagnosis are obtained from microscopic examination of the blood. When the iodin reaction of Ehrlich shows the appearance of glycogen in the leukocytes, this indicates, according to Goldberger and Weiss, a progressive formation of pus (excluding diabetes and pneumonia); when the abscess is stationary, the typical iodin reaction is wanting. Schnitzler in particular emphasizes the importance of this method of examination.

Curschmann has drawn attention to the diagnostic importance of marked leukocytosis. When pus is formed, the leukocytes increase and suppuration is certainly present when the number of leukocytes reaches or exceeds 25,000 to 30,000. Leukocytosis is said to be a more certain sign of pus than palpation or other physical signs. The observations of Curschmann have been confirmed by several other observers—*e. g.*, DaCosta, Joy and Wright, Schnitzler; my own hospital experience points to the same conclusion. Naturally, pneumonia must not accompany the attack.

Exploration with a Pravaz needle may positively prove that pus is present; opinion varies as to its value. Some authors recommend it warmly; others condemn it; Roux, for instance, says that it is sometimes dangerous, sometimes useless, always unnecessary. No doubt the aspiration of pus clinches the diagnosis in a doubtful case, whether the pus comes from the appendix or a perityphlitic abscess, and it is also a good way of convincing nervous patients of the necessity of an operation. On the other hand, small collections of pus may escape detection even on repeated aspiration, and in this way the diagnosis is, if anything, made still more uncertain; and, lastly, the patients become even more unwilling to submit to an operation. There is some difference of opinion as to the danger of aspiration: some claim that it is dangerous on the grounds that infection may be carried to uninfected parts, so that the exudate, for example, may become infected, whereas others argue that peritonitis is never due to this cause. I agree with the latter view, and may add that if no pus is evacuated with the needle, there is no danger of spreading infection by the needle or the cannula. When, on the other hand, pus is found, an operation will always be performed anyhow. I never aspirate now, because in cases in which aspiration is indicated I prefer to proceed at once to operation.

To sum up, it may be said that there are only four symptoms which strongly point to the presence of pus—viz., distinct fluctuation of the tumor, positive results on aspiration, high fever, and considerable leukocytosis. Some of these symptoms, however, are absent in a great many cases, and yet pus is found on operation, usually in the form of the " pus nucleus " described above. How is this fact explained? The explana-

tion is that a perityphlitic tumor almost invariably contains some pus ; thus a correct diagnosis does not depend so much upon a rigid analysis of the clinical symptoms as upon correct conclusions that can be drawn from our knowledge that in perityphlitic tumors there is almost invariably a small accumulation of pus—the " pus nucleus."

Clinical Picture (Continued) in the Different Terminations of Perityphlitis.—When the disease has reached its height, its further course varies. In what follows the disease will be described as it formerly developed before operative treatment was so common.

When a spontaneous cure results, the vomiting and the fever first pass off, and then the tumor and the resistance begin to diminish. Even with a considerable leukocytosis at the onset it does not necessarily proceed to the formation of an abscess. This may occur very rapidly, and the swelling may disappear in two or three days, or is, at any rate, very much smaller. In the latter case the rapid disappearance may sometimes be due to the removal of a mass of feces ; but when we consider how rapidly an edematous swelling may disappear in furuncle as soon as the pus plug is removed, it can readily be understood that the same may occur in perityphlitic swellings. It is possible that in these cases the pus escapes into the bowel through the appendicular wall or through the lumen of the appendix, and that after the evacuation of the pus the edema is absorbed. As has already been pointed out, this is not the only nor the most common way for the pus to disappear : it is probably more often directly absorbed by the peritoneum.

Most of the cases of perityphlitis that recover without operation are cases of this kind in which there is a small amount of pus surrounded by an edematous swelling, and possibly, in addition, a little serous exudate. After the absorption of the tumor the patient appears well, and may remain in good health, but usually there are recurrences ; all that has been said about the recurrences in simple appendicitis applies equally to this form.

When the pus spreads, the conditions are different. The temperature may drop to normal within a few days, but, as a rule, some fever of a continuous, intermittent, or remittent type persists. The abscess continues to increase in size, and a number of local symptoms develop which depend on the size of the abscess and on the direction in which it is spreading. The general health fails, the appetite is lost, and eventually death supervenes unless the pus is evacuated.

The various directions in which the pus may travel have already been described ; the subsequent course of the disease depends on the organ or tissues involved, or into which the pus is discharged. In the most common form of evacuation—viz., into the bowel, particularly the cecum—small quantities of pus appear in the stools for a number of days or pure pus may be passed by the rectum. After this, pus is no longer seen in the stools, not because the abscess has healed and ceased to discharge or because the opening into the bowel is closed, but because the amount of pus, being very small, becomes so intimately mixed with the stools in their passage through the bowel to the

anus that it is not detected. When the abscess begins to discharge, the temperature falls, the general health improves, and recovery may result. When the abscess discharges into the bladder, pus appears in the urine and secondary cystitis may develop.

[Fowler [1] refers to a case of vesical calculus due to rupture of an appendicular abscess into the bladder.—ED.]

Perforation through the skin need not be described, and perforation through the diaphragm will be dealt with in the section on Subphrenic Abscess.

As already pointed out, perforation into the intestine may lead to complete recovery, but the adhesions that remain may, of course, be a source of danger and may lead to constriction of the bowel. Again, the greater part of the pus may be discharged, but a little may remain behind for years, sometimes without producing any symptoms; or there may be a recurrence, or more serious results, such as phlebitis or diffuse peritonitis, may start from the pyogenic micro-organisms which have been dormant so long. This is more likely to occur when the pus is not discharged through a perforation, but is merely absorbed.

The clinical picture of diffuse inflammation of the peritoneum due to appendicitis and perityphlitis remains to be described, but no detailed description is necessary, as the condition has already been exhaustively dealt with in the special sections on Diffuse Septic and Acute and Chronic Suppurative Peritonitis. Attention will only be drawn to a few points which have a direct bearing on this subject, and I shall largely adopt Rotter's views, with which I agree in the main:

It may be mentioned here that Burckhardt objects to the term " diffuse " or " general " peritonitis, and prefers to use the term " progressive." Many cases of apparently general peritonitis are, according to him, only progressive peritonitis; " in these cases the peritonitis is limited to a portion of the abdominal cavity, but presents grave general symptoms, such as we are accustomed to see in advanced cases of general peritonitis, using the term in its proper sense. Sprengel, too, agrees with Burckhardt in preferring the term " progressive " to the term " general."

The course and the onset of diffuse peritonitis are somewhat different when it starts from the appendix than when it starts from a perityphlitic abscess.

In the first group there is almost invariably rapid perforation of the appendix; the organ is either still free or the adhesions that may have formed are insufficient to prevent the entrance of infective material into the free peritoneal cavity.

It is rare to see perforation of an appendix that is still free from adhesions; Fowler describes such a case in which, twenty-nine hours after the onset of the disease, a violent general septic peritonitis developed, and in which the appendix was gangrenous and perforated at its distal extremity, but there were no inflammatory products in its immediate vicinity, and nothing was found in its lumen—*i. e.*, neither fecal concretions nor hardened feces. Similar examples are found fairly often in recent literature.

[1] Fowler, *Medical News*, May 21, 1898.

In such cases the picture of appendicular disease may not appear at all, and the patient apparently succumbs to diffuse peritonitis with collapse, low temperature, etc. In other cases the violent pain of perforation follows the mild symptoms of a local appendicitis or perityphlitis.

The course of these forms depends on the virulence and number of the bacteria that enter the peritoneal cavity.

The extremely rare cases of very acute peritoneal sepsis in which no time is given for the formation of an exudate always run a rapidly fatal course whether or not an operation is performed.

Other cases with an equally rapid onset present the syndrome of perforation and run a different course.

Some of the cases with the characters of diffuse peritonitis run an acute course and prove fatal; only a few recover. These cases, as a rule, are purulent, saniopurulent, or fibrinopurulent; I believe, however, that if the operation is performed sufficiently early, a purely serous exudate may occasionally be met with in these cases.

Again, the onset may be as above, and the diffuse symptoms may recede and only those of a local process remain, with a swelling or an exudation tumor in the appendicular region. I agree with Rotter in the view that in these cases the whole peritoneum was invaded, but that, as the amount of infectious material was so small, the peritoneum could resist it, and that the process persists only near the primary focus of invasion—*i. e.*, around the appendix, where the amount of infectious material is greater, or there may be a secondary invasion which the tissues around the appendix cannot resist, and hence the development of the perityphlitic tumor. As before remarked, Moskowicz is of the opinion that more often than not the peritonitis is not circumscribed, but commences as a diffuse peritonitis which later localizes itself as perityphlitis.

In the second group of cases peritonitis follows a peri-appendicular abscess. This may occur in two ways—either the abscess perforates into the general peritoneal cavity and produces acute sanious or purulent—*i. e.*, septic—peritonitis, as above, or the inflammation extends in the peritoneum without perforation, so that, in addition to a diffuse peritonitis, there are a number of circumscribed sacculated collections of pus in the peritoneum (progressive purulent fibrinous peritonitis of Mikulicz). In the latter case the course is slower and recovery is not out of the question, even without an operation. From the above it is clear that in this group the symptoms of diffuse peritonitis make their first appearance after the formation of a perityphlitic abscess of greater or less extent. It would be important and interesting to know when and under what conditions the incidence of peritonitis must be feared. Unfortunately, this is impossible, and it is this uncertainty and the constant fear of an unexpected turn in the course of the disease that induce the advocates of surgical interference to advise operative interference in every case of perityphlitis.

Diffuse peritonitis occurs in cases with continuous fever, in cases

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with an increasing abscess, and even in cases when there is no fever and the general health seems to be improving. This general peritonitis may come on without external cause when the pus slowly works its way through the protective adhesions and without any clinical sign suddenly perforates. Sometimes predisposing causes can be recognized, such as getting up and walking about or vigorous movements in bed ; for these reasons it is always dangerous to move these cases. The administration of a laxative has been known to produce the same result.

It need hardly be mentioned, in conclusion, that phlebitis of the mesenteric veins, with all its consequences, as well as peritonitis, may occur at any time during the course of perityphlitis, and that it was pointed out in the sections on the morbid anatomy that these events may occur even years after the process has apparently become quiescent. (For the clinical aspect of these conditions the reader should refer to the description of abscess of the liver and to inflammation of veins in general.)

[Thrombosis may occur in either of the lower extremities as a result of appendicitis. As bearing on this, Dr. B. N. Tebbs has kindly analyzed for the editor 400 cases of appendicitis treated at St. George's Hospital in the five years October, 1898, to October, 1903. There were 8 cases of thrombosis in the legs : in 4 the left leg was affected and in one of these the right leg was subsequently affected ; in 3 cases the right leg was attacked and in one of these the left arm was subsequently affected ; in one of these embolism occurred a week before any thrombosis could be detected ; in one case the side on which the thrombosis occurred was not noted. Embolism occurred in 3 of these 8 cases and was fatal in one ; in the other 2 cases there were two attacks of embolism. Middeldorpf[1] has recorded a case of appendicitis in which gangrene necessitating amputation of the thigh occurred and was thought to be due to parietal thrombosis of the external iliac artery, which provided the embolus found in the femoral artery after amputation.—Ed.]

Pleurisy is not an uncommon complication of appendicitis. Wolbrecht gives the percentage as 38 ; the exudation is generally on the right side, and is serous, seldom purulent. This, however, is not a complication peculiar to perityphlitis, but appears with any other form of peritonitis, and is rather free or localized. (See p. 779.) Other complications of the lungs or signs of pleurisy are attributed by Gussenbauer and Sonnenburg to emboli originating from thrombi, which are always found in appendicitis and perityphlitis. Emboli have been observed in patients even operated upon between the attacks. [Oppenheim[2] believes the emboli come from veins in the pelvis, and that operative manipulation is not responsible for their detachment. Purulent embolism may occur after appendicectomy, and some days later thrombosis of the femoral vein may appear, the emboli presumably having come from veins in the pelvis from which the thrombosis subsequently spreads to the more superficial veins.

[1] Middeldorpf, *Deutsch. med. Wochenschr.*, 1903, p. 540.
[2] Oppenheim, *Berlin. klin. Wochenschr.*, 1902, vol. xxxix., p. 94.

Dieulafoy's[1] description of purulent and putrid empyemata of appendicular origin and of the spontaneous production of gas in these empyemata has already been referred to (p. 867). In the 400 cases at St. George's Hospital analyzed by Dr. B. N. Tebbs there were 2 cases of empyema, 1 of pyopneumothorax, 2 of pleurisy, and 4 of pneumonia. Appendicitis may give rise to more wide-spread infection. Anderson[2] has recorded a case where pneumococcic endocarditis of the aortic valves was thought to depend on infection from a necrotic patch in the appendix. It has been suggested that cholecystitis and cholelithiasis may be due to colon bacilli derived from appendicitis (Ochsner[3]).

Martin[4] has described a case of intestinal intoxication due to chronic appendicitis, but in which the cause was not discovered until the appendix was removed. The symptoms were loss of flesh and appetite, slight fever, cachexia, and very offensive motions. The appendix was greatly thickened and contained very foul contents, which probably escaped into the intestine and produced intestinal decomposition and toxemia. It is possible, as suggested by Godlee,[5] that general toxemia is comparatively often due to appendicitis.

Parotitis is a very rare complication of appendicitis; it did not occur at all in 400 cases at St. George's Hospital. Fiske Jones[6] has recorded a remarkable case in which there were three separate attacks of double parotitis coming on after recurrent attacks of appendicitis with abscesses; on each occasion the right parotid became swollen forty-eight hours after the abscess was opened, and forty-eight hours later the left parotid became affected.—ED.]

RECURRENCES.

Inflammations of the appendix and perityphlitis show a decided tendency to recur, and this fact is another important argument in favor of operative treatment.

The reason why and the method in which these relapses occur have already been explained, but a few points require some additional remarks. There are numerous statistics as to the frequency of recurrences. Rotter claims 21 per cent.; Sonnenburg, 16 per cent. Sahli's figures are more valuable, for they deal with a larger number of cases—*i. e.,* 4593 from private practice; they were taken from a collective investigation in which many practitioners participated. Here 20.8 per cent. of recurrences were found. Of course, the only cases included were those in which a spontaneous recovery occurred without operative interference. [In his 257 cases Fitz found relapses in 11 per cent.; Hawkins,[7] in 250 cases, in 23.6 per cent.—ED.]

The majority of recurrences occur within a year—herein Rotter

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[1] Dieulafoy, *Clinique Médicale de l' Hotel-Dieu,* 1901–02, vol. iv., p. 84.
[2] Anderson, *Canadian Lancet,* April, 1898.
[3] Ochsner, *Phila. Med. Jour.,* October 6, 1900, p. 652.
[4] S. Martin, *Medico-Chir. Trans.,* vol. lxxxiv., p. 197.
[5] R. J. Godlee, *Lancet,* 1903, vol. ii., p. 1551.
[6] D. F. Jones, *Boston Med. and Surg. Jour.,* vol. cxlvii., p. 565.
[7] H. P. Hawkins, *Diseases of the Vermiform Appendix,* p. 112.

Sonnenburg, and I agree; they are less common in the second year, and after that recurrences are exceptional; I have, however, seen one after nine years, and in one case, basing my diagnosis largely on the statements of the patient, after eighteen years; it is questionable whether in the last case one can fitly speak of a recurrence; it seems possible that such a thing can occur, for a case of Kümmel's had the first attack when she was twenty-one, the second at thirty, the third attack at thirty-five, and the fourth at thirty-six years. [Lockwood[1] reports a case in which the first attack occurred at the age of forty and the second fifteen years later.—ED.]

As a rule, there is only one relapse, but in several cases 4, 6, or as many as 10 have been noted. If all the cases are regarded as recurrences in which the patient complains of pain and a little resistance in the right iliac region every three to six weeks, combined with constipation and vomiting, a large number of cases with many more than ten "attacks" would result. C. Beck operated upon a young man who in fifteen years had experienced 36 attacks, mostly slight in character. The clinical signs pointed to a functional disturbance of the bladder, to which the appendix was firmly adherent. Without an operation it is, of course, often difficult to be certain as to the anatomic basis of these attacks, but a number of cases that have been operated upon during one of these attacks have been shown to be chronic appendicitis.

It is very interesting to note that perforation is not specially common in relapsing cases. In 55 cases operated upon by Kümmel all had a number of recurrences; the appendix was perforated only in 22 cases, and not in 33, and in 27 of these 33 chronic inflammation of the organ with ulceration and stricture, but without perforation, or only simple chronic inflammation and thickening of the muscular coat and swelling of the mucous membrane, could be found. Other operators, as Sonnenburg and Lennander, have published similar observations. [Godlee[2] considers that small suppurating foci, either in the appendix or its immediate neighborhood, are probably often present.—ED.]

DIAGNOSIS.

Appendicitis and perityphlitis can usually be easily recognized from the characteristic position of the tumor and of the pain, the frequency of initial vomiting and constipation, and of the frequent febrile onset. But there are often difficulties which may even be quite insuperable; in the first place, the initial pain may be misinterpreted from the fact that the appendix is in an abnormal position; there may be severe general or intestinal symptoms from the onset, which mask the true nature of the disease, while other morbid conditions in the right iliac fossa may simulate perityphlitis, or, lastly, late secondary symptoms may appear.

I believe that a reliable differential diagnosis can be made only by a careful consideration of all the details. As I cannot give all the details

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[1] C. B. Lockwood, *Appendicitis, Its Pathology and Surgical Treatment*, p. 128.
[2] R. J. Godlee, *Lancet*, 1903, vol. ii., p. 1551.

here, and as a superficial discussion is of no value, I must refer the reader for most of the details to the special paragraphs on the symptomatology and shall limit my remarks here to a consideration of a few of the most important points. Of late years several treatises have appeared dealing solely with the diagnostic errors in connection with perityphlitis (Mühsam, Huber, Daver, and others).

(*a*) In the very earliest stages, when there is much pain, the following conditions must be considered : Renal, intestinal, and biliary colic and gastralgia.

The differential diagnosis is, as a rule, quickly and easily made, but, conversely, diseases originating in these organs may simulate perityphlitis, particularly the pathologic effects of gall-stones in displacement of the liver or in cirrhosis. In isolated cases a movable kidney on the right side, with symptoms of strangulation, has imitated perityphlitis.

[The diagnosis between lead colic and appendicitis may give rise to difficulty, and the two conditions may occur together ; Apert[1] has recently described cases of appendicitis and lead colic in workers engaged in pearl setting, which were extremely puzzling from the point of view of diagnosis.

Acute intrathoracic disease, such as pleurisy, empyema, pyopneumothorax, or pneumonia, on the right side, may, from referred pain in the right iliac fossa, strongly suggest that there is appendicitis, especially when the physical signs in the chest are absent or are overlooked. Barnard[2] and M. H. Richardson[3] have recently drawn attention to these cases. When the pain and muscular tenderness are, as they may be at first, entirely limited to the lumbar region, the condition may be diagnosed as lumbago (Mackenzie[4]).—ED.]

(*b*) The diagnosis is difficult or even impossible when the appendix is abnormally long or when it is displaced together with the cecum. In such cases a true peri-appendicular swelling may be mistaken for a subphrenic, a perinephritic, or a gall-bladder swelling ; or, again, the tumor may be found in the region of the umbilicus or in the left iliac fossa, or, as has been recorded, in the pelvis ; in such cases, particularly when the history is obscure, the diagnosis is difficult. In women a diagnosis must be made between perityphlitis and diseases of the right adnexa, especially pyosalpinx.

(*c*) In another group errors in diagnosis may arise from the presence of severe intestinal and peritoneal symptoms which imitate conditions other than appendicular disease. Thus the picture of acute intestinal obstruction, or intussusception of the ileocecal valve, or even tuberculous peritonitis, may be simulated.

Conversely, perityphlitis is simulated when, in peritonitis from some other cause, the pain is localized in the ileocecal region. Thus peri-

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[1] Apert, *Soc. Méd. des Hop.*, Paris, February 27, 1903.
[2] H. L. Barnard, *Trans. Clin. Soc.*, vol. xxxv., p. 122.
[3] M. H. Richardson, *Boston Med. and Surg. Jour.*, April 17, 1902.
[4] J. Mackenzie, *Brit. Med. Jour.*, 1903, vol. ii., p. 66.

typhlitis has been diagnosed in perforation of gastric, duodenal, and typhoid ulcers; also in intussusception of the ileocecal valve.

A simple but intense enteritis localized chiefly in the region of the cecum suggests appendicitis. Even typhoid has been suspected in a case of appendicitis, and, conversely, the latter has been diagnosed and actually operated upon when typhoid was really found.

[As already pointed out, appendicitis and perforation may occur in the course of typhoid fever, so that the two conditions coëxist; the surgical aspects have been discussed by Deaver.[1]—Ed.]

(*d*) In rare cases other diseases in the right iliac region, such as new growths, cause confusion. On the one hand, fever may occur in malignant neoplasms, with secondary inflammation and suppuration, and a perityphlitic tumor may be thus imitated; or, on the other hand, a perityphlitic tumor may be hard, and while there is no fever, there may be loss of appetite and of flesh, pallor, etc., with other apparent signs of a malignant growth, so that only very careful examination can prevent mistakes in such instances.

[Primary carcinoma of the vermiform appendix, which is a rare disease, as a rule presents the clinical manifestations of relapsing appendicitis; in a case published by the editor[2] there were 4 attacks; in one of Harte and Willson's[3] cases the appendix was removed for suppurative appendicitis. Barker[4] describes cases of ruptured caseous glands, rupture of a pyosalpinx, a twisted ovarian cyst, and hematoma of the right broad ligament as having exactly imitated appendicitis. Eve[5] has drawn attention to the resemblance between tuberculosis of the cecum and appendicitis.—Ed.]

Occasionally, particularly when no tumor can be felt, a retrocecal peri-appendicular affection may lead to the diagnosis of psoitis or psoas abscess, and hip-joint disease may be simulated by the situation of the pain and the position in which the thigh is kept.

The diagnosis of chronic appendicitis or perityphlitis presents no difficulties when the subjective symptoms attract attention to the ileocecal region, and the thickened appendix or perityphlitic tumor is easily recognized. It is more difficult when the prominent symptoms do not point to this region, or, again, when pain is absent or when the symptoms imitate some entirely different disease. Ewald aptly describes these cases as "larval appendicitis," as the clinical manifestations are masked by other unusual features. Since operative procedure has directed attention to these cases, they have been found to be by no means uncommon, and my own opinion is that they are frequent.

Most often the patient complains of the stomach or bowel or both, discomfort of the whole abdomen, and more or less severe pain. Often it is referred definitely to the epigastrium, umbilicus, or neighborhood of

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[1] Deaver, *Trans. Coll. Phys. Phila.*, 1898, vol. xx., p. 9.
[2] Rolleston, *Lancet*, 1900, vol. ii.
[3] Harte and Willson, *Medical News* (N. Y.), August 2, 1902.
[4] A. E. Barker, *Brit. Med. Jour.*, 1903, vol. i., p. 479.
[5] F. S. Eve, *ibid.*, 1903, vol. i., p. 608.

the gall-bladder, so that the stomach, bowel, or gall-bladder are thought to be the seat of the disease. The pain is intermittent or continuous, comes on without any cause or as the result of exertion or the act of defecation. There is generally constipation. Sometimes there is definitely functional disturbance of the stomach, with loss of appetite, flatulence, retching, vomiting, or pain during digestion. Some patients gradually develop neurotic symptoms, and may become thin, so that the patient is thought to be primarily neurotic or neurasthenic. Occasionally bladder trouble is simulated, when the appendix is adherent to the bladder. Riedel quotes a case illustrating well this error of diagnosis, in which simple appendicitis granulosa occurring in a small appendix of 3 centimeters and almost completely fixed caused marked symptoms for eighteen years; these ceased completely on removal of the organ.

Now, since it is recognized that these conditions may be caused by a simple inflammation of the appendix, this sequence of events must be borne in mind when clinical symptoms are not absolutely definite. The ileocecal region should always be carefully examined for any indication of disease of the appendix. Special stress must be laid on the fact that there may be no evidence pointing to appendicitis, since it may develop quite insidiously and without any local symptoms.

In contrast to larval appendicitis I have described a case of pseudo-perityphlitis in which the clinical aspect so closely resembled perityphlitis that laparotomy was performed in a surgical hospital. No anatomic lesion was found in the appendix, which was removed. The patient was a hysterical young male, in whom the same symptoms appeared a few years later and were speedily cured by faradization of the ileocecal region. I have met with two other cases of the same kind in which there was a localized, quite exceptional hyperalgesia of a raised fold of skin in the right lower quadrant of the abdomen, analogous to the hyperalgia of the abdominal wall in pseudoperitonitis.

[Talamon[1] described recurrent nocturnal attacks imitating appendicitis in a boy aged eight years, and Fischer[2] has recently described 2 cases of pseudo-appendicitis in children aged ten years. More puzzling still are cases where slight appendicitis in a neurotic subject sets up hysterical manifestations simulating diffuse perforative peritonitis and leads to unnecessary laparotomy.—ED.]

Lastly, reference should be made to those obscure cases in which secondary symptoms appear, often after years of quiescence, such as abscess of the liver and empyema of the pleura; in most of these cases we are dealing with so-called occult, cryptogenetic sepsis. In these cases the appendix should always be thought of when the origin of the sepsis is obscure; for an inflammation of this organ, now latent, may have caused a phlebitis or an intraperitoneal abscess as a late secondary manifestation.

I saw only recently such a case in a young man of thirty years with septic fever and chills, loss of flesh, and temporary rigidity and increased peristalsis of

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[1] Talamon, *La Med. Mod.*, March 31, 1897.
[2] L. Fischer, *Pediatrics*, January 1, 1902.

certain coils of intestine. Nothing could be found to explain these symptoms, and there was no history of past perityphlitis. As it is advisable to combine all the symptoms under one heading, and as the chief symptoms seemed to be those of intestinal stenosis with increased peristalsis, I diagnosed an intraperitoneal abscess with adhesions and kinking of the intestine, and, at the same time, septic fever. As there was nothing to point to the cause, I diagnosed a latent perityphlitis because this condition is relatively common. On laparotomy a large encysted abscess was found between the coils of the small intestine, and at the autopsy, soon afterward, several other intraperitoneal abscesses, progressive purulent fibrinous peritonitis, phlebitis of the radicles of the portal vein and the trunk of the portal vein, abscesses of the liver, and an old, almost healed perityphlitic abscess with perforation of the appendix.

A few words as to the question whether it is possible to differentiate forms of perityphlitis and appendicitis which have a different etiology from the simple form from the latter. This can probably be done only in rare instances, because there are no characteristic differential features. I do not think it is possible to tell at the bedside whether the tumor is due to decubital ulceration and perforation of the cecum or to ordinary appendicitis. When appendicitis occurs in a decidedly tuberculous individual, the diagnosis of tuberculous appendicitis may be made, but further than that we cannot go. The diagnosis of actinomycosis can, of course, be made only by chance, unless the abscess is opened, when the diagnosis is naturally self-evident.

PROGNOSIS.

Appendicitis and perityphlitis are serious diseases in the same way that typhoid fever is. The majority of the cases recover, but the outcome can never be foretold and an unfavorable turn may occur at any moment. The medical man can, therefore, only say, in the early stages, or later in the stage of resolution, when the disease is running a favorable course, that the patient at the actual moment is in no immediate danger, but that the next hour may be fraught with serious consequences; and just as in typhoid there is always the possibility and dread of intestinal hemorrhage or perforation, so here complications are always possible.

After recognizing this point it must at the same time be admitted that the disease can undergo spontaneous resolution. The two conditions of appendicitis and perityphlitis must, however, be considered separately as regards the prognosis.

Whether in appendicitis a complete return to normal is possible, we must leave for the present. It is well known that very often a second attack never occurs. When total obliteration occurs, the probability of recurrence is reduced; the frequency with which this occurs cannot be stated, for attention has only recently been directed to simple appendicitis, and there are no reliable statistical data available; all that can be said is that even in simple appendicitis a complete restitution to the normal does not always occur, and that several sequelæ may develop after an attack.

To this class belong, in the first place, relapses, which may appear at any time, from causes which have already been explained.

[While unable to agree with Lennander that a recurrence is to be anticipated at some period or another in the history of every case, Treves[1] has no doubt that the balance of probability is in the direction of a second attack.—ED.]

Other consequences are, however, more important, and may affect the appendix and remain limited to this organ, or may involve other parts. It is, for instance, always impossible to predict whether a large perityphlitic exudation or diffuse peritonitis will not occur in a sudden and unexpected manner.

Whereas little is known in this connection about appendicitis, a great deal can be said about perityphlitis, and very imposing and valuable statistics are available. The information obtained from operations and statistics justifies the statement that perityphlitis, in the great majority of cases, is curable without operative interference. The statistics published before 1890 are particularly valuable, because they refer to a period when perityphlitis, being treated more from the medical point of view, was in most instances allowed to run its natural course.

A few figures may be quoted : Fürbringer, in three years, saw 120 cases, with recovery in 94 (78 per cent.) ; improvement in 14 (12 per cent.) ; death in 12 (10 per cent.) ; Renvers, 91 to 92 per cent. of recoveries ; Guttmann, 96 per cent. In my 130 hospital cases there were 85 recoveries ; 4 deaths without operation ; 30 improvements, and 11 referred to the surgical clinic. These figures refer to former years ; now operation is so common that I have not given the actual record. The high percentage of recoveries among my cases is explained by the fact that many of the patients insisted on leaving the hospital within fifteen to twenty or even twelve or ten days, without waiting until complete recovery had occurred. Curschmann reports 453 cases, with 4.5 per cent. of deaths, and 9 referred to the surgeon.

The material collected by Sahli from a number of medical men is remarkable, as it includes 7213 cases ; of these, 473 were operated upon, while 6740 were not. Of the latter, 591 (8.8 per cent.) died, and 6149 (91 per cent.) recovered ; recurrences occurred in 4593 cases, and of these, 3635 recovered without a second recurrence.

Rotter has published some particularly careful statistics, which are important, as they all date from 1893–95—*i. e.*, from more recent years ; all the figures are arranged on a uniform plan, and have been elaborated from various points of view. The statistics include 213 cases ; of these, 19 (8.9 per cent.) died. Of the 213, as many as 21 showed signs of diffuse peritonitis on admission and were all operated upon, with 7 recoveries and 14 deaths. These 14 deaths account for nearly two-thirds of the total mortality of 8.9 per cent.—*i. e.*, 6.5 per cent. Of 192 cases with circumscribed perityphlitis, 156 (82 per cent.) recovered under medical treatment ; of the remaining 36 (18 per cent.) cases, 33 were operated upon ; of these, 2 died ; 3 died without surgical intervention ; in other words, there were only 5 deaths (2.5 per cent.) in 192 cases of

[1] F. Treves, *Brit. Med. Jour.*, 1902, vol. i.

circumscribed perityphlitis ; of these, as Rotter shows, 1, or possibly 3, could have been saved.

[Treves [1] considers that the mortality—all forms of appendicitis being included—is not above 5 per cent.—ED.]

The conclusions from these figures will be given in the section on Treatment, and only the main prognostic conclusions to be drawn from them will be mentioned here.

Circumscribed perityphlitis in the greater number of cases is a curable disease : about 80 per cent. recover under medical treatment, and a considerable number of the remaining cases are curable by operative interference.   When the cases are carefully watched and surgical intervention is invoked at the right time, the mortality from perityphlitis can be reduced to a very small figure—*i. e.*, to about 3 or 5 per cent. Death can, of course, not be completely prevented, even when the cases are carefully watched, for there are cases in which accidental complications occur, cases in which the diagnosis remains obscure, and, lastly, cases in which, in an altogether unforeseen and incalculable manner, the whole peritoneum becomes diffusely involved at the very beginning of the disease.

The various forms of diffuse peritonitis are the main source of danger in perityphlitis ; in fact, with modern treatment, this complication is almost entirely responsible for the fatal cases.   The acute septic form is the most dangerous, especially when combined with perforation ; the prognosis is, comparatively speaking, most favorable in progressive suppurative peritonitis.

TREATMENT.

First and foremost, in expressing an opinion upon the treatment of appendicitis and its subsequent peritoneal sequelæ, a definite answer must be given to the following question : Should the medical attendant recommend operation in every case, or at least in the vast majority of cases, or is so-called internal treatment indicated and successful in a certain number of cases?   Since a definite answer must be given we are of the latter opinion.

The treatment in perityphlitis is on more uniform lines than is the case in many other diseases, but the few methods in vogue are very different.   The theoretic conception of stercoral typhlitis is responsible for purgation, while the recognition of perforation has led to the opposite form of treatment—*i. e.*, an attempt to place the bowel at rest by restricting the diet and giving opiates ; in addition, warm or cold applications locally and local blood-letting were all formerly used, and surgical treatment was employed only when the presence of an abscess was absolutely certain.

Treatment has been carried out along these lines as long as the disease was known.   Within the last decade, and with the recognition of the fact that the appendix is primarily affected, new lines of treatment have been adopted.   One group of medical men with advanced views advises operation and removal of the appendix in all cases ; nearly all

[1] Treves, *Brit. Med. Jour.*, 1902, vol. i.

the recent discussions have been chiefly concerned with the limitations of surgical intervention, and have led to a certain amount of agreement between physicians and surgeons. The latter have begun on the recommendation, chiefly of American physicians, to limit the number of cases in which operation is indicated; while the former have realized that the knife is needed oftener and earlier than they believed some fifteen years ago. As the result of all these discussions, publications, and the careful analyses of the sequelæ and the recurrences in appendicitis and perityphlitis, it is now possible to formulate certain individual rules and regulations for the treatment of these states.

The antagonism between physicians and surgeons which existed until recently can well be explained as follows : the former pointed to the fact that the majority of cases recovered under medical treatment, while the latter showed that every case was dangerous and that there was always a possibility of the process involving the whole peritoneum, and that there was, in addition, constant danger of recurrences.

No good would result from quoting additional figures to those already given in the section on the Prognosis, since statistics in general give very little information of value unless they are carefully analyzed, like Rotter's statistics, which show that it is necessary to consider each case carefully on its merits, and not to treat every case of perityphlitis surgically, or, on the other hand, to allow the proper moment for operation to pass by. Some cases should be operated without delay, while others recover on medical treatment. The medical treatment will be dealt with first, and then the indications for surgical interference. Appendicitis and perityphlitis will be dealt with together, as they cannot well be separated, so far as the treatment is concerned.

Medical, Non-operative Measures.—The most important thing is to enforce quiet, both general and for the intestine in particular. If the patient is not yet on his back, he should be put to bed at once; this applies not only to cases where there is a tumor, but also to simple appendicitis, in which there is also always danger of perforation. Rest in bed should not be relaxed until the patient is well on toward convalescence.

The diet is very important. Some authors, *e. g.*, Sahli, go so far as to forbid all food by mouth during the first few days, not only in perforative but in all acute cases, and feed the patient by the rectum with nutritive enemata and small quantities of water. There can be no serious objection to this plan, and while I recommend it in acute cases, I do not see any real objection to a little water by the mouth in tablespoonfuls, though not ice cold, as this amount of water is entirely absorbed in the first part of the small intestine. Stress need hardly be laid on the fact that all nourishment given by mouth should be liquid (soups, with egg, somatose, artificial infants' foods; milk should be given with care, for it may produce flatulence, and in this way stimulate peristalsis). If the case progresses favorably, care should be taken for some time, and everything that tends to distend the intestine and leads to gas-formation should be avoided.

The only means to put the intestine at rest is opium. The great importance of this drug in the treatment of perforation was recognized more than sixty-five years ago by the Dublin school (Graves, Stokes), and was then applied by French clinicians (Petrequin, Grisolle) to perforation of the appendix in particular. In Germany the opium treatment was introduced by Volz, and it has since occupied an important position. The same objections made against its use in acute intestinal obstruction (see pp. 640–643) can be urged in perityphlitis; many surgeons do this. I cannot agree with the objectors, for by forbidding opium, both the patient and the medical attendant are deprived of the best means of combating the disease. It is only those who advise operation in every case of perityphlitis who can condemn the use of opium, and as I do not adopt this standpoint, I strongly recommend opium. The chief objection—viz., that the drug masks the symptoms—is unimportant in comparison with its actual value, particularly if the case is carefully watched.

The preparations of opium itself—*i. e.*, the extract and the tincture—are preferable to morphin, which should only be used, and then subcutaneously, when opium cannot be given by mouth, and if the administration in suppositories (which are not always available at the onset) is impracticable. To start with (Sahli), 10 to 15 drops of the tincture should be given every hour for several hours, then 5 to 7 drops every two to three hours until the pain is gone. When the pain returns, large doses again; as soon as the pain stops completely the opium should also be stopped. The constipation may be allowed to continue for from four to eight days without any bad results, particularly as little food is eaten.

The objection that opium masks the proper time for operation is, as I have said, incorrect. The pulse, the temperature, the general constitutional condition, the condition of the local tumor, should give a careful observer sufficient guidance to determine the correct time for operative intervention.

In addition to the three factors, quiet, diet, and opium, the external application of compresses is important; they may be either moist and warm or ice cold. Without entering into any theoretic discussion, I may say that in acute cases cold is better.

[Lees[1] strongly advocates the persistent use of the ice-bag in acute appendicitis on the ground that it "rapidly relieves pain and obviously diminishes the local inflammation."—Ed.]

As soon as the fever drops and the acute tenderness on pressure disappears, Priessnitz compresses and later warm poultices should be used. The patient's own sensations should also be taken as a guide; thus, in some cases, cold can be applied longer than in others, or warm applications can be applied early or late.

Formerly I frequently applied leeches in acute cases; here, as in many other local inflammatory conditions, they appear decidedly to relieve pain and also to affect favorably the course of the disease. No

[1] D. B. Lees, *Brit. Med. Jour.*, 1903, vol. ii., p. 1454.

particular drawback arises from their omission. [It is better not to apply leeches or blisters to the abdominal wall, as the subsequent condition of the skin increases the chances of suppuration should an operation be performed (Lockwood[1]).—ED.]

When the process becomes quiescent, rest in bed should still be enforced, but a little more food may be given—minced chicken, calves' brain or thymus, scraped beef, rolls, boiled mush or rice, etc. Warm poultices should be continued; the exudate under this treatment will be seen to disappear rapidly. Painting with iodin and blisters I consider quite unnecessary.

There is no objection to the use of warm baths, with the addition of bran, salt, or mud, during convalescence; there is, however, no urgent necessity for their use. I strongly disapprove of massage in any form. Even though it may do good in some cases by hastening the absorption of an exudate, the possibilities of serious harm are so great that the measure must emphatically be condemned.

There is one other method to be considered which I have intentionally postponed to the last—viz., the use of purgatives and the general plan of evacuating the bowels. As long as the idea of a stercoral typhlitis held the field, this treatment, of course, played an important rôle; as this old idea must now be given up in the light of our present knowledge, the *a priori* indication for purgation must also be abandoned. Not only, however, is it unnecessary theoretically, but it is known to be harmful in most cases, and although, at the present time, a few observers—e. g., Bourget—are in favor of it, I must say that I agree with the majority of observers in condemning it (of the many who are against, we will cite only one of the latest authors upon this important point—viz., Baeumler). In the first few days of the inflammation everything should be done to keep the intestines at rest and to inhibit peristalsis. Even constipation lasting for a week is useful and does no harm. It is only when the initial storm is over and there is presumably (for there can be no certainty on this point) no further danger of perforation that the question of evacuating the bowels can be considered, and even then this object should be brought about with gentle enemata and not by the administration of purgatives.

It would be wrong to deny that occasionally the signs of perityphlitis disappear promptly after a purge. In these cases there are violent pains in the right iliac region, vomiting and constipation, and a distinct resistance or a palpable tumor, all of which disappear after a copious evacuation of the bowels. In my opinion, however, these are not cases of genuine acute appendicitis or perityphlitis, but examples of the condition already described, in which the appendix, inflamed or otherwise changed, becomes fixed to the cecum and in this way produces local fecal accumulation or a mild exacerbation of the inflammation, or appendicular colic.

As soon as the acute attack is over, the bowels should be kept regularly open; this advice is universally given, and though I indorse it, I

[1] C. B. Lockwood, *Appendicitis, Its Pathology and Surgical Treatment*, p. 226.

do not believe that a regular action of the bowels in any way prevents a recurrence.

[As the colon bacillus probably plays a very important rôle in appendicitis, Wright[1] has suggested that treatment should be directed to immunization of the patient against the colon bacillus, with the objects—(1) Of preventing relapses ; (2) to prepare a patient, who is going to be operated upon in the interval between two relapses, so as to minimize danger from accidental infection of the peritoneum by pus during the course of the operation.—Ed.]

Surgical Treatment.—The non-operative treatment has been dealt with first because I share the view of many clinicians that appendicitis and perityphlitis can, and in many cases do, get well without an operation, in such a way that the permanent anatomic alterations leave behind them no functional disturbances. The figures quoted in the section on the Prognosis bear this view out.

It is true that operative intervention has increased the proportion of recoveries. This, however, does not by any means prove, as some insist, that the disease properly " belongs to the surgeon," and should always be treated surgically, but only that operations should be performed more frequently than formerly, and that the indications for surgical treatment must be made more concise.

[Osler[2] says : " So impressed am I by the fact that we physicians lose lives by temporizing with certain cases of appendicitis that I prefer in hospital work to have the suspected cases admitted to the surgical side." Many would agree with this, as surgeons do not necessarily operate on all cases that come under their care ; another plan is for both a physician and a surgeon to watch the case. Both Dieulafoy[3] and Osler point out that there is no medicinal treatment of appendicitis. —Ed.]

The statements of surgical enthusiasts might almost lead the reader to believe that most cases of perityphlitis die unless they are operated upon ; that this view is erroneous is shown by statistics, especially by the remarkable data of the Vienna Pathological Institute ; among 45,000 (in round numbers) autopsies performed in twenty-seven years there were only 148 (*i. e.*, 0.3 per cent.) deaths from perityphlitis, some of which were from intercurrent peritonitis ; and it should be noted that during twenty of these years (1870–89) operations for perityphlitis were comparatively rare. There is a reaction at the present time against over-operating, and thoughtful surgeons (Helferich, Willy Meyer, Gersuny, and others) are discussing the problem now, as many did toward the end of the above period : When are we to operate for appendicitis ? This query implies that these cases cannot be treated according to any rigid rule, and that every case must be considered on its merits. Taking into account the enormous mass of communications upon this subject, it is impossible in a text-book to criticize the indi-

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[1] A. E. Wright, *Brit. Med. Jour.*, 1903, vol. i., p. 1073.
[2] W. Osler, *Practice of Medicine*, 1901, p. 530, fourth ed.
[3] G. Dieulafoy, *Acad. de Med.*, May 11, 1897.

vidual opinions of all observers (a comprehensive review is given in Böhm's collective investigation).

Surgical enthusiasts argue as follows : It is impossible to say that in any given case perforation into the general peritoneal cavity may not occur ; there is, therefore, always grave danger, even in cases with a mild onset, and this danger is present throughout the course of the disease. Further, no case of appendicitis is ever completely cured, and the patients are, therefore, in constant danger of a recurrence, with the same possible dangers as in the first attack ; even in cases that are apparently cured, the danger exists and may appear acutely at any time in the form of phlebitis, hepatic suppuration, etc. In addition, cases that are operated upon are usually more rapidly cured.

From this standpoint operation would naturally be advised in all cases and at an early date, as it would appear that the appendix ought to be completely removed. As I shall, however, show, this conclusion is only apparently correct.

Apart from the fact that the existing circumstances often make an operation impossible,—for example, when the patient refuses consent or because, especially in country practice, an operation is impracticable,— the following objections against this view, which are more important, must be considered :

While the operation, as a rule, is followed by favorable results, and whereas many surgeons never record a death provided there is not diffuse peritonitis or sepsis at the time of the operation, it must not be forgotten that these operations are performed by men of great experience, with all the appliances of a thoroughly well-equipped hospital at their disposal, and that it is doubtful whether the results would be equally good if inexperienced practitioners felt bound by current scientific opinion to operate in every case that occurred in their practice. While the mortality from operation is small, deaths nevertheless occur even with the most practiced and experienced operators, and, at any rate, the result can never be guaranteed. Indeed, my personal objection to the general demand for operation dates from the time when an otherwise healthy and strong girl, nineteen years of age, suffering from moderate symptoms of uncomplicated appendicitis, was operated upon between two attacks by one of our most brilliant surgeons and died. According to American communications, the mortality percentage (Willy Meyer) is 2. According to Sprengel's statistics, based upon 232 cases operated upon in the interval, it was barely 1 per cent. [In 1000 cases operated upon during the quiescent period, Treves [1] had 2 deaths.—Ed.]

This percentage, while small, is nevertheless relatively large in a disease with so small an absolute mortality ; in addition, some disagreeable sequelæ of the operation must be taken into account, such as a persistent fecal fistula or ventral hernias. Altogether, as Helferich says, the disadvantages of operative interference, while not very great, are still not so insignificant as to be neglected. Further details, espe-

[1] Treves, *Brit. Med. Jour.*, 1902, vol. i., p. 1589.

cially in relation to early or so-called preventive operations, will be touched upon later.

From a consideration of all these points the following conclusions may be drawn:

No rule based on general principles can be made that every case of appendicitis and perityphlitis should be operated upon.

The experience of the last decade has, however, shown that in emergencies and in many other conditions operations are necessary, and that they should be performed more frequently than formerly and upon considerably extended indications.

The problem is, therefore, to determine when an operation is called for. As Gersuny says, this decision cannot be based on prognostic data alone. Each case must be considered on its own merits, and the decision formed from these observations. For the sake of a more comprehensive review the cases will be divided into several groups, and special indications will be given for each category.

The most important and universally acknowledged indication for operation is to evacuate the pus from a perityphlitic abscess as soon as fluctuation can be detected. The formation of a large abscess is often unavoidable, even with our present perfected methods of treatment. It need hardly be mentioned, of course, that the abscess must be opened should it happen to be located in some other portion of the abdomen. The question of operation where a fluctuating abscess is not clearly indicated, but formation of a deep-seated abscess is suspected, will be considered later.

The second group of cases comprises those in which diffuse peritonitis is already fully developed when the medical man has to make his decision. A large proportion of these cases, it is true, prove fatal, even when operated upon; the operation and the anesthetic may be the means of hastening this result; on the other hand, chronic cases, particularly of the fibrinopurulent type, may recover without an operation, and in very rare instances this may occur in perforative peritonitis. Still, unless the case is already too far advanced, operation is, as a rule, the only chance.

Relatively, the best results are obtained in cases that run a less rapid course or that are subacute and chronic and of the fibrinopurulent type; the majority of cases of diffuse peritonitis cured by surgery belong to this category; the chief object in these cases, apart from the treatment of the perityphlitic abscess, is to provide free drainage for the pus collected between the coils of the intestine.

Fairly favorable and often remarkable results are obtained in acute perforative peritonitis due either to perforation of the appendix or to bursting of a perityphlitic abscess into the peritoneal cavity. These cases should be operated upon as soon as possible after the perforation, for life depends on hours, but occasionally recovery occurs after a delay of twenty-four hours.

The object of operation is to remove the source of infection and the pus from the peritoneal cavity, the evacuation of the abscess, and, in

addition (sometimes this alone), the removal of a gangrenous perforated appendix.

The prognosis is most unfavorable in acute and fulminating forms of septic peritonitis; in these cases the fatal issue is due to the severe toxemia, even when the local affection of the peritoneum is unimportant or absent. Only a few individuals escape death with this form of the disease, even if operated upon early. This has led some to advise against an operation in the severe fulminating forms, in order not to add the shock of the operation and of the anesthetic to the general symptoms of the disease; for occasionally these patients recover spontaneously after the operation has been declined on the ground that the case is hopeless. It need hardly be pointed out that such exceptional occurrences must not be taken into consideration in deciding whether to operate.

Symptoms of intestinal obstruction urgently call for operation in both acute and chronic cases of perityphlitis. When the symptoms of obstruction are due to intestinal paresis as a result of diffuse peritonitis, the fate of the patient may be considered settled with or without an operation; but, as has been pointed out above, the syndrome of intestinal obstruction in perityphlitis may be produced by other factors which can be removed surgically. The operation should, therefore, be undertaken at once, as soon as perityphlitis is diagnosed. A number of recorded cases show the evil effects of delaying operation, even for only one day. When, on the other hand, the diagnosis cannot be positively made and there is any doubt as to the pathogenesis of the obstruction, the case should be treated according to the general rules laid down in the section on Occlusion of the Bowel.

In the cases mentioned at present it is relatively easy to decide for or against an operation; the difficulty arises in deciding whether we should operate in the cases of acute appendicitis and perityphlitis seen in daily practice.

It has been shown above that for the last fifteen years this has been an open question—viz., whether or not to operate in every case of acute appendicitis at its immediate onset and perform appendicectomy. Most American, many English and French, and a certain number of German surgeons are in favor of operating in every acute case so soon as it is diagnosed. Certainly the fact that again and again in cases which at the commencement gave the impression of a slight attack, we find the most pernicious, even phlegmonous, gangrenous, or perforative changes in the appendix, leading almost at any moment to the worst form of general peritonitis, compels one to operative procedure on principle. All the more urgent is this since it is often impossible to come to any conclusion as to the pathologic condition of the appendix from the clinical aspect of the case.

Still, taking into consideration every known factor, we cannot entertain an unconditional demand for operation in every case. Some reasons for this have been given above. As regards early or prophylactic operation at the commencement of an acute attack, the following statement may be made here:

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According to general opinion, preventive operation can be of use only when it is undertaken within twenty-four or forty-eight hours of the commencement of an attack (in a few cases it was even then too late, but the disease ran an extremely acute course; they are, moreover, so rare that they may be neglected). As a matter of fact, in general practice the opportunity for such early operative interference is not at all common; when the disease has reached the third or fourth day, operation had better be postponed.

Even when the opportunity occurs of operating within twelve to thirty-six hours of the commencement of an appendicitis unaccompanied by any peritoneal symptoms, it should, in my opinion, be carried out unconditionally only when the assumption of a severe phlegmonous, "diphtheric," or gangrenous form is probable. This condition cannot be determined in the early stages by the clinical symptoms of the attack itself. It may, however, be suspected when appendicitis comes on in the course of an infective angina, or when, as mentioned above, the disease occurs in an epidemic form, for in these cases severe pathologic conditions will probably be found to exist.

Sometimes extremely virulent peritonitis complicates a case in which no single sign or symptom indicates the severity of the pathologic condition.

From consideration of the various published statistics and experience of numerous observers I have come to the conclusion that operation ought not to be performed as a routine practice in all cases of acute appendicitis, for although ultra-acute cases are most distressing and disappointing, they are fortunately rare as compared to the average run of cases, which run a less treacherous course and terminate favorably; but, unfortunately, the only course to be adopted in these exceptional cases is careful and continuous observation of the patient. Even in cases which appear quite simple and free from danger, signs may be detected which show that an operation should be undertaken without a moment's unnecessary delay.

[Treves[1] says much the same—viz.: "Our knowledge of the pathology of the disease and its general mortality will not sanction the practice of opening the abdomen in every case of appendicitis as soon as the diagnosis is established." After pointing out the urgent necessity for immediate operation at the earliest possible moment in all ultra-acute cases, he lays stress on the fact that the great majority of cases of appendicitis recover spontaneously without either an operation or the formation of an abscess. He adds that the ultra-acute cases are actually rare, and that, relatively to the whole mass of examples of all degrees, suppuration may be said to be uncommon.—Ed.]

The question when operation should be undertaken in a case where there is a definite inflammatory swelling present is still freely discussed. Many observers have given up the practice of operating in every case, and are in favor of considering each case on its own merits, and treating the cases medically at first and operating later if necessary; as a re-

[1] F. Treves, *Brit. Med. Jour.*, 1902, vol. ii., p. 1589.

sult of this selective method, which I consider the best, more operations are performed than formerly. Early operation has many drawbacks which should be neglected only for urgent reasons.

[Relapses may occur after operations carried out in the acute stages (Treves[1]), and Mynter[2] incidentally mentions that there were two relapses in 27 cases thus treated (though they may have been connected with the original abscess).—ED.]

One of the chief objects, of course, apart from opening the swelling and thus preventing contamination of the peritoneum, is to remove the source of all the trouble—the appendix. It is difficult, however, to find the organ when the tissues are in an inflamed condition, and removal of the organ may, under these conditions, be very dangerous. For this reason, then, and because a second operation for removal of the appendix is often necessary, and because the great majority of the cases recover under medical treatment, the general view at present is to operate more frequently and earlier than formerly, but never to operate unless there are very definite reasons for surgical interference. What are these indications?

It has been said that every case should be operated upon when perforation or the presence of pus is diagnosed, but, as already shown, this is not always easy. The question of operation must, therefore, be decided by a number of points which practical experience has shown to indicate the advisability of surgical interference; these points are: The temperature; the pulse; the pain, and the growth of the inflammatory tumor.

A high initial temperature with chills alone does not call for operative interference when the fever disappears rapidly—*i. e.*, in the course of two or three days (of course, without antipyretics, which, by the way, should never be employed in appendicitis). If a temperature which is uncommon in perityphlitis—103° F. and above—still persists on the fourth day, it is well to operate, for the infection must be virulent and an unfavorable turn may be expected.

The longer the duration of the disease, the greater the importance that must be attached to even relatively low temperatures—for example, 102° F. on the sixth day—as a sign that operative interference is necessary. When the temperature steadily falls and no other untoward signs appear, it may be well to wait; but if the temperature begins to rise again, and when this rise persists for twenty-four hours at the most, an operation should be performed.

In some instances, however, it would be wrong not to operate because the temperature is not high,—*i. e.*, either was low from the beginning or fell subsequently,—for, in the first place, the temperature may not be especially high in perityphlitis, or, again, there may be a large abscess, even when the temperature becomes normal on the sixth to the tenth day. These variations are probably due to the differences in the virulence of the bacteria; it must also be remembered that, particularly

[1] F. Treves, *Brit. Med. Jour.*, 1902, vol. i., p. 1589.
[2] Mynter, " Appendicitis," *ibid.*, quoted by Treves.

in acute diffuse peritonitis, the temperature may fall rapidly, though, at the same time, the general condition becomes worse.

Generally speaking, therefore, it may be said that a high temperature calls for operation, but that a low temperature does not exclude the necessity of surgical intervention.

The pulse is important as an index. A rapid pulse *cæteris paribus* shows that an operation is necessary, and though no hard-and-fast line, such as a pulse-rate of say 120, can be drawn above which an operation should be performed,—for there are many physiologic fluctuations in the pulse-rate,—the rapidity and quality of the pulse are both most important in arriving at a decision as to the question of operation.

The pain is less important than the pulse or the temperature. In the first place, the intensity of the pain varies in different individuals, and, in the second place, there may be a very serious condition without much pain. As a rule, however, long-continued spontaneous pain or pain on pressure usually points to severe suppuration, and is, therefore, indication for operation.

Lastly, the growth of the tumor is important, and if growth does not become stationary after a few days, the presence of pus is highly probable, and an operation is necessary (the temperature usually remains high under these circumstances); the same applies when, after a fall in the temperature, the tumor does not get smaller; the difficulties of determining the size of the tumor, particularly when the abscess is hidden deep down in the abdomen, must, of course, never be forgotten. Late vomiting is, according to E. Rose, an indication for operating.

[In addition to these factors a steadily increasing leukocytosis from hour to hour indicates that operation is necessary.—Ed.]

A new departure in surgery consists in the plan of operating in the interval between attacks—*i. e.*, when the process is quiescent. In fact, the good results obtained when the temperature is normal and there is no acute inflammation have led many surgeons to prefer the " *operation à froid*," and to consider the operation during the acute stage to be an emergency measure pure and simple. The surgical advantages of this method are obvious : the operation is, of course, easier and less danger-ous ; drainage is unnecessary, and healing by first intention is more readily secured, so that many of the disagreeable consequences connected with the operation wound in the abdomen are done away with.

In the acute stage removal of the appendix is important, but when the operation is performed during the quiescent stage, this is the essen-tial object of the procedure. What are the conditions under which the latter operation is advisable? They are chiefly two : the occurrence of relapses and the persistence of permanent sequelæ after an attack of appendicitis or perityphlitis.

The numerous sequelæ that may occur have already been described ; they are such a source of great distress to the patients that they welcome the relief promised by an operation. The physician can fitly advise this operation, because so far no death has been reported ; in addition, there is really no other way to relieve these cases, so that to advise opera-

tion is good practice, warning the patient of the danger, which it is impossible completely to avoid.

[Treves,[1] who in 1887, in a paper read before the Royal Medical and Chirurgical Society of London, recommended the removal of the appendix during the period of quiescence, has performed this operation over two thousand times with two deaths.—Ed.]

It is more difficult to advise an operation with the object of preventing possible recurrences if the patient is not suffering from any of the sequelæ; if there have been several recurrences, the removal of the appendix is, of course, advisable, even though no serious symptoms persist after the attacks, for the danger is the same in each attack, and, in addition, the changes in the neighborhood of the appendix are increased by each recurrence, and there is, therefore, an increased tendency to complications each time; hence many surgeons advise removal of the appendix in every case, even after only one attack of appendicitis, the organ being removed during the stage of quiescence. Still, I cannot indorse the radical advice that these surgeons give, for it is not by any means clear that a patient who has suffered one attack of appendicitis must necessarily have another; according to the statistics given above, only about one-quarter of the cases relapse (20 to 25 per cent.), and even though more careful statistical investigation were to show that relapses are more frequent, the proportion would still be relatively small. In a matter of this kind practical considerations and human nature must be taken into account, and however strongly an operation is urged on a person who has had one or several attacks, there will still be a natural reluctance to undergo any operation, for if the patient inquires, after his first attack, whether he is likely to have another, and he is told that recurrences occur in only about 20 to 25 per cent. of the cases, he will very probably refuse operation. How many will consent to it? Most people would certainly prefer to wait and see if there is going to be a second attack. However correct the dogma of operating in every case after the first attack may be, therefore, it cannot, from a practical point of view, be carried out.

Not being a surgeon, I will limit myself, as in the section on Occlusion of the Bowel, to stating the indications for operative interference, and shall not attempt to give any description of the surgical technic.

SUBPHRENIC ABSCESS (Abscessus et Pyopneumothorax Subphrenicus).

Subphrenic abscess is a localized collection of pus situated underneath the diaphragm. It may be intraperitoneal or extraperitoneal, for the term "subphrenic abscess" is purely topographic, and gives no further information as to the origin or nature of the abscess. Maydl, in his exhaustive monograph, mentions fifteen synonyms of the disease, and medical writers have for some decades been calling attention to subdiaphragmatic abscesses and to pyopneumothorax subphrenicus. Thus

[1] F. Treves, *Brit. Med. Jour.*, 1902, vol. i., p. 1589.

Cossy described a case of pneumothorax of the upper part of the abdominal cavity containing gas derived from the gastro-intestinal tract under the title "*faux pneumothorax*"; Bouchaud gave an account of this condition much earlier as "localized tympanites." In fact, G. H. Barlow, as early as 1845, from the physical signs during life, definitely diagnosed a cavity below the diaphragm filled with air and fluid, which was subsequently confirmed by the postmortem examination. These and similar communications, however, attracted no attention, and it is only recently, since Leyden introduced the name "pyopneumothorax subphrenicus" and drew up clear and distinct rules for its diagnosis, that this affection has become generally known. Leyden gave this new name to "the formation of large cavities below the diaphragm filled with air and pus which project more or less into the thoracic cavity and give rise to marked physical signs, greatly resembling those of true pyopneumothorax."

As regards their causation and general characters, the simple subphrenic abscess and those containing gas belong to the same class; they are separated only because the clinical manifestations of the latter are so peculiar and characteristic.

Subphrenic abscess is comparatively rare; in 1894 Maydl was able to collect only 179 cases (Lang, in 1895, including 3 new ones, only tabulated 176), and the greater number of these cases were published only after Leyden's monograph had drawn attention to this disease, but the number of recorded cases increases every year. [Since laparotomy for perforating gastric ulcer has become the rule, subphrenic pyopneumothorax has become even less frequent than before.—Ed.]

ETIOLOGY.

In the great majority of cases purulent peritonitis localized under the diaphragm starts from the viscera in the immediate neighborhood of the diaphragm, the most important of which are the stomach, the liver, and the duodenum, in this order. Some other starting-points will be mentioned later, but it is remarkable that the appendix, which is at a considerable distance from the diaphragm, so often gives rise to subphrenic abscesses; in fact, in Maydl's statistics the appendix came next to the stomach as the most frequent starting-point of this condition.

[In 1901 Elsburg[1] collected 73 cases of subphrenic abscess due to appendicitis. Of these, 20, or 27 per cent., were extraperitoneal and contained less pus than those which were intraperitoneal (35, or 48 per cent.). In 18, or 25 per cent., the anatomic relationship to the peritoneum was doubtful. In 25 per cent. of the 73 cases the diaphragm was perforated, and in 15 per cent. the abscess contained gas, which may be due to the Bacillus aërogenes capsulatus or members of the colon group. The abscess is not always on the right side of the abdomen, as is shown by a case of left-sided subphrenic abscess secondary to the appendix recorded by Christian and Lehr.[2]—Ed.]

[1] C. A. Elsburg, *Annals of Surgery*, 1901, vol. xxxiv., p. 729.
[2] H. A. Christian and L. C. Lehr, *Medical News*, January 24, 1903, p. 147.

The most important lesion in the causation of this condition is gastric ulcer, either with or without perforation. [Osler says 80 per cent. of subphrenic abscesses are due to gastric ulcer.—Ed.] The ulcer is usually situated on the lesser curvature or toward the cardiac end of this curvature, and only rarely on the greater curvature. In exceptional cases carcinoma of the stomach is the cause.

Duodenal ulcer produces subphrenic abscess relatively often, the ulcer being always near the pylorus and in the superior horizontal or first part. [According to Osler,[1] duodenal ulcer is responsible for 6 per cent. of the cases. Cullen,[2] on the basis of 184 collected cases of duodenal ulcer, states that an abscess forms outside the ulcer in 10 per cent. of the cases, and that the ulcer ultimately opens into the abscess. Bainbridge,[3] however, was able to collect only 11 cases where perforation of a duodenal ulcer gave rise to a periduodenal ulcer; it is remarkable that 6, or more than half of these 11 cases, were in women, in whom duodenal ulcer occurs five times less often than in the male sex. It may be pointed out, however, that a perforated ulcer which is regarded during laparotomy as duodenal may, as shown by more extended examination at the necropsy, be really inside the pylorus.—Ed.]

There are a few isolated observations on record of subphrenic abscess following perforation of a typhoid ulcer in the lower part of the ileum, after traumatic perforation (swallowing a fish-bone) of the cecum; and after perforation of the transverse colon and the hepatic flexure by dysenteric ulcers.

It has already been stated that perityphlitis is a frequent cause of subphrenic abscess, and Bamberger described such abscesses containing gas a long time ago. [In 73 cases of subphrenic abscess due to appendicitis 15 per cent. contained gas (Elsberg).—Ed.] As a rule, the suppurative process in these cases is not confined to a localized abscess under the diaphragm, but gradually extends upward from the appendix behind the ascending colon and over the right kidney, and eventually becomes localized between the liver and the diaphragm. It is rare for suppuration starting from the appendix to be strictly confined to the subdiaphragmatic space.

Subphrenic abscesses are not uncommonly secondary to hepatic lesions, and chiefly occur in connection with two affections of this organ —viz., hydatid cysts and cholangitis; in the former case the wall of the cyst may be intact while there is infection in the neighborhood, or the cyst may rupture; in the second case the infection may involve either the gall-bladder or the bile-ducts, and depend primarily on a calculus. Other forms of hepatic suppuration may, of course, also produce the lesion, and in septic abscesses of the liver subphrenic abscess has often been observed.

[An abscess near the surface of the liver may leak and set up an abscess between the diaphragm and the convexity of the liver; infection

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[1] W. Osler, *Principles and Practice of Medicine*, 1901, p. 601, fourth ed.
[2] G. M. Cullen, *The Scottish Med. and Surg. Jour.*, 1897, vol. i., p. 635.
[3] Bainbridge, *Med. News*, 1903, vol. lxxxii., p. 433.

may also track up between the layers of the suspensory ligament. Cantlie [1] has described abscesses in the latter situation as suprahepatic, but he believes that the abscess originates in the lymphatics in this ligament and not in the liver substance.—ED.]

The position of the kidneys readily explains why these organs are often the starting-point of subphrenic abscess. I have already pointed out the course taken by the pus in cases of perityphlitis, and how the kidneys may become involved, and the same description applies to suppuration starting in the kidneys themselves, whether caused by contusions or penetrating wounds of the lumbar region or by pyelonephritis due to tuberculosis, calculi, or suppurative ascending infection.

In exceptional cases the lesion starts from the spleen, especially in hydatid and in abscess of that organ.

Sometimes the lesion follows a non-penetrating injury of the upper part of abdomen, or, indeed, of any part of the body, such as a fall, a blow, a crush, lifting a heavy weight, jumping, etc. In such cases there is either a small amount of exudate or a minute contusion of one of the viscera in the subphrenic space, such as the liver, spleen, kidney, possibly the intestine, etc.

In rare instances subphrenic abscess is due to tuberculous osteitis of the ribs or vertebræ. In other instances it is due to rupture of an intrathoracic abscess through the diaphragm, such as an empyema or some suppurative lesion of the lungs (abscess, gangrene, bronchiectasis, tuberculous cavity, suppurating hydatid), and in exceptional cases purulent pericardial exudates. The perforation may occur in different parts of the diaphragm, and may be large or small, single or multiple.

Meltzer has made the interesting observation that the lesion may follow croupous pneumonia even without the intervention of a metapneumonic empyema. In fact, the normal state of the pleura and of its lymphatics is said to favor the passage of pus downward through the lymph-channels of the diaphragm from the lungs.

Further, some cases of subphrenic abscess have been known to follow suppuration in connection with the female genital organs, either puerperal or of obscure origin; while some cases seem to suggest that septic endocarditis—e. g., from a whitlow—may produce a subphrenic abscess as a result of embolic infarction of the spleen.

The abscess may arise in two ways—viz., the infection may either pass along the lymph-channels, or the primary abscess may perforate directly into the subphrenic space. Both methods are anatomically possible and have been known to occur; the commoner is direct perforation, especially in cases due to a perforating ulcer of the stomach, duodenum, or the large intestine. In such cases a circumscribed adhesive peritonitis first forms, just as in perityphlitic abscess; unless this occurs, diffuse peritonitis would follow the perforation; even in the latter case there may be a subphrenic pyopneumothorax, usually accompanied by other circumscribed abscesses in other parts of the peritoneal cavity.

About half the abscesses contain gas; this form of abscess usually

[1] J. Cantlie, *Brit. Med. Jour.*, 1899, vol. ii., p. 646.

results after perforation of some air-containing organ, such as the intestine or the stomach, and the condition is a form of perforative peritonitis. As the abscess frequently perforates into the lung, it was for a long time supposed that the gas was derived from this source, but careful clinical observation has shown that while this may occur, it is by no means the rule. Another possibility must be considered, namely, the spontaneous development of gas in an abscess that originally contained no gas (for an explanation of this possibility the reader should refer to previous paragraphs, but in any case it is a rare event).

[In a review of 60 cases of subphrenic abscess which were treated by operation, Grüneisen[1] supports the view that gas is not, as Leyden supposed, due to perforation of a hollow viscus, but to the activity of gas-producing organisms.—Ed.]

ANATOMY.

The anatomy of subphrenic abscess differs in no respect from that of other forms of localized peritoneal abscess; it very closely resembles perityphlitic abscess, and for a detailed description the reader should turn to the section on the Anatomy of the latter lesion. Maydl has made a special study of the topographic anatomy of this lesion.

In the first place, it is important to decide whether the abscess is intraperitoneal or extraperitoneal; in the latter case it is situated posteriorly, and is usually due to perityphlitis or to lesions of the kidney or of the ribs.

In most cases the suppurating area is only on one side of the body—*i. e.*, it is a right- or a left-sided abscess. In the intraperitoneal form the suspensory ligament of the liver forms the boundary-line; in the retroperitoneal form, the spinal column. The abscess only rarely occupies the whole subphrenic space on both sides of the middle line, since to allow this to occur, either the suspensory ligament must be destroyed or the pus must burrow behind the liver and in front of the spine. In rare instances there are two independent abscesses, one on each side of the ligament. It will be seen from all these possibilities that the size of the abscess cavity is subject to great variations.

Right-sided abscesses usually start from the liver, from perityphlitis, from the duodenum, and less commonly from the stomach; left-sided ones, as Leyden distinctly pointed out, usually depend on disease of the stomach. The abscesses starting from the kidney, the ribs, the thoracic organs, or by metastasis may, of course, be found on either side.

As already stated, the median boundary is formed by the suspensory ligament; the other boundaries vary according to the exact situation of the abscess; the diaphragm, on the one side, the liver, the spleen, the pancreas, the omentum, the stomach, the duodenum, the colon, the kidney, the anterior or posterior abdominal wall, or the thoracic wall, on the other. As a rule, there is only a single cavity which may

[1] Grüneisen, *Arch. f. klin. Chir.*, vol. lxx.

later extend downward—as, for instance, in cases due to perityphlitis. Occasionally a number of smaller abscess cavities are found in addition to a single large one; these may be close to the large one or at a considerable distance from it and in other parts of the abdomen. The genesis of these multiple abscesses may be twofold—*i. e.*, either— (1) Progressive purulofibrinous peritonitis, or (2) less commonly diffuse perforative peritonitis running a protracted course.

When the abscess contains gas, the diaphragm is forced upward as far as the third rib or second intercostal space; the heart, when the lesion is on the left side, is also displaced upward, and when the lesion is on the right side, the liver is pushed downward. In addition to gas, the abscess contains offensive sanious pus, which may be mixed with gastric or intestinal contents, such as particles of food, ascarides eggs, etc.

The abscess may perforate and discharge the pus in a number of different positions. The most common and most interesting termination is perforation upward through the diaphragm into the thorax. Leyden, on the data of the limited number of cases known to him, stated that perforation into the lung was more frequent than into the pleura; Maydl has collected 29 cases of perforation into the lung and 34 into the pleura (2 into the pericardium). It must be noted, however, that the extraperitoneal abscesses due to perityphlitis have a special tendency to discharge into the pleura. Occasionally pleurisy appears without perforation of the diaphragm.

[In 60 cases of subphrenic abscess which were treated by operation there was a pleural effusion in 40 (Grüneisen[1]).—Ed.]

It is also interesting to note that the contents of the subphrenic abscess differ from that in the pleura; the latter may be simply serous or serous and hemorrhagic, whereas the former is purulent or sanious, or the pleural contents may be simply purulent and the contents of the abscess putrid and containing gas. Perforations into the stomach, the intestine, or the gall-bladder are rare. Perforation into the general peritoneal cavity is relatively common and extremely dangerous—almost as dangerous is the development of progressive purulent fibrinous peritonitis. [A subphrenic abscess may set up adhesive pylephlebitis; I have seen this in a case due to perforation of a gastric ulcer.—Ed.]

CLINICAL FEATURES.

It is true, according to Maydl, that G. H. Barlow more than half a century ago diagnosed a gas-containing subphrenic abscess, but this observation was ignored until Leyden's publication, who, therefore, deserves the credit of having given a clear account of the clinical features of subphrenic pyopneumothorax. Formerly this lesion was confounded with true pyopneumothorax, even by such a careful observer as Wintrich. This error is due to the fact that in both diseases there is a cavity containing gas and presenting practically the same physical signs. In

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[1] Grüneisen, *Arch. f. klin. Chir.*, vol. lxx.

both there is an area on the front of the chest extending downward from the third rib, and also occupying the liver region, in which the percussion-note is loud and low, and occasionally somewhat tympanitic; the liver dulness is lower down than normal, and there are no breath-sounds over this area; there is, however, amphoric breathing or a metallic sound, the latter being especially clear on combined percussion and auscultation; pectoral fremitus is absent; Hippocratic succussion can be elicited, and when the patient changes his position, changes in the sounds can often be made out. In left-sided cases the cardiac dulness is absent from its normal position, and is found further up and to the right.

These are the signs of pneumothorax or pyopneumothorax, which in many cases is diagnosed; some peculiarities in the physical signs will, however, soon become apparent to a careful observer, and there will be some suspicious features in the history. For the differential diagnosis the following factors are of importance; these features were first enumerated by Leyden, and later corroborated by other authors, among them Scheurlen, A. Fraenkel, Fürbringer, Guttmann, Kijewski, Chelchowski, Bieganski, Herrlich, Meltzer, Maydl, Sachs, Lampe, Pasinelli, and others.

The history of the case directs attention to the abdomen; there are vomiting, pain in the stomach, and gastric hemorrhage, symptoms pointing to perityphlitis or intestinal disturbance; in other words, the general symptoms point to some disorder below the diaphragm, the actual site of which can often be made out with some degree of certainty. In addition, the signs indicative of pulmonary disease, such as cough, etc., are all absent. The patient, however, may not make any complaint of abdominal discomfort, or, if he does, it may be quite indefinite, while some unimportant pulmonary symptom, such as a cough, may lead one completely astray and direct attention to the chest instead of to the abdomen; or, again, the secondary manifestations, due to the subphrenic abscess, may appear in the pleura or the lung, and be equally misleading; or, again, an abscess situated above the diaphragm may have perforated below it; or, again, according to Meltzer, croupous pneumonia may have preceded the abscess. The history, therefore, is of use only in a general way, and the diagnosis must be based on the physical signs—*i. e.*, as stated above, on the detection of a cavity containing air and fluid in the lower half of the thorax, with all the apparent signs of pneumothorax. The question to be decided is merely whether this cavity is situated above or below the diaphragm. The following points are of use in arriving at this decision:

In subphrenic pyopneumothorax a change from amphoric or metallic to normal and vesicular sounds can be discovered on the anterior chest-wall at the level of the second, third, or fourth rib. The lung, which is compressed and pushed upward, moves well on deep inspiration, and above the uppermost zone of the cavity, where no breath-sounds could be previously detected, distinct vesicular breathing can now be heard. Nothing pathologic can be found in the lung, provided the examination

is made before any secondary changes have occurred, such as perforation of the abscess into the lung or secondary pleurisy.

In right-sided cases the liver dulness is not in its normal position, but is found lower down, where its margin can be distinctly palpated. In meteorism the liver dulness may completely disappear without any evidence of displacement of the organ. The displacement of the liver downward, moreover, is not a particularly valuable sign, as some writers believe, for it is also seen both in true pneumothorax and in pyopneumothorax. The same applies to displacement of the heart to the left in right-sided cases, although in true pneumothorax the displacement is usually greater ; in subphrenic pyopneumothorax there is probably never complete disappearance of the cardiac dulness, and, besides, the displacement of the organ is more in an upward than in a lateral direction. The statement that in subphrenic pneumothorax the intercostal spaces do not bulge, as they do in true pneumothorax, is not borne out by facts, for in both lesions the intercostal spaces may be either obliterated or bulging.

Exploratory puncture provides some other data of value in the diagnosis. Scheurlen has called attention to the possibility of aspirating two different kinds of fluid from two intercostal spaces, situated one immediately above the other. The withdrawal of pus from the lower, and of serum from the upper, space, indicates subphrenic abscess. Fürbringer and Guttmann, however, have pointed out that though this may be true in the great majority of cases, it does not necessarily hold good in all cases ; for there are rare cases of multilocular pleurisy in which some pockets contain pus and the others serum. Another sign, sometimes discovered by chance, is, when present, quite pathognomonic. It was described by Fürbringer, who found that when the needle happens to be introduced in such a way that it goes through the diaphragm, it performs very wide excursions, corresponding with the movements of the diaphragm. The needle moved upward with inspiration and downward on expiration. Chelkowski, however, states that in exploratory puncture of the pleura he has occasionally seen equally active movements, but in an opposite direction—*i. e.*, downward on inspiration and upward on expiration ; otherwise he corroborates Fürbringer's observation.

[Litten [1] observed in several cases of subphrenic abscess a well-marked, depressed, undulating curve, traveling along the side of the thorax during inspiration, and a convex prominent line moving upward during expiration. He regarded this so-called " diaphragm phenomenon" as pathognomonic of subphrenic abscess, but it has not met with general acceptance, as the diaphragm line may be present in normal individuals and absent in patients with subphrenic abscess (Elsberg [2]).—Ed.]

Pfuhl has advocated *manometric measurements* as a means of diagnosis ; a needle introduced into the abscess is connected with a manometer,

[1] Litten, *Deutsch. med. Wochenschr.*, 1892.
[2] C. A. Elsberg, *Annals of Surgery*, vol. xxxiv., p. 740.

and a rise of pressure during inspiration shows that the abscess is below the diaphragm, while a fall on inspiration and a rise on expiration prove that the fluid is in the pleura. According to Rosenbach, however, the results of this test are sometimes misleading, inasmuch as large pleural exudates may paralyze the diaphragm and thus produce the same manometric phenomena as subphrenic abscesses.

A few remarks may be made about elevation of the diaphragm to a higher level in subphrenic abscess; this is due to various causes : in the first place, to the pressure of the abscess and of the fluid and air it contains ; in the second place (Herrlich), to relaxation of the diaphragm, probably from the action of the sanious pus which is in immediate contact with its under surface ; finally (Leyden), to the pain produced by the subphrenic abscess, which probably interferes with the normal contraction of the diaphragm.

[By means of skiagraphy the position of the diaphragm and its movements can be seen, and may clearly show whether the diaphragm is depressed or pushed up.—ED.]

Although the signs are rarely all seen together in any one case, it is usually possible to localize the abscess—*i. e.*, to tell whether it is above or below the diaphragm. The other general symptoms, such as fever, pain, etc., are the same in pyopneumothorax above or below the diaphragm, and will be described in the paragraphs on subphrenic abscess containing gas.

The signs of simple subphrenic abscess are often less definite, and hence the diagnosis of this form is more difficult than that of the air-containing abscess. In the first place, as in other intraperitoneal abscesses, there is fever, continuous, intermittent, or remittent, with or without a chill. In exceptional cases, where the disease develops slowly, fever may be absent. (The reader should refer to what has been said about the temperature in the section on Perityphlitic Abscess.) There are usually loss of appetite, considerable prostration, and often rapid emaciation, all features which impress their characteristic signs upon the disease.

The patient's subjective symptoms attract attention to the upper part of the abdomen, where pain may vary in intensity and in position, being in front or behind, to the right or the left, according to the exact seat of the abscess. The pain may be strictly localized or may radiate widely over the abdomen or thorax. In addition to this varying pain there is usually tenderness on pressure, most marked, of course, over the abscess. In some cases, however, there is very little pain of either kind.

The upper part of the abdomen may or may not be prominent ; sometimes there is a little edema ; fluctuation can be obtained only when the abscess is very superficial. König and others have also called attention to the fact that we must not expect to aspirate pus over the whole extent of the soft and doughy resistance which is often felt, for much of the fluid poured out may be edema and the pus may be deep down and out of reach.

Exploratory puncture is a more certain means of diagnosis in simple abscess than in abscesses containing gas; the material obtained may be either ordinary pus or offensive sanious matter. In abscesses due to perforation and containing gas, material from the gastro-intestinal canal may also be found, and, of course, at once clears up the diagnosis.

In many cases the **differential diagnosis** between pleural exudate and subphrenic abscess must be made; and, occasionally, the question arises whether both lesions are present together. Attention has already been called to the significance of exploratory puncture in determining these points. Bieganski has endeavored to make use of various signs obtained by percussion and palpation in the diagnosis, but to my mind they are all ambiguous and consequently unreliable; the following sign is probably the most reliable of all : dulness on the anterior surface of the chest, which is more distinct in front than behind, and does not reach to the angle of the scapula posteriorly, with the upper limit dulness extending obliquely from above in front to below behind. This is only the case, however, when the abscess is so situated as to be nearer to the anterior wall. When the pus is further back, as in cases due to retroperitoneal, paranephritic, or perityphlitic lesions, the dulness is chiefly on the posterior thoracic wall, so that percussion alone gives no reliable information, and recourse must be had to other factors in order to make the diagnosis (the future course of the case, puncture, etc.). A. Fraenkel has clearly stated how difficult the diagnosis occasionally is : " When there are a moderate area of dulness on the posterior surface of the chest on the right side, high fever, a general loss of strength and of flesh, nothing of importance in the sputum, especially an absence of tubercle bacilli, and when an exploratory puncture made over the area of dulness only brings away simple—*i. e.*, serofibrinous—exudate or nothing at all, an intra-abdominal abscess should always be suspected."

A subphrenic abscess containing gas may appear quite suddenly, with considerable pain. This is easily understood from a consideration of the pathogenesis of the disease and of the fact that the lesion may be produced by gastric or intestinal perforation; the subsequent course of the disease varies in the same way as in perityphlitis. In rare cases there may be spontaneous recovery, due to rupture and discharge of the abscess, while other cases are cured by the only efficient treatment known—viz., surgical interference. In a third series the patients die, either as a result of complications (pleurisy, affections of the lungs, pericarditis) or from progressive peritonitis of a purulent type, or from rupture of the encysted abscess into the peritoneal cavity with diffuse and septic peritonitis, or, lastly, from general exhaustion with fever and emaciation. It need hardly be mentioned that subphrenic abscess in any case is a serious and a dangerous disease; it is also clear that the prognosis is better in the retroperitoneal than in intraperitoneal subphrenic abscesses. [In Elsberg's 73 cases of subphrenic abscess due to appendicitis the mortality was 40 per cent.—Ed.] (For treatment see later in connection with other forms of abscess.)

INFLAMMATION AND ABSCESS OF THE GREAT OMENTUM (Epiploitis).

As the result of operative interference in cases of perityphlitis it is now well known that the omentum is frequently found to be the seat of acute inflammation. The resulting thickening of the omentum often contributes materially to the so-called perityphlitic tumor. Further acute and chronic inflammation of the omentum is also found accompanying or secondary to other diseases, such as tuberculous and carcinomatous peritonitis. Primary epiploitis, generally acute or subacute, but occasionally chronic in its course, has been recognized only in the last twenty years. Championnière was the first to describe it; then Reynier, Morestin, and others, particularly French authors. Schnitzler, who gives the literature of the subject up to 1900, published several observations, as did Braun, and several cases have come under my notice the last few years.

Etiology.—Nearly every case follows an operation for hernia, particularly after radical cure, in which part of the omentum is ligated. In one case of Schnitzler's in which the appendix was removed during the interval, the omentum, which was adherent, was separated after ligation and sewn to the small intestine to cover a defective portion of peritoneum. Silk ligatures were used in all cases except one, in which catgut was employed.

[Fecal abscess in an appendix epiploica due to the passage of a sharp foreign body from the colon into the fatty appendix has been described by Bland Sutton [1] and is referred to again among the rare forms of circumscribed peritoneal abscess.—ED.]

Clinical symptoms of postoperative epiploitis became manifest in from a few days to six weeks after the operation—in a few cases apparently after three to six months. I must confess that the reported cases with a latent period of three to five years do not all appear clinically sound (*e. g.*, Guinard's). Still it is possible (compare a case of my own in which four years after apparently complete recovery from appendicitis there appeared grave sepsis with abscesses in the liver and emboli in the lungs, originating in old and small perityphlitic abscesses).

The infective agent is considered by most authors to be the silk ligature, but sometimes infection might take place during the operation. Perhaps the bacteria develop the more readily because they find an omentum either previously damaged or altered by disease.

Symptoms.—These make their appearance without any apparent external cause, occasionally after severe bodily exertion : they are fever, pain, vomiting, constipation, and a swelling—*i. e.*, the general symptoms of acute peritonitis. The rise of temperature begins in exceptional instances with a rigor, and is then considerable ($103°$ to $104°$ F.), but lasts only a few days. Generally the rise is gradual and does not exceed $102°$ F.; occasionally there is no fever. Vomiting is seen in cases which begin acutely at the commencement only, or lasting a few days. Pain is a constant symptom even in the absence of fever and

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[1] J. Bland Sutton, *Lancet*, 1903, vol. ii., p. 1148.

vomiting. It has the character of peritoneal pain, varies in different cases in severity and distribution, but is, as a rule, limited to the area of the swelling; occasionally it is colicky in character. Constipation is the rule; in a few cases symptoms of obstruction and feculent vomiting were observed.

The essential sign is the presence of resistance or an apparent swelling. The position and seat of the swelling vary: usually it is in the upper half of the abdominal cavity, in the right or left upper quadrant, sometimes extending from the midaxillary line to the midline of the abdomen; it may be in the umbilical region, and is very seldom limited entirely to either of the lower quadrants of the abdomen. The swelling usually extends superficially and is indefinite; sometimes it resembles a new growth and is mistaken for carcinoma. The resistance is occasionally doughy only (with edema of the skin over it), but in other cases is quite hard. The surface is smooth or lumpy. Pressure is more or less painful. As a rule, the swelling is immovably fixed to the anterior abdominal wall; when small (of the size of an orange), it has been found to give ballottement.

Sauget has distinguished three kinds of such postoperative forms of epiploitis: (1) Simple plastic without adhesions; (2) the same with adhesions; (3) purulent. Schnitzler is probably right, however, in his view that even in the apparently simple plastic cases a small amount of pus is always present in the tumor. The possibility and probability of this have been demonstrated in connection with the pathology of perityphlitic tumors.

Course.—Seldom very acute, usually insidious, occasionally chronic, lasting months or years. In many cases spontaneous and complete absorption occurs without much pus-formation and without surgical interference. Subjective symptoms cease, the tumor gradually diminishes, and eventually disappears. In other cases a larger or smaller abscess forms, which, if not operated on, bursts into the bowel or bladder. In any case adhesions may remain, giving rise to grave sequelæ, particularly intestinal obstruction. The diagnosis is easy, bearing in mind operative procedure with a lesion of the omentum (ligature, etc.), followed by the above symptoms and the appearance of a swelling or tumor.

Treatment.—See below (p. 932).

OTHER RARE FORMS OF CIRCUMSCRIBED PERITONEAL ABSCESS.

Though abscesses in the right iliac fossa may be due to various causes, the majority start from the appendix. Subphrenic abscesses have a still more varied origin, although they all have this in common, that they are situated immediately under the diaphragm and that the presence of gas gives rise to definite and characteristic physical signs. There are, in addition, a number of other possible forms of abscess in the peritoneal cavity, which, however, are so rare that they cannot be arranged in "groups." These have various anatomic starting-points,

and occur in such a number of different positions that they can be described only seriatim.

Abscesses starting from the liver and the bile-ducts are usually subphrenic. Occasionally, however, they are in other positions, especially when they originate from the gall-bladder, the common bile-duct, or the cystic duct; for they are then often in front of the liver, close to the anterior abdominal wall, or somewhat below or under the liver, lying between it and the stomach and mesentery. These pericholecystitic abscesses may contain pus or pus mixed with bile or gall-stones.

They may prove fatal by sepsis without producing sufficiently marked physical signs to allow of their diagnosis during life, or perforate into the general peritoneal cavity, causing death from diffuse peritonitis, or through the abdominal wall or into the intestine, and may then terminate in recovery, with the passage of large gall-stones. According to Naunyn's careful statistics, perforation most commonly occurs through the abdominal wall, then into the duodenum, the colon, and rarely the stomach; in one case into the jejunum and the ileum. Perforation into the urinary passages, the portal vein, and the retrocecal connective tissues is also recorded. I have already mentioned perforation upward through the diaphragm when describing subphrenic abscess. [Graham [1] collected 35 cases of bronchobiliary fistulæ; an interesting form of fistula which in some instances may be the result of a subphrenic abscess, but is more often due to an abscess in the substance of the liver communicating with the lung.—ED.]

Abscesses originating from the spleen—and these are rare—may be found either in the subphrenic space or in the left half of the abdomen, and may sometimes reach a large size. They are due to one of three causes: rarely, suppurating hydatid cyst of the spleen, more commonly suppurating infarct, and relatively most commonly various kinds of injury; I saw a case of the latter kind in an officer who had fallen from a horse.

Diseases of the stomach and the intestine other than perityphlitic and subphrenic abscesses are a recognized source of purulent circumscribed peritonitis, especially carcinoma and ulceration. Although circumscribed acute and subacute inflammation and abscess are not very common after these lesions, they are sufficiently frequent to deserve attention. The situation of the peritonitic process will, of course, vary according to the anatomic seat of the primary lesion; as the only way to illustrate the various possible combinations would be to quote all the published cases, I shall confine my remarks to the following points:

Rosenheim, Tournier, and others have described a purulent form of perigastritis (retroventricular abscess) which may start from a gastric ulcer or carcinoma. Duodenal ulcers relatively often give rise to subphrenic abscesses, occasionally hidden away at the back of the abdomen. Suppuration starting from the jejunum, the ileum, or the colon may, of course, occur in a number of different situations. Suppuration starting from the rectum (periproctitis, ulceration, trauma) rarely breaks into the peritoneum.

[1] J. E. Graham, *Trans. Assoc. Amer. Phys.*, vol. xii., p. 247.

Another lesion repeatedly mentioned by different writers has been given different names, and has not been interpreted in the same way by all observers—viz., the lesion which Windscheid calls pericolitis. He describes a disease characterized by an acute swelling on the right side of the abdomen, fever, and pain, which he considers to be a circumscribed peritonitic exudate around the ascending colon. Pal has also described 8 similar cases under the name of " primary submucous circumscribed colitis "; in most of the cases there was fever, and in all of them there was a circumscribed, painful, inflammatory swelling in the region of either the ascending or the descending colon or of the flexures of the colon ; in one case an abscess was opened and emptied, and in another the pus perforated into the left lung. Pal is inclined to regard the process not as a pericolitis and resembling perityphlitis, but as a primary circumscribed inflammation of the submucous coat of the colon— *i. e.*, a submucous infiltration due to the invasion of pathogenic microorganisms ; the affection may either terminate in resolution after absorption of the exudate or may go on to abscess or perforation.

It is impossible to enter into a description of all the cases that have been reported as belonging to this category, although many of them can be interpreted differently from a clinical standpoint ; for the present a definite decision as to the true anatomy and pathogenesis of these lesions must be deferred until more postmortem data or observations made during laparotomies on such cases are available.

[In the course of postmortem work the editor has come across cases of fibrinopurulent peritonitis around the descending colon (pericolitis sinistra), which appeared to be due to ulceration of the sacculi of the descending colon, which contained hard fecal concretions. The general clinical appearance was that of appendicitis on the left side ; the cases were in elderly persons, and the process was regarded as allied to perforation of a stercoral or distention ulcer above a malignant stricture of the colon, but as a localized instead of a general peritonitis. In a recent case there was an abscess cavity on the left side of the abdomen containing fecal material and apparently due to perforation of a stercoral ulcer in the descending colon.—Ed.]

Pal, in support of his view, refers to an observation by Eisenlohr :

The patient, a drunkard with cirrhosis of the liver, developed tenderness on pressure and spontaneous pain in the right hypochondrium, with fever, chills, meteorism, distention of the abdomen, vomiting, and diarrhea ; the swelling soon disappeared ; later there was ascites. At the autopsy, a few weeks later, a cavity about four inches in diameter was found, containing thick, pultaceous, yellowish material, and situated entirely outside the intestinal wall, below the upper portion of the ascending colon, between the hepatocolic ligament, the anterior surface of the capsule of the kidney, and the descending part of the duodenum. The position of the abscess corresponded to the upper part of the ascending colon, the wall of which was greatly thickened. No trace of inflammation nor of any inflammatory products could be found in the visceral peritoneum of the cecum or the appendix. There were, moreover, no signs of suppuration in the mucous membrane of the intestine (special attention being paid to the duodenum, ascending colon, and cecum), or of past scarring, thickening, or perforation. In a case like this no opinion can be expressed as to the pathogenesis of the paracolic abscess. Possibly it started from some superficial tear in the mucous membrane

of some neighboring part of the intestine, but there is no proof of this. It is clear, at all events, from Eisenlohr's description, that the starting-point was originally outside the intestine, and not in its wall.

This case shows, in the first place, that it may be impossible to find the starting-point, and, in the second place, that these lesions may sometimes develop in the mesentery and the retroperitoneal space. Occasionally the micro-organisms may be carried to this situation from the bowel through the lymphatics, and it is quite conceivable that the original point of entry—*i. e.*, some slight abrasion of the mucous membrane of the intestine—may have healed completely by the time the abscess developed. In other cases the lesion starts from the mesenteric and retroperitoneal lymph-glands, sometimes only from a single gland, the glandular infection being probably secondary to the intestine.

[Bland Sutton [1] has recently drawn attention to fecal abscesses in the appendices epiploicæ due to small, but sharp, foreign bodies penetrating from the colon into the substance of an appendix epiploica, and has described two cases where a fecal concretion with a foreign body for a nucleus was found in an enlarged and inflamed appendix epiploica.—Ed.]

It will be sufficient merely to draw attention to the kidneys, the urinary passages, and the female genital organs as a possible source of infection in localized intra-abdominal abscesses, and to the fact that various injuries, whether they involve the skin or not, are a not uncommon cause of circumscribed peritoneal abscesses.

The anatomic description of these abscesses is the same as of peritonitis in general and of perityphlitis in particular; one point only requires emphasis, namely, that the contents of any intra-abdominal collection of pus may sometimes, but not necessarily, have a feculent odor without free communication with the intestine.

Very little need be added, from a clinical point of view, to what has already been said about perityphlitis, the prototype of this class of lesions; the general and the local symptoms *mutatis mutandis* are the same. Almost without exception there is fever—of the continuous, remittent, or intermittent type, occasionally combined with chills. The general nutrition suffers, partly as a result of the fever, partly because less nourishment is taken, and in prolonged cases there may be great emaciation. The local signs vary greatly and may be completely absent; symptoms may also be absent, so that with the irregular fever and the atypical chills, the patient's condition is that of a septicemia with septic fever of obscure origin. Occasionally the history of the case is a guide to a correct diagnosis, or some remote sign, such as visible peristaltic movements of the bowel, as in one of the cases mentioned above, clears up the case. As a rule, however, disagreeable or painful sensations direct attention to the abdomen, and there is some area of localized tenderness to indicate the situation of the disease, such as, *e. g.*, the gall-bladder region, the lumbar region, the left iliac fossa, the left hypochondrium. Daily examination of the case may lead to the detec-

[1] J. Bland Sutton, *Lancet*, 1893, vol. ii., p. 1148.

tion of some deeply seated resistance or tumor having almost any relation to the intestine. When the inflammatory swelling is superficial, the diagnosis becomes easier, and when the skin is inflamed, the diagnosis is at once cleared up. When at all feasible, an exploratory puncture should be made in order to clinch the diagnosis. [The possible dangers of wounding intestines and of spreading infection by an exploratory puncture are considerable, and it is much safer to make an exploratory incision.—Ed.]

The starting-point of the trouble can sometimes be determined with certainty, but this is often impossible, or can be suspected only with some degree of probability. The position of the abscess and the history of the case are points of the greatest importance, while occasionally other factors are of assistance, such as the presence of bile in fluid withdrawn by aspiration, which would at once suggest a communication with the bile-ducts.

It is most important that in every case of intra-abdominal abscess in which the physical signs and clinical history do not justify a definite diagnosis, the appendix should first be thought of, next the female genital organs; and then the gall-bladder; the other possible points of origin should be considered when lesions of these organs have been excluded. (For the course and the termination of these abscesses the reader should refer to what has already been said in the section on Perityphlitic Abscess, which may be considered the prototype of all intra-abdominal abscesses.)

TREATMENT OF SUBPHRENIC AND OTHER FORMS OF ABSCESS.

The treatment must be symptomatic and tentative while the diagnosis remains uncertain, and may include cold or warm applications, inunctions, etc. When the diagnosis of abscess is once made, there is only one method of treatment—viz., to open the abscess (for the technic of this operation the reader should refer to surgical hand-books). I may point out, however, that in the light of our present knowledge simple puncture and aspiration are not sufficient, and that the abscess should be incised. The operative results are not unfavorable, and are steadily improving with the progress of surgical technic. One thing is certain— viz., that an operation is the only reliable form of treatment when the diagnosis is made.

In cases of post-operative epiploitis experience shows that, as in the case of perityphlitic tumors, a large abscess does not necessarily result, and, therefore, may not require opening. Such an inflammatory swelling may completely disappear with rest and the employment of such remedies as cold, hydropathic, or warm applications, sapo kalinus viridis, or tincture of iodin.

ACUTE CIRCUMSCRIBED NON-PURULENT PERITONITIS.

Etiology.—This condition, with a few exceptions, is always secondary to some acute process in the abdominal organs covered with

peritoneum. The peritoneum over coils of intestine which are acutely ulcerated may become involved; this is seen occasionally in typhoid ulceration when the destructive process extends down to the serous coat. In these cases, moreover, circumscribed peritonitis may subsequently become diffuse. In about 2000 cases of typhoid Liebermeister noticed severe peritonitis 16 times without perforation of the bowel. Circumscribed peritonitis also occurs in dysentery, particularly over certain areas in the ascending, descending, or transverse colon, and may subsequently become diffuse, an event which may occur in all the other acute forms of intestinal ulceration.

Acute partial inflammation of the peritoneum is also very common over the invaginated portion of the bowel in intussusception, the mesentery being especially involved, and over strangulated loops of intestine or coils of bowel which are in a state of acute axial rotation; under these conditions, again, the circumscribed inflammation of the peritoneum comparatively often passes on into diffuse peritonitis. Attention has already been called to the fact that in acute idiopathic forms of intestinal catarrh the peritoneal covering is usually unaffected. The statistics on the subject bear this out extremely clearly. In Woodward's statistics of the three years of his service in the Civil War, there were 113,801 cases of acute diarrhea, which, to judge from the symptoms, were non-ulcerative in character; of these, 1368 died. Among all the postmortem examinations made in the fatal cases (a few were not examined postmortem) there was only one case (No. 111) in which the peritoneum over the transverse and descending colon was inflamed for about six inches, but in this case the description of the state of the intestine is not very thorough or complete. In specific forms of acute enteritis, such as mercurial or phlegmonous enteritis, inflammation of the peritoneum over certain areas is not at all uncommon (see paragraphs on pericolitis and paracolitis). (For acute perityphlitis without suppuration the reader should refer to p. 874.) Non-purulent perigastritis is rare, and chiefly occurs in severe cases of acute gastritis due to corrosive poisoning.

This form of peritonitis commonly starts from the female genital organs. It is true that most cases of peritonitis starting in this situation run a chronic course (pelvic peritonitis with all its special varieties, such as perimetritis, peri-oöphoritis, perisalpingitis); occasionally, however, the course is acute. The most common causes are puerperal troubles, the inflammatory process extending from the vagina through the uterine cavity and its wall to the peritoneal covering. Infection during operations must also be considered (the introduction of dirty sounds, intrauterine pessaries, sponge-tents, etc.); occasionally a chemic irritant may be the responsible cause (intra-uterine injection of tincture of iodin or chlorid of iron). The gonococcus, in the overwhelming number of cases, produces more chronic forms of pelvic peritonitis.

Peritonitis limited to the surface of the liver is rare (perihepatitis acuta); the only condition in which acute peritonitis appears in this situation is when an acute abscess of the liver reaches the surface of

the organ. The other forms of perihepatitis all run a chronic course. The opaque, folded appearance of the liver surface in acute yellow atrophy is not due to inflammation, but is the result of the loss of liver substance and is a mechanical folding. The pain in acute hepatic congestion (as in acute infectious diseases, in tropical hepatitis) is not due to inflammation, but to stretching of the peritoneum covering the liver. While inflammation of the peritoneum near the gall-bladder (and limited to the gall-bladder region—pericholecystitis acuta) is rarely seen postmortem, clinically it may be regarded as reasonably certain that this condition is partly acute infective cholecystitis. The only morbid conditions of the spleen accompanied by acute perisplenitis are embolic infarction and some focal lesions of the spleen, not of embolic origin, sometimes found in relapsing fever (Ponfick). In the common form of splenic enlargement so often seen in infectious diseases, in which the organ is greatly swollen and the capsule tense and thinned, the peritoneum is not inflamed, though in more chronic cases perisplenitis occasionally appears.

While the fact cannot be verified by autopsy, clinical evidence, however, justifies the view that abdominal injuries may sometimes be followed by acute circumscribed peritonitis; when abdominal injuries are accompanied by a solution of continuity, this is undoubtedly true. It remains to be proved whether circumscribed non-purulent peritonitis can ever be attributed exclusively to exposure to cold. Rehn, who quotes such a case from Heyfelder, does not criticize this view, although he assumes the possibility of a rheumatic acute diffuse peritonitis.

The pathogenesis of the inflammation in all these cases is by no means clear, as special investigations with a view of explaining their etiology are often difficult to carry out, especially after death. The reader should refer, therefore, to what has been said as to the pathogenesis of peritonitis in general, but it may be added that both the " chemic " and the " bacterial " mode of origin can be assumed in the majority of the individual cases which come under observation.

Anatomy.—The seat of the inflammation is, of course, determined by the abdominal organ primarily affected. As a rule, the inflammation remains limited to the immediate neighborhood of the original focus, but it may spread and even become diffuse. Sometimes only the signs of commencing peritonitis—slight hyperemia—are seen ; in other cases, however, macroscopic and microscopic lesions of peritonitis are very marked, especially masses of fibrin. The further removed from the starting-point, the less marked are the inflammatory changes in the peritoneum. That serous effusion occurs is probable, and in some cases certain ; but the clinical recognition of the small amounts of fluid hidden between the coils of the intestine is often difficult or impossible ; lastly, in other cases, the changes seen are those of peritonitis sicca.

Absence of pus is characteristic of this form of peritonitis. It is only in exceptional cases—namely, when the process becomes diffuse— that suppuration occurs. In other cases, again, gangrenous changes rapidly occur near the starting-point of the inflammation, and perforation

and perforative peritonitis, of course, follow. In general, however, the local effects of the acute peritoneal inflammation are good, inasmuch as a protective barrier is formed which prevents the further spread of the peritonitis and also lessens the dangers of perforation. On the other hand, of course, local adhesions may lead to a variety of troublesome sequelæ, which will be dealt with in the next section.

Clinical Features.—The differences in the etiology naturally determine differences in the clinical course of this affection. The signs of circumscribed peritonitis may remain entirely latent when they appear in some disease that runs a very severe course, such as toxic gastritis, dysentery, or typhoid fever ; conversely, it may be the first sign of some latent primary affection, as in perimetritis, perityphlitis, etc.

The most important and often the only evidence of acute circumscribed non-purulent peritonitis is the pain ; its intensity may vary, but in general it is exactly the same as so-called peritoneal pain ; it is localized, or at least most intense in one spot, is made worse by pressure, and is continuous after it has once attained a certain degree of severity. It is, of course, impossible to describe all the possible positions of the pain, and the following remarks will be sufficient in this connection : In typhoid fever, as is well known, pressure, even though considerable, over the iliac region is not painful ; marked pain on pressure points to local peritonitis with fair certainty. The same applies to dysentery. In acute perimetritis the uterus is very painful on pressure. Occasionally, it is true, local tenderness on pressure is extremely slight, and the most important guide to the diagnosis is wanting ; on the other hand, if the primary disease is very painful, the existence of circumscribed acute non-suppurative peritonitis may be masked, because the peritonitic pain is obscured by that of the primary lesion ; this is illustrated by toxic gastritis. Sometimes in acute local peritonitis the spontaneous pain is at first widely spread and only becomes localized later ; but even in these circumstances the original starting-point is usually tender on pressure from the outset.

Peritoneal friction-sounds are very uncommon in this form, and if present at all, are most likely to be heard over the liver and the spleen. When audible, however, friction-sounds are an absolutely reliable sign of the disease. General symptoms, such as fever, chills, vomiting, hiccup, meteorism, which are characteristic of general suppurative peritonitis, are absent or appear only when the inflammation of the peritoneum becomes diffuse.

The course largely depends on the nature of the primary disease. When the primary affection is cured, the local peritonitis may also disappear without leaving any trace. Very commonly, however, some opacity or thickening of the peritoneum, or in some cases adhesions with neighboring organs and the abdominal wall, is left behind ; these adhesions, of course, in their turn may subsequently lead to a number of sequelæ ; or, again, there may be acute gangrene at the starting-point, and perforation.

There is no special treatment in the majority of cases. The primary

disease completely dominates the clinical picture, and the treatment of the primary affection is usually *per se* directed against the peritoneal complication ; the special treatment of the secondary circumscribed peritonitis is practically limited to the use of cold or hot applications for the relief of pain, the choice of hot or of cold applications depending on the individual inclinations and the reaction of the patient ; sometimes the application of chloroform or menthol ointment exerts a certain psychic effect, and the patient feels that something is being done for the relief of the pain. The administration of opium may sometimes be important. In acute circumscribed peritonitis, without damage to the skin, due to some external injury, the application of leeches in addition to cold may be useful.

CHRONIC PERITONITIS.

Chronic peritonitis is that form of the disease which has an insidious onset and runs a chronic and protracted course. Peritonitis which has an acute onset and later becomes chronic was considered under the heading of Acute Peritonitis and is usually distinguished from the genuine chronic form. For clinical requirements, rather than from a scientific standpoint, the following classification may be recognized : (*a*) Chronic exudative ; (*b*) chronic indurative and adhesive ; (*c*) tuberculous ; (*d*) carcinomatous peritonitis. The last-named form will be described in a special section on the New Growths of the Peritoneum.

CHRONIC EXUDATIVE PERITONITIS (Peritonitis Chronica Exsudativa).

ETIOLOGY.

Opinion as to the causation of this form is still dominated by the view, originally put forward by Louis, that chronic exudative peritonitis is always tuberculous, and even at the present time there is no consensus of opinion. My own standpoint, based on reading and on personal experience, is the following : the great majority of the chronic forms of peritonitis with a gradual onset and a slow course are really tuberculous ; but, in addition, there are undoubtedly other, though rare, cases which lead to the formation of a fluid exudate, have an insidious onset and a slow course, but are neither tuberculous nor carcinomatous.

In the cases of this kind the question has always been raised whether there is a so-called idiopathic chronic peritonitis in which the peritoneum is primarily involved, while there are no other anatomic lesions anywhere else in the body. The objections that have been raised against this view have been greatly strengthened by the fact that the diagnosis of the clinical cases adduced as arguments was rarely confirmed by autopsies ; many of the observations, in fact, could be criticized *intra vitam* and definitely characterized as " not idiopathic." (For the detailed discussion of this subject the reader should refer to p. 743.)

The question as to the existence of an idiopathic form of chronic

exudative peritonitis, however, merely transfers the inquiry from the clinical to the etiologic standpoint; for even if there is not an idiopathic form in the strict sense defined above, the existence of a form of peritonitis that begins gradually, runs a chronic course, and is neither tuberculous nor carcinomatous, is not by any means excluded; for anatomic and clinical observations prove that this does occur; the problem as to the nature of the causes underlying this form of peritonitis is an obscure one.

One definite cause is trauma—*i. e.*, injuries directly involving the abdomen. A number of such cases are on record. The following case reported by Henoch may be quoted : A child, maltreated by its father, developed enormous ascites, followed by the formation of adhesions ; at the autopsy there was no trace of tuberculosis.

It is difficult to decide whether the form of peritonitis sometimes seen in nephritis should be classified anatomically as a chronic exudative or as a dry, indurative form of the disease, for, as a rule, nephritis alone produces ascites. The same applies to the chronic peritonitis often seen in the course of portal engorgement (in cirrhosis of the liver) and in venous stasis in the region of the inferior vena cava.

From an unbiased consideration of the question it is impossible to deny the occasional occurrence of cases of peritonitis which begin slowly, run an insidious course, and cannot be classified under any of the headings just described—in which, in fact, it would be straining a point to attempt to include them under the heading of tuberculous peritonitis. In such cases it must be assumed that bacteria or bacterial poisons gain an entrance into the peritoneum in such an attenuated form that they produce extremely chronic forms of inflammation. It must be left to future research to determine the point of entrance and the character of the poison in these cases; this task, however, is difficult because most of these cases recover. Particular attention must be paid to the female genital organs, because, as experience shows, this form of peritonitis is very common, especially in young women.

There are certain cases which Borchgrevink, among others, has reported. The patients, ranging in age from sixteen to thirty-one, previously healthy, with no hereditary taint and no evidence of tuberculosis in other organs, present all the clinical symptoms of an exudative peritonitis. From the commencement of the symptoms to the disappearance of the exudation the illness lasts from five weeks to four months. Under observation for a further period of one and a half to two and a half years they remain perfectly healthy and follow their ordinary occupation. According to the general view, they are cases of "simple" peritonitis, and this view is probably correct. Since, however, Borchgrevink was able to prove, through the infectivity of the peritoneal exudation, the true tuberculous nature of the disease, one is compelled to fall back once more upon the old view taken by Louis. Probably, then, a simple chronic exudative peritonitis is conceivable, but it remains to be proved that it actually exists.

[Some cases of chronic exudative peritonitis are allied to, and an

extension of, the usually more local change involving the capsule of the liver (*vide* "iced liver," p. 942). In some cases there is a chronic inflammation of several serous membranes—the peritoneum, pleuræ, pericardium—a condition called "polyorromenitis, Concato's disease, polyserositis," etc. Kelly [1] has collected 39 cases of this kind. Though the tendency of modern writers is to consider the change tuberculous, even in the absence of clear evidence of tuberculosis, some cases cannot be explained in this way.—ED.]

ANATOMY.

There is nothing characteristic about the pathologic anatomy of this form. The fluid exudate is the same as in the other forms; occasionally there are tough masses of fibrinous exudate, but at other times these are absent. The fluid may be either freely movable or encysted. The peritoneum itself, when the disease is of long duration, may be thickened, shiny, smooth, and white; sometimes it is covered with small hard nodules of connective tissue, which, however, do not show the histologic characteristics of tubercle (peritonitis fibrosa). Other anatomic changes are either due to the primary affection (nephritis, alcoholism) or are of such a character that they at once show that the peritonitis does not belong to the simple chronic exudative type.

CLINICAL FEATURES.

The physical signs of chronic exudative peritonitis are easily enumerated, but it is very difficult to give an accurate clinical picture of the disease. This is due to the fact that a number of the cases on which some of the most classic descriptions of this disease (*e. g.*, Galvagni, Vierordt) were based do not really belong to this category. A few critical remarks on this subject have already been made, and the subject cannot be further discussed for fear of transgressing the limits of this hand-book. The following is an attempt to describe the clinical picture of this disease.

The disease occasionally comes on after some definite injury, exposure to cold, or after some other probable external cause; more commonly, however, there is no assignable cause, no definite onset, and the course of the disease is very slow and insidious. The patient's attention is attracted to the abdomen by two points: pain or progressive enlargement or by both together.

The pain may be spontaneous or may appear only on pressure; it is rarely violent, and, as a rule, is moderate or quite insignificant, and in some cases is entirely absent. Occasionally some parts of the abdomen are more painful than others, but, as a general rule, the whole abdomen is uniformly affected.

The fluid exudate, which produces the common physical signs, gradually increases and may be very considerable before it attracts the patient's notice. The amount of fluid varies: it may be large, *i. e.*,

[1] A. O. J. Kelly, *Amer. Jour. Med. Sci.*, 1903, vol. cxxv., p. 115.

several liters—and, as a rule, is freely movable; it may, however, be encysted by fibrinous adhesions. The physical and chemic properties of the fluid are the same as those of any ordinary inflammatory exudate. In cases where ascites persists for a long time edema of the lower extremities and dilated abdominal veins may appear.

In some cases, particularly in the traumatic forms, large masses of fibrin may form; or when the fluid portion of the exudate has disappeared, thick masses of connective tissue may develop.

In some of the cases there is a rise of temperature, rarely exceeding 101° to 102° F.; in many cases, however, the disease runs its course without any fever whatever.

This exhausts all the phenomena directly dependent on the peritonitis. Symptoms due to disorders of the organs of digestion are quite inconstant and variable in character; the appetite may be good or there may be anorexia; there may be diarrhea or constipation, meteorism or the normal amount of gas, in the intestine. Vomiting and hiccup are, as a rule, absent. The severe symptoms of peritonitis are also absent —*i. e.*, changes in the circulation, action of the heart, the urine, the general constitutional condition, septic and toxemic symptoms. There may be a little mechanical interference with respiration due to pressure on the diaphragm from below, in cases with a large fluid exudate. Disturbances of the general nutrition and some degree of anemia are often seen in very chronic cases, but may be completely absent.

The termination of the disease in the majority of cases is by recovery, either spontaneously or with the aid of artificial means. The exudate disappears slowly, in the course of weeks, or often more rapidly, and finally complete recovery occurs. Sometimes adhesions remain after the fluid is absorbed, or there may even be wide-spread cicatricial indurative adhesions, which, in their turn, may produce the disturbances described in the next section.

Occasionally, as stated by some writers, there are remissions and exacerbations resembling those in tuberculous peritonitis. Of this I have no personal experience, and merely quote this statement for what it is worth, without attempting to decide whether the cases in point were in reality tuberculous or not.

Death from uncomplicated chronic exudative peritonitis seems to be rare, and it is a question whether death ever ensues in this disease in the absence of those secondary disorders produced by the formation of cicatrices and adhesions in the abdomen.

TREATMENT.

A number of the published cases of this disease recovered spontaneously without external aid. As a rule, it is advisable, and if there is much pain, it is necessary to keep the patients in bed and to see that they are properly fed. If there is much anemia, iron and arsenic are indicated. In very chronic cases a change of residence and of climate is advantageous, the change being made according to the general principles governing the choice of a climate or residence.

Local applications to the abdomen are recommended by many writers. While it is difficult to prove to what extent they can do good, it cannot be denied that they are occasionally useful. The chief applications used are Priessnitz compresses and poultices. Compresses that slightly irritate the skin (salt water or one of the artificial or natural mud-baths) are useful. With the same object the application of sapo kalinus viridis may be recommended. I am not, however, in favor of the use of gray ointment, for its so-called absorptive action is too uncertain and its effect on diuresis (H. Vierordt) too slight to justify its use, while the fact that it may have bad effects must be borne in mind. When it is necessary to irritate the skin, tincture of iodin (with tinctura gallarum āā) is better than blistering, which is not necessary. Diuretics, diaphoretics, and cathartics are useless. Ascites should be tapped when it becomes so marked that it does harm mechanically.

CHRONIC INDURATIVE AND ADHESIVE PERITONITIS.

One form of chronic peritonitis deserves separate description because it is of the greatest clinical importance—viz., that in which the anatomic changes are essentially or exclusively indurations and adhesions of inflammatory origin. It is important to emphasize the fact that the tuberculous or carcinomatous forms of adhesive peritonitis are not included in this description.

This form of chronic peritonitis may be diffuse and involve a large part of the abdomen, or it may be localized and limited to circumscribed areas. The diffuse form, unless due to tuberculosis or carcinoma, is rare. The partial form is common and of great clinical interest. Virchow, who specially investigated, and after Rokitansky was the first to call attention to the disease, says: "I consider this partial form of chronic peritonitis one of the most important morbid conditions, and cannot recommend it sufficiently to the attention of medical men." Since Virchow's exhortation much attention has, in fact, been given to the disease, and its importance, particularly as bearing on displacements and interference with the permeability of the intestinal tract, has been recognized by all clinicians; and Treves, and more recently Riedel, have made many valuable contributions to our knowledge of the subject. In the last decade medical men have recognized that this form of peritonitis may be the cause of a great number of internal disturbances, and this knowledge is largely due to the surgeons, who, from the increased frequency of laparotomy in recent times, have gained an insight into the conditions existing during life.

ETIOLOGY.

Partial Indurative and Adhesive Peritonitis.—All the three factors concerned in the causation of peritonitis in general may be active in this form—viz., bacterial, chemic, and mechanical irritants. By careful study of the general pathogenesis of peritonitis it is possible to determine approximately which of these three factors is at work in

any given case. On these points the reader should refer to previous sections.

I shall confine this description to the clinical causes of the affection, some of which can be recognized without difficulty during life, while others become apparent only at a postmortem examination.

Localized indurations and adhesions may be the results of a past attack of acute or subacute peritonitis. Thus, after absorption of the serous exudate the only remains of the acute or subacute peritonitis are a few adhesions or scattered cicatricial thickenings of the peritoneum. When a purulent exudate has been operated upon, recovery, as we know, cannot take place without the formation of more or less adhesions ; in encysted purulent peritonitis there are adhesions from the outset, while adhesions often follow non-suppurative circumscribed acute peritonitis. In these cases the etiology of chronic peritonitis is clear. The most interesting form of chronic peritonitis is that with a gradual onset, either unmarked by any definite manifestations or masked by the symptoms of some other disease, and with a slow insidious course which is chronic from the outset. The following are the main clinical features which may present themselves : The most important, because the most frequent, form of chronic peritonitis is that starting from the female genital organs ; this form is so common that, according to Winckel, to quote only one of the more recent writers, nearly 80 per cent. of all women, whether old or young, show evidence of pelvic peritonitis when examined after death. For these bad effects sexual intercourse and labor are chiefly responsible. Gonorrhea is a most important cause, the gonococcus passing through the wall of the cervix to the perimetrium, or more commonly entering the tubes and there producing pyosalpinx and perisalpingitis. It is also stated that menstruation *per se* may give rise to peri-oöphoritis. (Compare with this Gersuny's views (see later) as to the etiologic importance of hemorrhages from the female genital organs.) In addition, neoplasms and numerous varieties of chronic inflammation of the genital organs in which the irritation is transmitted to the peritoneum may cause this form of chronic peritonitis.

The intestine is etiologically second in importance only to the genital organs.

The chronic form of peritonitis around the appendix is well known. In old hernias (inguinal and femoral) the serosa may also become inflamed, but this form of the disease is not very generally recognized. Fecal accumulation may set up quite a characteristic form of localized peritonitis (and mesenteritis—see below) without producing any simultaneous changes in the mucous membrane of the intestine. Since this form is most commonly found in the region of the hepatic and splenic flexures, although, of course, it also occurs in other situations, Virchow spoke of it as " partial hypochondriac peritonitis." Pressure is the chief causal factor both in this and in localized hernial peritonitis. Foreign bodies, such as gall-stones or fecal concretions, act in a similar manner. Changes in the peritoneum usually accompany ulceration of the mucous membrane of the intestine and malig-

nant disease in any part of the intestine. In chronic intussusception the two surfaces of the peritoneum that are in contact usually become adherent to each other; but in this lesion of the bowel all evidence of peritonitis may be absent, even though the disease may persist for months; the reasons for these differences in individual cases are not clear. In stricture of the intestine the peritoneum of the dilated portion of the intestine immediately above the constriction is often inflamed and thickened; this is presumably due to the decubital ulcers so often found in this portion of the intestine; Rokitansky has shown, however, that it may occur without decubital ulceration. Simple chronic catarrh of the bowel without any local complications does not produce localized adhesive peritonitis. Ulceration or carcinoma of the stomach may, like the same conditions in the intestine, lead to localized inflammation of the peritoneum. This may happen in some other rare gastric lesions, as, *e. g.*, in diffuse hypertrophic cirrhosis.

[Calwell[1] finds that perigastric adhesions are present in 40 per cent. of cases of gastric ulcer and in 2 per cent. of all bodies.—ED.]

The liver and the gall-bladder, particularly the latter, may also be the starting-point of chronic adhesive peritonitis. Surgery of the gall-bladder as practised in the last few years has shown the great importance of adhesive pericholecystitis, the existence of which was, of course, already known to pathologists.

Peritonitis around the gall-bladder may lead to the formation of adhesions between the gall-bladder and neighboring organs, such as the omentum, stomach, duodenum, colon, and the abdominal wall. When this occurs, there is usually cholelithiasis, but—and this is important—it may also occur in the absence of gall-stones—*i. e.*, in simple chronic cholecystitis. Chronic perihepatitis, which often complicates a large number of liver diseases, such as cirrhosis, syphilitic hepatitis, malignant disease, hydatid cysts, is not of the same clinical importance. In "corset liver" a form of peritonitis is seen in the furrow on the surface of the liver, and is due to pressure. Peritonitis from pressure is usually found on the anterior surface of the liver, but it may occasionally be seen at the blunt margin of the left lobe in cases of great enlargement of the heart. The partial form of peritonitis, which in rare cases develops around the portal vein and constricts this vessel, is due to duodenal ulcer, to changes in one of the glands in the portal fissure, or perhaps from injury (Frerichs). Attention must be drawn to the adhesions seen in right-sided pleurisy, which are caused by extension of the inflammation through the diaphragm, so that the peritoneum covering the opposed surfaces of the diaphragm and the liver become affected and adherent.

Curschmann has described a peculiar form of chronic hyperplastic perihepatitis under the name of "Zuckergussleber," which occupies a special position. In this affection the serous covering of the liver is homogeneous, milky white, and looks like icing; it consists of firm cicatricial tissue which may be from 5 to 10 or even from 12 to 14 mm.

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[1] Calwell, *Brit. Med. Jour.*, 1899, vol. ii., p. 1185.

thick (Weckerling-Rumpf); the liver substance is normal in so far that hyperplasia of the periportal connective tissue and induration from obstruction are both absent; in a few cases only abnormal growth of Glisson's capsule was continued from the surface into the perilobular tissue. This peculiar change has been found once only as an isolated disease of the peritoneum; in this case there was a large abscess cavity filled with stinking pus, and situated between the umbilicus, the greater curvature of the stomach, and the omentum; the pleuræ and pericardium were normal. In all similar cases other serous membranes were attacked; the pericardium always, generally in the form of obliteration by dense adhesions; the right pleura is always affected, being either obliterated by adhesions or showing pleurisy with exudation; the left pleura is often involved; in a certain number of cases the serous covering of the spleen, and in a few instances the general peritoneum as a whole, are affected. The most constant clinical symptom in all cases was ascites.

[This association of several serous membranes has been spoken of as polyorromenitis [1] or Concato's disease in Italy, polyserositis (Kelly [2]), and multiple progressive hyaloserositis (Nicholls [3]).—Ed.]

The pathogenesis of this process has led to several different views. F. Pick calls it "pericarditic pseudocirrhosis of the liver," and is of the opinion that a latent pericarditis causes a disturbance of the circulation in the liver and so leads to connective-tissue hyperplasia. This brings about grave ascites from obstruction to the portal circulation. Hübler strongly opposes this conception of the "Zuckergussleber," and Pick's own observations point rather to a real cirrhotic condition of the liver than to "Zuckergussleber," and indicate the usual known sequelæ of total synechiæ of the pericardium. Siegert's explanation of "Zuckergussleber" appears noteworthy. He thinks we have to do with chronic inflammation with exudation of the capsule of the liver appearing primarily or secondarily (spreading from the pericardium and right pleura). A satisfactory etiologic explanation has not yet been arrived at; quite distinct factors have been adduced.

[Hale White [4] described this condition as general or universal chronic perihepatitis, and regarded it as practically always associated with *general* chronic peritonitis and a sequel of chronic interstitial nephritis. Nicholls [5] and Kelly [6] have recently written elaborate monographs on "Zuckergussleber" or "iced liver," and its association with similar chronic inflammations of the pleura, pericardium, and peritoneum. Nicholls tabulates 14 cases of "Zuckergussleber," among which only 2 of Hale White's 22 cases of general chronic perihepatitis are included. Though the chronic hyperplastic overgrowth of connective tissue, together with hyaline metamorphosis, may be almost confined to the

[1] F. Taylor, *Brit. Med. Jour.*, 1900, vol. ii., p. 1693.
[2] A. O. J. Kelly, *Amer. Jour. Med. Sci.*, 1903, vol. cxxv., p. 115.
[3] A. G. Nicholls, *Studies from the Royal Victoria Hosp.*, Montreal, 1902, vol. i., No. 3.
[4] W. Hale White, *Trans. Clin. Soc.*, vol. xxi., p. 219; *Allbutt's System of Medicine*, vol. iv., p. 118.
[5] A. G. Nicholls, *Studies from the Royal Victoria Hosp.*, Montreal, 1902, vol. i., No. 3.
[6] A. O. J. Kelly, *Amer. Jour. Med. Sci.*, 1903, vol. cxxv., p. 115.

liver, it is usually part of a generalized lesion affecting in turn one serous membrane after another. He believes that the process is definitely inflammatory, and is probably due to infection with micro-organisms of a somewhat low grade of virulence, but capable of a sclerogenous effect. The micro-organisms likely to produce this effect are the Bacilli tuberculosis, typhosus, and coli. The parts especially affected by this chronic hyaline serositis—viz., the upper surfaces of the liver and spleen, and the bases of the pleuræ—are in a constant state of unrest, and thus when they are once inflamed, irritation is kept up. In 11 out of his 14 cases there had been an acute inflammatory attack of some kind, such as hepatitis and pericarditis. The progressive lesion may commence in the peritoneum (most commonly), pericardium, or pleura. He does not consider that granular kidney plays an important part. Kelly has specially considered the relation of chronic obliterative pericarditis with the "iced liver," and Pick's pericarditis pseudocirrhosis, and, contrary to Pick's view, believes that the liver has little to do with the production of ascites, since this can be explained in most cases by concomitant chronic peritonitis or perihepatitis. He groups together under the heading of multiple serositis Pick's pericarditic pseudocirrhosis and the "iced liver," and states that although some distinction—anatomically at least—may be drawn between cases of serositis confined to the peritoneum and cases of multiple serositis, the two groups are clinically much alike, and an "iced liver" may occur in both.—Ed.]

Of symptoms, one alone is constant—viz., ascites developing early and often grave. Other symptoms change largely according to the part played by other serous membranes and the primary or secondary affection of the liver. The latter is, to commence with, often enlarged, toward the end diminished in size and width, and tough in consistence.

[According to Nicholls, there are two clinical types— (1) primary perihepatitis, in which ascites is the leading feature; after a time the disease spreads to the right pleura and pericardium; the liver and spleen are usually enlarged. (2) Primary pericarditis, in which the earliest symptoms are those of adherent pericardium or indurative mediastino-pericarditis; the process then spreads to the right pleura and eventually to the capsule of the liver. In both forms jaundice and gastro-intestinal hemorrhages are absent, the urine is diminished in amount and rarely albuminous, and the ascitic fluid is rich in albumin (3 per cent.), so as to suggest an inflammatory exudate rather than a passive transudation. The cases last from two to sixteen years, and usually die from some acute infection, such as pneumonia.—Ed.]

The spleen often shows partial peritonitis, which may be completely circumscribed and limited to the area of the spleen affected by some morbid process, such as an old splenic infarct or a nodule of growth, but more commonly affects the peritoneum of the whole organ, as in the chronic splenic enlargements of leukemia, pseudoleukemia, malaria, and some of the acute infectious diseases. A rare form of isolated calcified perisplenitis is described on page 762.

Sometimes affections of the pancreas, the bladder, and the kidneys produce partial chronic peritonitis.

Local inflammation of the peritoneum may develop around tumors and new growths in contact with the peritoneum.

Lastly, partial peritonitis may develop from glands, especially mesenteric glands (peritonitis chronica mesenterialis), and may lead to serious clinical consequences. This rare and special form of the disease can, of course, be discovered only at an autopsy. The primary glandular affection is usually due to infection conveyed from the intestine.

Special mention must finally be made of chronic mesenteritis of the sigmoid flexure and the contraction of the mesentery of the cecum and the lower end of the ileum; this form of the disease has recently been described by Riedel. A radiating thickening of the mesosigmoid is common and well recognized, and when well marked, is the most common cause of volvulus of the sigmoid flexure. The primary cause of this lesion is fecal accumulation. Riedel met with this form of chronic mesenteritis several times during abdominal operations on the cecum, the lower part of the ileum, and the ascending colon; here, too, the development of the process must be attributed to fecal accumulation. Lately, Riedel has made further observations to the effect that probably the contraction of the mesentery of the sigmoid and cecum occurs in most cases primarily in the mesentery itself. In favor of this, and against the assumption that fecal accumulation is the exciting cause, is the fact that the formation of cicatricial tissue is always greatest at the root of the sigmoid mesocolon, while toward the periphery—*i. e.*, close to the intestine—they always become fewer in number and are seldom found in the bowel itself.

External trauma, especially long-continued or frequently repeated pressure, may also produce partial chronic peritonitis in various parts of the abdominal cavity. The clinical history of many of the cases, together with the anatomic findings, makes this interpretation almost irresistible; these cases are analogous to partial peritonitis following internal pressure. Virchow is even inclined to attribute the majority of the connective-tissue proliferations that are found in the mesentery (chronic omental peritonitis) to external pressure, and it has been proved over and over again that severe blows on the abdomen (*e. g.*, a kick from a horse, a fall, etc.) may give rise to adhesions and clinical symptoms (colic, intestinal obstruction, etc.), and that these symptoms may make their appearance a long while after the injury.

Gersuny has directed attention to another etiologic possibility. He thinks that in women, more often then is usually thought, hemorrhages may take place into the peritoneal cavity (in connection with expulsion of ova from the ovaries, in menstruation from the tubes, etc.). When the fibrin remains in situations which are out of the reach of the active peristalsis of the small intestine, the formation of connective tissue and adhesive pseudomembranous peritonitis result. This explains, or is one of the explanations, why adhesions so seldom form over the small intestine, but are common on the lateral aspect of the descending colon,

on the lateral and lower aspects of the cecum after acute appendicitis, and the posterior surface of the retroverted uterus. As an illustration of this view of Gersuny's, and at the same time of the rapid development of adhesions, the following case of Moser's may be quoted : Laparotomy was performed on October 31 on a case of typhoid fever for suspected perforation. This was absent, the coils of intestine were all free, and there were no adhesions. At the postmortem (death occurred on November 4) there were found, in Douglas' pouch, quite scanty collections of clotted blood ; there were adhesions over the whole of the intestines, for the greater portion over the ascending and transverse colon ; over cecum and vermiform appendix the adhesions were so strong that these organs could only be dissected out with scissors. Five days previously there had been no sign of adhesions.

The formation of adhesions after abdominal operations is of the greatest importance, particularly now when laparotomy is so frequently undertaken. These adhesions often help in bringing about the object for which the operation was undertaken, and may, indeed, be a necessary part of the curative process. In other cases, however, they form a troublesome sequel of the operation, and numerous attempts have, therefore, been made, both experimentally and by practical surgeons, to prevent the formation of post-operative adhesions.

It is doubtful whether there is a "rheumatic" form of adhesive peritonitis ; at all events it has not yet been proved to exist.

In some of the cases of the affection it is quite impossible to determine the etiology.

Diffuse Adhesive and Indurative Peritonitis.—In tuberculosis and carcinomatosis adhesions are often widely spread in the peritoneal cavity, and may be so extensive as to lead to complete obliteration of its cavity. In other forms of peritonitis this diffuse adhesive change is rare, but it may be found in tumors of the peritoneum and in traumatic peritonitis. This form of the disease may originate in two ways : in the first place, there may be diffuse dry peritonitis, either after the absorption of a fluid exudate or without any fluid exudate whatever ; or, in the second place, the process may be dry and at first circumscribed, and by degrees extend and involve large portions of the peritoneum.

Syphilis is a rare, but very interesting, etiologic factor in the production of this form of peritonitis ; in adults this affection is so rare that it is doubtful whether it ever occurs without general syphilitic disease of the abdominal viscera ; in one case of Lancereaux's, which has been widely quoted, the whole anterior abdominal wall was adherent to coils of the intestine which were adherent to each other, so that the whole formed a tough white mass ; but in this case nearly all the abdominal organs, particularly the liver, showed syphilitic changes. In a case reported by Krapezky in a man of sixty-one years there was " peritonitis et mesenteritis multiplex circumscripta fibrosa," which finally proved fatal from intestinal obstruction ; but the author's assumption that this was a case of syphilitic peritonitis seems to me very doubtful. It is true

that from his description the starting-point of the peritonitis could not be determined, but to my mind the following fact alone militates against the theory of a syphilitic origin—viz., that the patient was at the time suffering from secondary syphilitic lesions (condyloma of the palate, roseola of the skin), and that the infection dated only from the month of September, so that the patient died within eight weeks after the infection. Observations of this kind certainly lend little support to the hypothesis of a purely syphilitic peritonitis in adults.

In the fetus this form is decidedly more common ; Simpson was the first to show that hereditary lues may be a cause of peritonitis in the fetus, which may either be circumscribed or diffused. Usually the peritonitis is of the dry adhesive form, although occasionally there may be some serous or purulent exudation. This disease, as I learn from the literature, never develops during extra-uterine life, but always during the latter months of fetal life, and almost always causes the death of the fetus in utero.

It is hard to estimate the rôle that alcoholism and renal disease play in the genesis of the disease. It is certain that these factors may cause chronic peritonitis, but it is not always possible to determine the form of the disease, for, in addition to the fibrous adhesions there is almost invariably some fluid exudate produced by the primary disease in nephritis and by cirrhosis of the liver in alcoholism. For the same reason it is difficult to form a definite opinion about cases, such as the one described by Friedreich (p. 753), and those examples of peritonitis which come on in the course of long-standing engorgement in the area of the portal vein or of the inferior vena cava where no puncture of the abdomen is ever performed.

ANATOMY.

The morbid anatomy of both the diffuse and the circumscribed forms of peritonitis have been dealt with on pp. 750 to 764, to which the reader should refer for the details. The following special points will be mentioned here :

Whereas partial peritonitis is occasionally present as an anatomic condition without causing any clinical manifestations, particularly in limited areas on the surface of flat organs (the liver, the spleen), they may, under other conditions, be of great importance, inasmuch as they secondarily lead to the development of important anatomic changes. In the first place, they produce approximation, adhesion, and displacement of certain organs. It would only be a waste of space to enumerate all the possibilities that have been observed or might conceivably occur. As this work is specially concerned with the diseases of the intestine, I will briefly recapitulate what has already been said in previous sections.

Chronic peritonitis may lead to the formation of fissures and holes, or of bands and cords connecting the various abdominal organs, the intestine, and the abdominal wall. The adhesions may be annular, and may constrict the intestine or tether it to some other organ, or to

some other coil of intestine, or produce kinking and bending of the intestine.

Chronic omental peritonitis causes the omentum to become adherent " either to hernial sacs, to the upper or lower part of the abdominal wall, to the pelvic organs—*i. e.*, to the ovaries and tubes—or to the surface of the intestine. It may be displaced backward by the intestine, and become adherent to the surface of the liver, stomach, or spleen ; or it may become rolled up to form a round cord which runs transversely across the abdomen at the level of the transverse colon, and runs across the abdomen to any one of a number of various positions on the opposite side " (Virchow).

Chronic mesenteritis leads to shortening of the bowel, and hence to serious anomalies of position and interference with the permeability of the intestine. Riedel described white, cicatricial plaques in the mesentery of the lower part of the ileum, the cecum, the ascending colon, splenic flexure, and sigmoid, which were constricted and bent ; or " the mesentery of the sigmoid flexure was so drawn together by radiating strands of adhesive tissue in its substance that the intestine formed an acute angle and the two branches of the flexure were closely approximated like the two barrels of a gun." The peritoneum covering the intestine itself may be perfectly normal in such cases, and the changes be strictly limited to the mesentery. Further, Riedel describes as his experience in several cases a primary peritonitis præirenalis retrahens, which may form glistening triangular cicatrices and displace the kidney.

Gersuny has repeatedly observed a peculiar form of adhesion— " flexure adhesion "—which he regards as typically peritoneal. It consists of a band-like pseudomembrane which runs from the point of junction of the descending colon and the sigmoid flexure transversely over the outer part of the mesocolon, and has about the same point of attachment as the mesocolon itself : one end is attached to the bowel, where the lateral portion of the mesocolon commences ; the other end to the parietal peritoneum, close to the origin of the mesocolon.

CLINICAL FEATURES.

Chronic adhesive peritonitis does not always produce symptoms ; in some cases the lesion is a harmless anatomic condition, and it may even be beneficial and a means by which the organism protects itself against danger ; for example, when adhesions form around an abscess or an ulcer in any part of the abdomen ; in this way extension of the morbid processes is interfered with, and more especially rupture into the free peritoneal cavity is guarded against. In fact, surgeons formerly purposely produced these adhesions in the course of some operations (*e. g.*, in opening an abscess of the liver or hydatid cyst).

On the other hand, adhesions may produce dangerous complications. Formerly the great clinical importance was not duly recognized, and they were only considered responsible for some severe intestinal symptoms (occlusion and stenosis), and for some functional disturbance of the female genital organs. Only recently, and especially since lapa-

rotomies have become so common, has it become clear that this form of peritonitis may be a prolific source of very distressing symptoms, which are occasionally masked under the guise of some well-defined clinical picture (gall-stone colic, cardialgia, intestinal colic, etc.), or may imitate some form of nervous disease (hysteria, hypochondriasis, neurasthenia), or, lastly, give rise to collections of obscure symptoms which cannot be classified. This subject has wide pathologic bearings, which are, fortunately, now becoming recognized, so that the treatment of intra-abdominal adhesions is becoming more satisfactory.

The results of this form of peritonitis are usually brought about mechanically; the adhesions attach different organs to each other, so that their passive and active movements are interfered with, or they lead to alterations in the normal position and shape of the viscera. The clinical symptoms, then, are either an expression of these mechanical disturbances or secondarily of the functional disorders—usually of a nervous character—which result from these mechanical factors.

The situation of this chronic process is, of course, of the greatest importance in determining the character of the pathologic results. The more movable an organ, the sooner the symptoms appear, and the more severe their character. This form of peritonitis is, therefore, much more important when it involves the intestine or the stomach than the liver, the spleen, or the parietal peritoneum, and its extent is less important than its situation; the most severe functional disturbances may be produced by chronic peritoneal adhesions of small extent. It is only, however, when the process is wide-spread that symptoms are constantly present.

The following description will deal with a few of the more important clinical groups. It is impossible to mention all the possible conditions which have been met with, except by giving a large number of cases in full detail.

The peritoneal adhesions around the female genital organs which have been known for a comparatively long time may produce a great variety of local disturbances, and may also be the cause of very severe nervous symptoms.

The clinical consequences of this form of peritonitis on the intestine are still more important; as already stated in previous parts of this work, the mechanical effect of these adhesions is to cause constriction, kinking, and bending of the intestine, and, as a result, dangerous interference with the permeability of its lumen (stenosis and occlusion). In addition, internal hernial strangulation may be produced either by the formation of cords, threads, or bands, by fixation of Meckel's diverticulum or of the appendix, or by fissures and holes. Finally, retracting peritonitis and mesenteritis may produce volvulus. A recapitulation of all the possible results would occupy too much space, and the reader should, therefore, refer back to the sections on Stenosis and Occlusion. It is an interesting and important fact, however, which deserves notice, that the symptoms are often out of all proportion to the changes in the peritoneum. In some cases very extensive adhesions causing great

thickening of the peritoneum and completely inclosing the intestine produce very slight symptoms, whereas, on the other hand, slight circumscribed peritonitis may lead to complete occlusion of the intestine. The determining factor is the anatomic configuration of the adhesive peritonitis.

It is desirable to repeat here the statement, already made (Section III.), that peritoneal adhesions are not, as is commonly believed, the most frequent cause of habitual constipation. Though it is true that they may occasionally produce this condition, it is exceptional.

[Gastrocolic adhesions due to ulcer or carcinoma of the stomach may produce stenosis of the transverse colon and intestinal obstruction, which Bouveret [1] believes is as frequent as that due to carcinoma of the colon. —Ed.]

Knuckling of the colon, the cecum, and the sigmoid flexure by mesenteritis, and the resulting shortening of the mesentery, is very important in this connection. Riedel believes that cases of occlusion of the bowel that recover without surgical intervention (opium, water injections), and the cases of volvulus of the sigmoid flexure which have shown manifestations of this lesion before the fully developed condition occurs, are in reality cases of temporary occlusion of the bowel due to this mesenteritis and contraction of the mesentery.

According to Gersuny, the following are the clinical symptoms of what he calls the typical flexure adhesion ; constant pain on either side of the lower part of the abdomen, often immediately before an action of the bowels, violent attacks of pain on the left side, particularly with evacuation of formed feces ; generally chronic constipation, increased pain on movement, leading, it may be, to incapacity for work.

The adhesions about the cecum and the appendix are also of great importance.

Adhesions in the region of the gall-bladder, which were also carefully studied by Riegel, are also very important. They may unite the gall-bladder to the colon, the duodenum, the stomach, the omentum, or the abdominal parietes. They are frequently produced by cholelithiasis, but in many reported cases no gall-stones were found in the gall-bladder. Adhesions of this kind may for years be the source of pain and distress. The pain may be continuous and very severe, and further be aggravated by eating, intestinal peristalsis, bending forward, jumping, lifting weights, etc. The right hypochondrium, in addition, may be painful on pressure. In other cases, again, the pain comes on in paroxysms, although gall-stones are absent, and resembles biliary colic so closely that the latter condition may be diagnosed and an operation performed, which proves the absence of gall-stones. Surgeons, as well as clinicians (Riedel, Lauenstein, Fürbringer, A. Fraenkel, Wickhoff-Gersuny, Lennander, and others), all lay stress on the great importance of these adhesions around the gall-bladder, which I can indorse from personal experience. In addition to the pain, which usually constitutes the main symptom, there may be a number of other signs, such as

[1] Bouveret, *Rev. de Méd.*, April, 1899, p. 323.

flatulence, constipation, and even intestinal obstruction. In other cases the adhesions form around the common duct and produce the symptoms of obstruction of this duct. Lastly, in exceptional cases, compression of the portal vein has been known to occur.

Perigastric adhesions starting from the stomach, especially from gastric ulcers, may spread in the direction of the liver and produce this same train of symptoms. Perigastric adhesions may involve the transverse colon, coils of the small intestine, the anterior abdominal wall, or the mesentery.

[Calwell[1] has directed attention to a special form of "adhesion dyspepsia" due to perigastric adhesions in which the pain comes on half an hour after taking food, and so is intermediate between the immediate pain of gastric ulcer and the delayed pain of atonic dyspepsia. The pain is of a dragging nature, and is aggravated by exertion, but relieved by a mechanical support or bandage. A. T. Cabot[2] has also dealt with this subject.—ED.]

In some cases the inflammatory thickening of the peritoneum has been so extensive and prominent as to form a tumor, and given rise to the diagnosis of cancer of the stomach.

[In a case of this kind recorded[3] by the editor, in a man aged twenty-seven, there was no history of gastric ulcer, syphilis, or corrosive poisoning. The stomach was dilated, a tumor in the position of the pylorus was felt, and there were thickening about the umbilicus and a moderate degree of jaundice. At the autopsy there was a simple ulcer near the pylorus, with extensive and dense adhesions between the pylorus, the small intestine, and the pancreas. The fibrous enlargement of the head of the pancreas largely accounted for the tumor felt during life.—ED.]

Riedel has recently pointed out the clinical importance of the formation, either primarily or secondarily, of cicatrices in the peritoneal covering of the kidneys, which, in the first instance, displaces and then fixes the displaced (right) kidney. This may cause years of suffering, with occasional violent attacks of pain simulating, according to the anatomic position of the adhesions, cardialgia, gastric colic, gall-stone colic, etc.

In addition to these main groups of adhesions (around the intestine, stomach, gall-bladder, and the female genital organs) there are other varieties, and any part of the abdomen may become involved in the process, so that it would be quite impossible to enumerate all the conceivable forms which have been reported.

It is clear that the symptoms are in the majority of cases produced either by interference with the motility of certain organs, as the intestine, the stomach, the gall-bladder, the urinary bladder, or that the adhesions are stretched by the movements of these different organs. In both cases the functional effect is pain.

The pain, in fact, is the principal symptom of these cases, the nature

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[1] Calwell, *Brit. Med. Jour.*, 1899, vol. ii., p. 1185.
[2] A. T. Cabot, *American Surgical Association*, May 2, 1900.
[3] Rolleston, *Practitioner*, November, 1897.

of which very often remains quite obscure. The clinical character of the pain may vary : according to the attachment of the adhesions, the pain may have the character of an attack of gall-stone colic, of cardialgia, or of intestinal colic, or may be more or less continuous. The pain may become more severe as the result of various physiologic processes (intestinal peristalsis, emptying of the gall-bladder and the urinary bladder, and entrance of food into the stomach).

[Fecal accumulation in the ascending colon gives rise to peritoneal adhesions which fix the colon in position and pin the hepatic flexure down to the surface of the right kidney and the adjacent abdominal wall in the immediate neighborhood of the ilio-inguinal, iliohypogastric, and other nerves. Pain in the course of these nerves is thus produced, which strongly suggests a renal lesion. The effect of these adhesions may be to constrict the hepatic flexure and may closely imitate a movable kidney. Free exposure of the kidney, undertaken on an erroneous diagnosis, may displace forward from their posterior attachment the adhesions which constrict the hepatic flexure, and so may relieve the symptoms for a varying time. But the symptoms may return if no effort is made to obviate accumulation in the cecum and ascending colon (W. A. Lane.[1]—Ed.]

Occasionally the pain is modified by the movements or the position of the body ; some patients are obliged to bend over to obtain relief, while others are more comfortable when on their back. Sometimes the pain is felt only in certain parts of the abdomen ; in other cases it radiates. In some instances a tender point corresponds with the situation of the spontaneous pain ; the pain may be so severe and so continuous that life is made miserable, and the patients clamor for an operation ; further, morbid nervous conditions, such as hypochondriasis, hysteria, and general nervousness, often supervene, and may dominate the clinical picture to such an extent that the local manifestations in the abdomen are lost sight of.

Objective signs, with the exception of tenderness on pressure, are often insignificant or absent. I wish, however, to lay stress on the fact that I have seen a few cases in which a firm cord could be distinctly felt through thin abdominal walls, and that its true relation to chronic peritonitis was fully appreciated and the patient successfully operated upon.

The diagnosis of chronic indurative and adhesive peritonitis can rarely be made with certainty : sometimes it can only be guessed at, and in other cases it is impossible. Lauenstein rightly points out that since positive signs are, as a rule, absent, or are at best very insignificant, a diagnosis can usually be made only by a careful and exhaustive process of exclusion. It is true, as every one knows, that this mode of diagnosis is uncertain and fallacious, but, unfortunately, in the majority of cases, there is no other means available.

When a patient complains of pain, imitating biliary colic, it is often impossible to avoid mistakes, even with the greatest care. It is only

[1] W. A. Lane, *Clinical Jour.*, 1903, vol. xxi., p. 353.

when these attacks are accompanied by continuous tenderness on pressure and when the pain is increased by movement, by distention of the stomach, and by intestinal peristalsis, that inflammatory conditions in the wall of the gall-bladder and adhesions of the organ with the neighborhood can be suspected.

When the history points to gastric ulcer and there are attacks of cardialgia and other pains in the upper part of the abdomen without any evidence to show that an ulcer is still present, the existence of peritoneal adhesions may be assumed.

The history of these cases and any data that point to the possible formation of adhesions strengthen the diagnosis (trauma, old hernias, perityphlitis, etc.). It is very important, in the first place, to exclude all other lesions. Another important point is the fact that in spite of the persistence of the pain for years no great loss of general strength nor emaciation occurs.

The only method of arriving at a positive diagnosis in many cases is an exploratory laparotomy, about the value of which there can be no difference of opinion. Sometimes the patient demands it himself.

TREATMENT.

Very extensive peritonitis involving the peritoneal cavity widely cannot be benefited by any form of treatment, even surgical intervention being useless. It is also impossible to produce absorption of circumscribed peritonitis or of adhesions. No one will imagine that internal remedies, massage, or counterirritation can have any effect. The use of artificial and natural waters (salt, sulphur, peat, mud) has no influence on this chronic process, though baths sometimes, but by no means always, temporarily relieve the pain.

[The pain of adhesion dyspepsia is relieved by an easily digested diet, rest in bed, and the application of a mechanical support or bandage (Calwell [1]).—Ed.]

The inadequacy of all other means leads us to surgery. From the continuous pain many of the patients are urgent in their requests for operative intervention; the brilliant results of modern abdominal surgery, moreover, fully justify this treatment, and many operators have published remarkable results (Riedel, Maydl, Lauenstein, Salzer, Alex. Fraenkel, Gersuny, and others), and many cases in my own practice have been relieved by surgical treatment.

However true this may be, successful results from surgical treatment cannot always be guaranteed. Occasionally the exploratory laparotomy brings to light unexpected complications or a condition of affairs which could not be previously diagnosed, or one which, even if adhesions are found, is not amenable to surgical treatment, possibly because the adhesions are too extensive. Further, even though surgery is a perfected art, the possibility of accidents from the operation itself must not be lost sight of.

[As already pointed out, laparotomy itself may set up adhesions, so

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[1] Calwell, *Brit. Med. Jour.*, 1899, vol. ii., p. 1185.

it has been thought that operation ought not to be undertaken for painful peritoneal adhesions. With this view Nové-Josserand and Goinard [1] join issue and point out that by scrupulous antiseptic precautions during the operation, and by keeping up peristaltic action of the intestine after the operation by means of purgatives, etc., the formation of fresh adhesions may be prevented.—Ed.]

The patient should, of course, be told of all these possibilities, and be given an opportunity of considering the pros and cons. It must be admitted, however, that an operation is often the only means of relieving the pain, and that patients are often restored to a useful existence by surgical intervention.

The technic of the operation can, of course, be determined only after the abdomen has been opened, and the operator has had an opportunity of examining, both by inspection and by the finger, the anatomic conditions requiring treatment. Sometimes the mere division of a single adhesion may give relief after years of suffering; in other cases it is necessary to separate a number of adhesions, and the process may be tedious and even dangerous; in other instances a small piece of constricted intestine may require removal, or, again, a whole mass of adherent bowel may have to be resected. A description of the numerous details of the surgical procedures does not come within the scope of this work.

[Duplant [2] has recently given a detailed account of the surgical treatment of anterior adhesive perigastritis.—Ed.]

TUBERCULOUS PERITONITIS AND TUBERCULOSIS OF THE PERITONEUM.

This affection was known anatomically as early as the beginning of the last century (Bichat, Laennec, Bayle, and others), but it did not command the interest of clinicians until Louis' dictum, in 1825, that chronic peritonitis is usually tuberculous in character. For several decades clinicians investigated the minor details of the clinical and anatomic aspects of the disease, until Koch discovered the bacillus of tuberculosis, and the question was discussed how the entrance and the exit of the micro-organism from the peritoneum were effected; quite recently —that is, within the last fifteen years—the satisfactory results obtained from laparotomy have stimulated interest in the treatment of tuberculous peritonitis.

The affection is interesting because it is by no means rare. Reliable figures cannot, of course, be obtained from examination of the patients during life, but only from postmortem reports; this is due to the fact that the diagnosis cannot always be made with certainty *intra vitam*. Postmortem statistics, however, are available to show the incidence of peritoneal infection in cases of tuberculosis. The statistics of different authors show considerable variation, some giving 1.25 per

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[1] Nové-Josserand and Goinard, *Lyon Médical*, November 14, 1897.
[2] Duplant, *Rev. de Méd.*, 1903, p. 634.

cent., others as high as 16.16. This last high estimate is given by Borschke, who, in 4250 autopsies at the Breslau Pathological Institute (during six years), found 1393 cases of tuberculous disease, and tuberculous infection of the peritoneum in 226 of these 1393 cases— *i. e.*, in 16.16 per cent. Although I cannot support my view by statistics, it appears to me that in Vienna the disease is much more frequent than in other places where I have worked at different times.

ETIOLOGY AND PATHOGENESIS.

The cause of peritoneal tuberculosis is the entrance of the tubercle bacillus into the peritoneal cavity.

The factors favoring the development of the disease cannot be recognized sufficiently constantly in all cases to justify their being mentioned as special disposing elements in the etiology of the affection. The most remarkable point seems to be the age of the patient; the following was the age incidence of the cases in my clinic: Among 164 cases of tuberculous peritonitis (101 males and 63 females), 28 occurred between eleven and twenty, 37 from twenty-one to thirty, 50 from thirty-one to forty, 31 from forty-one to fifty, 12 from fifty-one to sixty, 6 from sixty-one to seventy years. Münstermann also states that the disease is most prevalent from thirty to forty. Ad. Frank (Czerny's clinic) gives thirty to forty as the decade with the greatest incidence. O. Vierordt, in his first publication, does not mention any case under twenty; this is probably an accident, for in a subsequent publication he says that he has met with cases in children. All other authors seem to agree that middle life is the period with the greatest incidence, while old subjects and children are relatively rarely affected. Sex plays a peculiar rôle in our statistics; among the cases discovered after death there are many more males, none of whom died after operation, while among the subjects who were operated on for the disease many more women (90 per cent.) were found. This is probably due to the fact that in women operations are far more often performed because some other diagnosis is more commonly made (diseases of the sexual organs).

[In his statistics of cases operated upon Osler[1] finds that females are affected twice as often as males, in the ratio of 131 to 60.—ED.]

Pathologic anatomists (Rokitansky and Förster, Weigert, E. Wagner, Grawitz) have found that in many cases of peritoneal tuberculosis there is often a concomitant cirrhosis of the liver, and have suggested that possibly portal stasis exerts a favorable influence on the development of tuberculosis of the peritoneum. Among 13 fatal cases of tuberculosis of the peritoneum examined after death in my clinic during the last few years, cirrhosis of the liver was found twice. O. Vierordt found cirrhosis 5 times in 24 cases, but only well developed in 1 of these cases; in the other 4 cases—only 2 of the 5 were drunkards, 1 was a young girl of twenty—Vierordt assumes that the cirrhosis was secondary to the tuberculous peritonitis, and that the interstitial proliferation of the

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[1] Osler, *Principles and Practice of Medicine*, 1901, p. 286, fourth ed.

connective tissue in the capsule of the liver passed into the liver from the hilum. Brieger does not express any definite opinion, and Courtois-Suffit believes that alcoholism *per se* is the predisposing factor " *avec ou sans cirrhose atrophique.*" Moroux found a combination of cirrhosis and tuberculous peritonitis in 13 cases, all the patients being drunkards, and believes that the peritonitis was preceded by the cirrhosis.

[In 121 cases of hepatic cirrhosis examined after death Kelynack[1] found tuberculous peritonitis in 12, or nearly 10 per cent.—Ed.]

How do the tubercle bacilli reach the peritoneum? There are three possible channels, viz., the blood-vessels, the lymph-channels, or extension by direct continuity from tuberculous foci in the vicinity of the peritoneum.

The disease is rarely conveyed to the peritoneum by the general blood-stream (Weigert), and presumably only in miliary tuberculosis. Under these circumstances small miliary tubercles may occasionally develop in the peritoneum as in any other organ or serous membrane, but there is merely tuberculosis of the peritoneum and not the anatomic changes of inflammation, presumably because the necessary inflammatory irritants have not gained an entrance to the peritoneum.

The lymph-channels are much more important, and the disease is commonly transmitted through them. It is always important to determine the source from which the tubercle bacilli get into the lymph-channels.

Postmortem data show that primary tuberculous peritonitis is exceedingly rare; among Borschke's 226 cases there were only 2 in which the tuberculous process was limited to the peritoneum; Münstermann, it is true, found 1 case among his 46 cases.

[In 5 out of Osler's 17 postmortem cases the disease was primary and local in the peritoneum.—Ed.]

In all these cases it is, of course, possible that a primary focus in some other part of the body was overlooked, but apart from this possibility it must be assumed that the bacilli actually passed through the uninjured intestinal wall to the peritoneum through the lymph-channels of the bowel-wall.

There is another group of cases in which, in addition to tuberculosis of the peritoneum, the pleura or the pericardium are involved—tuberculosis of the serous membranes. In some cases the infection of the pleura can be shown to be primary, and that of the peritoneum secondary, while in other cases the reverse must be assumed. The extension from one serous membrane to the other occurs through the lymphatics. It is, however, just as difficult to determine how the serous membrane first involved became infected, as it is to determine how primary peritoneal tuberculosis develops.

In another group—and this is the largest—the lungs are primarily affected, while no other abdominal organ except the peritoneum is infected. Weigert, von Recklinghausen, and Ponfick have described the passage of micro-organisms in this condition by the lymph-channels,

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[1] T. N. Kelynack, *Birmingham Med. Rev.*, February, 1897.

and have shown that it is frequently aided by the mechanism of reversal of the lymph-stream.

In other cases the primary focus is in the bones or in the skin ; thus special attention should be directed to the appearance of tuberculous peritonitis after hip-joint disease. Again, the primary focus may be found in the suprarenals. Lastly, there is an important group of cases in which the primary disease is in the bronchial or mesenteric glands.

Infection occurs quite frequently from the genitals, particularly in women. Salpingitis or affections of the uterus, the vagina, or the ovaries can all be starting-points. The examination of the vaginal secretion for tubercle bacilli is important, for even in children, as many clinicians have pointed out, positive results may be obtained. In male subjects diseases of the testes and of the epididymes are particularly important.

The relation between tuberculosis of the intestine and of the peritoneum has been exhaustively studied. Borschke's figures gave surprising results. Among his 226 cases there was not a single case of primary tuberculous disease of the intestine ; on the contrary, the intestine was perfectly free from tuberculosis in 86 cases ; in 140 cases there was a marked primary pulmonary tuberculosis, with pronounced tuberculosis of the bowel. On the other hand, Baumgarten found a small tuberculous ulcer close to the ileocecal valve, which, though the only tuberculous lesion in the whole body, had set up diffuse peritonitis, without perforation, most marked in the vicinity of the ulcer. It must, however, be granted that it is only in exceptional cases that the intestine is the primary seat of the tuberculosis which subsequently infects the peritoneum. I do not, of course, wish to deny that in cases of pulmonary tuberculosis in which the bowel, and subsequently the peritoneum, become infected the bacilli do not travel via the intestine to the peritoneum (from infiltrated Peyer's patches, ulcers).

The latter cases, in which the infection of the peritoneum occurs from deep intestinal ulcers extending down to the peritoneum, properly belong to the third category, in which infection occurs by direct continuity of tissues. In such instances, however, the tubercles and the peritonitis, as a rule, remain localized to the immediate vicinity of the ulcer ; some authors, among them Courtois-Suffit, claim that peritonitis originating in this way never becomes diffuse.

PATHOLOGIC ANATOMY.

The morbid appearances of tuberculous peritonitis vary. In some cases there is simply an eruption of miliary tubercles without any manifestations of inflammation, while in other cases the latter are present and the anatomic appearances are those of a simple acute inflammation. This is seen in acute miliary tuberculosis which runs an acute course, in cases in which the pleura and other organs are involved at the same time as the peritoneum. The parietal and the visceral layers of the peritoneum, the omentum, the mesentery, and especially the capsules of the liver and spleen are involved in the process and are covered by a large

number of small miliary tubercles, varying in size from exceedingly small punctiform nodules to nodules as large as a millet-seed; the extremely minute tubercles, which are the more recent, are gray, translucent, and barely visible to the naked eye. The rest of the peritoneum may be perfectly smooth, or at most a little injected, and often contains some fluid exudate, consisting either of serum or of serofibrinous material, or containing varying quantities of pus or of blood. The opinions of different observers as to the occurrence of a fluid exudate in acute miliary tuberculosis of the peritoneum vary greatly. Some authors state that this exudate is invariably present; on the other hand, Borschke found that among the 16 cases of miliary tuberculosis in his series of 226 cases of tuberculosis of the peritoneum, only 2 showed a fluid exudate. Acute tuberculous peritonitis is, as a rule, diffuse, and rapidly causes the death of the patient. The exudate is always freely movable unless there are old adhesions.

In another group of cases the tuberculous process is circumscribed— *e. g.*, over tuberculous ulcers of the bowel. In these cases the peritoneum is injected and occasionally considerably vascularized, thickened, and covered with tubercles and fibrin; the latter often give rise to adhesions with neighboring organs. This circumscribed form is also common in the pelvis, particularly around the female genital organs. Here, too, sacculated accumulations of pus are often seen, as in the form to be described in the following paragraphs. Circumscribed tuberculous peritonitis may also occur in other parts of the peritoneal cavity, although it is not so common as in the situations just mentioned.

The classic anatomic appearances are seen in the chronic and subacute forms of diffuse tuberculous peritonitis; there is a plastic fibrinous exudate that may lead to the formation of cicatricial masses, an eruption of tubercles that may often coalesce into large nodules, and, lastly, a fluid exudate of varying composition, which, as a rule, is more or less encysted.

The quantity of exudate in this form of peritonitis is often moderate, but it may amount to many liters (in one case under my care 11,500 c.c. of fluid were removed). The composition of the fluid is subject to variations: it may be simply serous, serofibrinous, or hemorrhagic, rarely purulent or mixed with pus and blood, so as to assume a dark and dirty color; occasionally, too, it is milky and opaque (see Ascites Adiposus). Sometimes bacilli can be found in the exudate, but they are not necessarily present in genuine tuberculosis of the peritoneum, and according to several observers, it may be quite difficult to detect their presence, even in the tuberculous nodules.

The tubercles are usually abundant; they permeate the fibrinous deposits as well as the portions of the peritoneum which are not thickened. They vary in size from very small miliary tubercles to cheesy nodules of considerable dimensions, which on cross-section are composed of cicatricial tissue.

[MacCallum[1] has described a remarkable case of pendulous tubercu-

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[1] W. G. MacCallum, *Johns Hopkins Hosp. Bull.*, vol. xv., p. 293.

lous masses in the peritoneal cavity, resembling " Perlsucht" in lower animals. A similar case was described by Bizzozero.[1]—Ed.]

The peculiar character of the process is due to the presence of large masses of fibrin and tubercles. Sometimes the whole surface of the intestine is covered by a mass of very vascular granulations. The fibrinous material causes the different coils of intestine to become adherent to each other, to the abdominal wall, the liver, the spleen, or the pelvic organs. The mesentery is affected in the same way. The great omentum is in many cases greatly altered, and is converted into a tough mass containing fibrin and numerous tuberculous nodules, and adherent to the visceral and parietal peritoneum.

The fibrinous masses often inclose cavities filled with fluid exudate, and a number of these cysts may thus be found in the same case.

There is always a tendency to cicatricial contraction, which is most marked in the mesentery and the omentum. The changes thus produced in the omentum have long been described; it is converted into a tough, thick cord which resembles a tumor, and is, in fact, often thought to be one during life; it runs transversely across the abdomen, or passes across the upper part of the abdomen in a diagonal direction. The mesentery also undergoes contraction. The different coils of the intestine may become adherent to each other and form a hard mass which also simulates an abdominal tumor; when the fluid is absorbed, the changes eventually develop that are spoken of as peritonitis deformans.

As the morbid process often progresses in stages, a variety of anatomic appearances are often seen side by side : in one place the peritoneum is normal; elsewhere there are a few tubercles and slight injection, a fluid exudate within a fibrinous capsule, masses of fibrinous exudate that are full of tubercles, and, lastly, tough cords of cicatricial tissue.

It is very important to note that in this disease the cicatricial masses may develop more rapidly than the tubercles, so that the latter become encapsulated and may thus disappear. In these cases no tubercles can be found on postmortem examination, and nothing is seen but the cicatricial tissue. This undoubtedly constitutes a spontaneous cure. (The reader should refer to the paragraphs on spontaneous cure in the pages devoted to the course and the prognosis of this disease.)

The process often, however, takes a different course; perforation may occur, usually into the intestine or into other organs, or through the abdominal wall. [Pye Smith[2] refers to a case in a child in which an abscess was opened and an intestino-cutaneous fistula resulted.—Ed.]

The situation of a cutaneous fistula is comparatively often either at or near the umbilicus, but perforation at this point is not pathognomonic of tuberculous peritonitis, for it is often met with in other forms of purulent peritonitis (Vallin). These features have all been described on pp. 750–763. Other serious sequelæ may depend on the development of permanent adhesions.

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[1] Bizzozero, *Morgagni*, 1867, vol. ix.
[2] Hilton Fagge and Pye Smith, *A Text-book of Medicine*, 1902, vol. ii., p. 489.

Changes in the liver and the frequent incidence of cirrhosis have already been mentioned (see p. 942). The spleen is often considerably enlarged (O. Vierordt), the reason for which is not clear. The mesenteric and retroperitoneal lymph-glands are, as a rule, increased in size and caseous.

Lastly, it may be mentioned that there is, as a rule, tuberculosis of some other organ besides the peritoneum. Special attention should be directed to the frequent involvement of the pleuræ and pericardium, already referred to in the paragraphs on the etiology of the disease (tuberculosis of serous membranes).

[In tuberculous peritonitis the glands in the anterior mediastinum, especially those in the first intercostal space close to the internal mammary artery, may be enlarged and tuberculous as the result of tubercle bacilli absorbed from the peritoneum. Durham's [1] observations as to the frequency and rapidity with which these glands become infected in peritoneal infection and in peritonitis have already been referred to (see p. 692).—ED.]

SYMPTOMS.

As a rule, the clinical features of this disease are so definite that the diagnosis is easy ; in some cases, however, other conditions may be simulated, among them cirrhosis of the liver and portal obstruction, an ovarian cyst, or even enteric fever, while in other cases no diagnosis whatever can be made.

Acute miliary tuberculosis of the peritoneum, as a rule, remains latent, and the grave manifestations of the general infection dominate the scene. This is due to the fact that in this form symptoms of inflammation are slight or completely absent. It is only when there is much fluid exudate, and even without this, when there is much abdominal pain, that peritoneal infection in cases of generalized tuberculosis appears probable.

Circumscribed tuberculous peritonitis—for instance, that over a tuberculous ulcer of the bowel—can be suspected only where there is local pain over the affected area. Other circumscribed forms of tuberculous peritonitis which form a palpable tumor containing encysted pus may be diagnosed under favorable circumstances in the same way as any other form of localized exudative peritonitis. The diagnosis of the tuberculous character of the peritonitis, of course, depends on other factors, such as the presence of a tuberculous focus elsewhere in the body.

The clinical picture of the main form of the disease—*i. e.*, the form that is always implied when tuberculosis of the peritoneum is commonly spoken of—depends on a variety of factors : in the first instance, on the presence of a large amount of exudate, or, secondly, on the formation of hard fibrous masses containing tubercles.

[Gee [2] divides tuberculous peritonitis into four forms : (1) The adhe-

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[1] H. E. Durham, *Jour. Path. and Bact.*, vol. iv., p. 360.
[2] S. Gee, *St. Bartholomew's Hosp. Jour.*, May, 1900.

sive, the commonest and usually the most easily detected form ; (2) the suppurative ; (3) the tympanitic ; and (4) the ascitic, often associated with the cirrhosis of the liver.—Ed.]

The local manifestations of peritonitis as they present themselves in the tuberculous form of the disease will first be described.

Pain is again the dominant symptom ; as a rule, it is slight, rarely very severe, and may either appear spontaneously or only on pressure. It should be specially insisted on that complete absence of the pain is by no means rare ; several patients under my care stated that they never felt any pain. This is an important point, for it shows that absence of pain does not exclude tuberculous peritonitis. The distribution of the abdominal pain varies ; occasionally there is localized tenderness on pressure.

In cases that run a rapid course there may be vomiting ; as a rule, however, this symptom is absent. Diarrhea is usually present ; in general there are not more than three or four evacuations, but the diarrhea may be more profuse. Severe diarrhea usually indicates tuberculous ulceration of the intestine ; moderate diarrhea may, however, be present, without any ulceration. Diarrhea is by no means constant, for in some cases the bowels act normally or may even be constipated (without any obstruction to the passage of feces). Hiccup is nearly always absent. Meteorism may be present or absent, and is not in any way characteristic of the disease.

The physical signs produced by the exudate vary according to its constitution. The well-known signs of an extensive, freely movable exudate are present, often with the additional sign of dilated abdominal veins. Two factors are characteristic of the exudate in tuberculous peritonitis—viz., first, that it is often encysted, either from the onset, or subsequently becomes so, though it may have been freely movable originally (persistence of a freely movable exudate must be considered the exception) ; second, that there are some peculiarities in the distribution of the areas of dulness.

The first phenomenon is easily explained by the tendency to the formation of plastic fibrinous exudates in this disease. It is obvious that this peculiarity may greatly complicate the diagnosis. It is well known, for instance, that encysted peritonitis often imitates an ovarian cyst. When the abdomen is very prominent, fluctuates all over, retains its shape in every position, is dull on percussion all over, with the exception, possibly, of a small area of relative resonance along the right side and in the epigastric region, which does not vary with changes in the position of the patient, this mistake may easily occur.

In any form of encysted peritonitis there may, of course, be variations in the areas of dulness in the abdomen.

Thomayer has, however, described a peculiar distribution of the tympanitic area which he regards as characteristic of tuberculous (and carcinomatous) peritonitis and of some diagnostic value. It is the following : in tuberculous and in carcinomatous peritonitis the area of dulness is not uniformly distributed all over the abdomen with a tym-

panitic note only in the areas corresponding to the air-containing intestine, but the tympanitic note is always distributed over a much larger area on the *right* side of the abdomen than on the left. This is due to the fact that the coils of the small intestine, owing to the mesentery being more on the right than on the left side, are drawn more to the right than to the left when mesenteritis retrahens develops; as this form of mesenteritis is chiefly produced by tuberculous and carcinomatous peritonitis, this peculiar distribution of the tympanitic note may be considered to be of importance in the diagnosis of these forms of peritonitis.

I agree with Thomayer that this holds good in the majority of cases, and also acquiesce in his reservation in other cases. It is clear that the sign that he has described may be absent when the retraction of the mesentery has not yet taken place. Again, the reverse of what has been described may be found in tuberculosis or carcinoma when a large tumor mass occupies the right side of the abdomen and a moderate amount of exudate is forced over to the left; and, again, the presence of adhesions between the intestine and the anterior abdominal wall may account for a different distribution of the tympanitic note, or, conversely, adhesions not due to tuberculosis may produce conditions which imitate those described in tuberculous peritonitis with mesenteritis retrahens. Still, the sign described by Thomayer may, with some reservations, be of use in arriving at a diagnosis.

The tumor-like growths of the omentum and tumors situated between the coils of intestine (so-called pseudo-tumors), described long ago by Bamberger, are also of great clinical importance. After absorption of the exudate, or when there is little exudate at any time, tumors may be felt in different parts of the abdomen. They are usually fixed, or, rarely, movable, and vary in size from that of an adult's fist to much larger tumors. A friction-rub can sometimes be heard or felt over them. One form of tumor which is particularly interesting appears as a hard cord running transversely or diagonally across the upper part of the abdomen, from one costal arch to the other—this is the omentum, which has become thickened and rolled up as a result of tuberculous inflammation and infiltration. When there is a large exudate, these tumors can usually be felt deep down by quickly depressing the finger-tips vertically downward, and, after paracentesis, can usually be felt without any difficulty. When very small, they can be felt only after tapping. It need hardly be mentioned that in many cases these tumors are absent, and, conversely, that similar tumors occur in non-tuberculous chronic peritonitis. Many cases of this kind have come under my observation, and others have been published. (See the section on Chronic Adhesive Peritonitis.)

Lastly, in some cases there are neither tumors nor fluid exudate, but only a diffuse eruption of tubercles, with more or less adhesions which cannot be diagnosed by physical examination.

The tumors, adhesions, and the scar tissue may interfere with the permeability of the intestine in many ways (by compression, constriction,

kinking), and so lead to the symptoms of stricture of intestinal obstruction.

Although far less essential than the local signs, some of the general symptoms are of some importance.

The temperature varies greatly in different cases : there may be no fever at all; this is an important point, because many are still inclined to the view that absence of fever excludes tuberculous peritonitis. It is true that in many cases there is fever, which either may be continuous, and of moderate or of considerable elevation, or may be remittent or intermittent, of a regular or an irregular type, and occasionally markedly hectic. It is important to remember—and I wish to corroborate this point—that there may be intervals in which the temperature remains normal. The respiration and the pulse are influenced by so many concomitant conditions that no constant rule can be made. It may be mentioned that the excretion of large quantities of indican, which is so characteristic of diffuse acute peritonitis, is absent in the tuberculous form of the disease.

[Ehrlich's diazo-reaction may be present. The majority of cases do not show leukocytosis, and its presence suggests a secondary infection. In 46 cases Shattuck[1] noted that it was absent in 34, or 70.8 per cent.—Ed.]

Vierordt lays stress on the general wasting in these patients, which is due to the peritoneal affection and not to advanced complications in the lungs or in other organs. However true this may be, stress should be laid on the fact that in many fatal cases there is no wasting. [Occasionally the skin is pigmented, and may lead to a diagnosis of Addison's disease (Osler[2]).—Ed.]

Berggrün and Katz have stated that colorless and fatty stools (see p. 91) are a characteristic sign of tuberculous peritonitis in children. These stools are certainly passed in this disease by children, but the same phenomenon is also observed in a number of other conditions, especially in most cachexias. At the same time the passage of acholic motions in a case that is undoubtedly suffering from peritonitis is an argument in favor of a tuberculous origin.

The spleen is often enlarged, but the examination of this organ is often difficult when considerable ascites or large tumors are present. The liver is usually unchanged, but sometimes it is enlarged, and may be smaller or hard, and present the signs of atrophic cirrhosis.

Concomitant tuberculosis of the lungs, the pleura, or the pericardium will give rise to their characteristic physical signs. The severity of the disease is, of course, greater when these complications are present.

[Oehler[3] points out that in boys in whom the processus vaginalis has not been obliterated, chronic tuberculous inflammation may spread to the spermatic cord and give rise to thickening.—Ed.]

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[1] F. C. Shattuck, *Amer. Jour. Med. Sci.*, vol. cxxiv., p. 1.
[2] Osler, *Principles and Practice of Medicine*, 1901, p. 287, fourth ed.
[3] Oehler, *Münch. med. Wochenschr.*, 1901, p. 1823.

DIAGNOSIS.

The diagnosis of tuberculous peritonitis is usually correctly made, in my own practice, at least. This diagnosis has rarely been shown to be wrong at the autopsy. It more often happens that the disease is found postmortem when it was not suspected during life. The general clinical manifestations are usually so characteristic in spite of the indefinite nature of some of the signs that a correct diagnosis is generally made. At the same time, mistakes are often made, and any one who has had occasion to study the manifold combinations of signs which may appear will agree that this is sometimes unavoidable. The greater number of erroneous diagnoses are made in the following circumstances :

The pseudo-tumors may be regarded as other tumors arising in one of the viscera (the stomach, intestine, gall-bladder, kidney, glands, female genital organs), especially when there is no accompanying fever. It is necessary to make out, first, whether there are signs of tuberculosis anywhere else ; a positive result is important, but a negative answer is not so important (for the details see the section on the Etiology). Again, the pseudo-tumors are rarely single ; they are generally multiple, and the one in particular that can be best palpated, namely, the rolled-up omentum, has such a characteristic position and shape that the diagnosis will instinctively be first directed toward tuberculosis ; in order to differentiate this tumor from gastric carcinoma the chemic reaction of the contents of the stomach must be investigated. It is true that multiple carcinomatous tumors may occur in the abdomen in carcinomatous peritonitis, but the course of the case, the discovery of a primary cancer, the appearance of the initial symptoms in some organ, such as the uterus or the stomach, which is rarely affected with tuberculosis, but often with carcinoma, will all help in making the differential diagnosis.

Since the day when Spencer Wells made the historic diagnostic error of confounding an ovarian cyst with sacculated tuberculosis of the peritoneum this mistake often has been committed even by the most experienced clinicians. The mere fact that these two lesions may easily be confounded is all that can be said on this subject here.

It is often difficult to distinguish an exudate that is due to hepatic disease, especially cirrhosis, from tuberculous peritonitis with a large effusion. Fever and pain may be completely absent in the latter affection, and even the fact that after paracentesis the liver is found to be cirrhotic does not alone decide the diagnosis, for, as I have said, the two affections may occur together, the cirrhosis of the liver either favoring the development of tuberculous peritonitis or the cirrhosis developing as a result of the peritonitis. It is well, therefore, to recognize that the two conditions may be combined. An alcoholic history, distinct splenic enlargement (although this is sometimes also found in peritoneal tuberculosis), the fact that the ascitic fluid is free, the character of the aspirated fluid (compare ascites), are all in favor of cirrhosis. The discovery of a primary tuberculous focus is, of course, of the greatest importance.

The diagnosis is relatively easy when there is a combination of pleurisy (bilateral) and pericarditis, a condition which occurs in Vienna

with surprising frequency. As transudation from cardiac or renal disease can usually be eliminated without difficulty, the only condition to be excluded in these cases is carcinomatosis of the peritoneum, which is usually easy when primary carcinoma has been found.

It is often much more difficult to differentiate a "simple" peritonitis from the tuberculous form when there is nothing definite in the history of the case, and when no information can be obtained by physical examination. In such cases the following dictum of Vierordt's applies, which I can indorse from personal experience: "The great majority of those cases of subacute and chronic peritonitis which are not due to injury and are not accompanied by tuberculosis of the lungs or other organs, or are not accompanied by a demonstrable malignant new growth, and are hence characterized as sarcomatous or carcinomatous, must be considered to be tuberculous." This all implies that in such cases the diagnosis, at best, is an assumption, and merely the most probable solution.

Occasionally, under such conditions, a control experiment upon animals is of great use (Borchgrevink, Rose-Naunyn), for injection of the exudation from the peritoneal cavity into guinea-pigs causes tuberculosis.

Again, the diazo-reaction, when marked and constant, can be used for clinching the diagnosis.

Stress should be laid on the fact that in doubtful cases injection of tuberculin may clear up the diagnosis. A distinct reaction in a patient who was previously free from fever may be considered a strong argument in favor of the diagnosis of tuberculosis.

Finally, laparotomy, provided it is justifiable, may decide the diagnosis.

COURSE; PROGNOSIS; RECOVERY.

The disease usually comes on insidiously; the patient has some little pain in the abdomen, but continues about his work until he begins to notice swelling of the abdomen, fever, loss of appetite, or increasing weakness, when he consults a doctor. In some cases the onset of the disease may be even less striking : there may be no pain at all and nothing noticeable except some dyspepsia, until hard lumps are felt in the abdomen. Again, in other cases there is a febrile temperature from the very beginning, and the course is subacute.

[In some cases the onset is extremely sudden, suggesting that a softened tuberculous gland has ruptured into the peritoneal cavity; vomiting and evidence of definite obstruction or severe gastro-enteritis may accompany the onset of this form of tuberculous peritonitis, which Gee[1] spoke of as the tympanitic form.—ED.]

O. Vierordt reports a case in which the disease began acutely on May 18th with vomiting, nausea, loss of appetite, colic, and diarrhea. On the fourth day, when the patient was examined for the first time, the temperature was 103.6° F. and the whole aspect of the case was that of typhoid fever. After about fourteen days the type of the fever

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[1] S. Gee, *St. Bartholomew's Hosp. Jour.*, May, 1900.

became remittent, rising as high as 104.4° F., while the stools were typhoid in character; there were meteorism, pain over the abdomen; but no splenic enlargement. In addition, there was pleurisy with effusion on the right side. Finally, the abdomen became very painful, and there was complete intestinal obstruction. On June 29th the patient died. The autopsy showed diffuse sanious tuberculous peritonitis, with a perforated tuberculous ulcer of the cecum; hemorrhagic pleurisy on the right side, and old induration of the apices of both lungs, with a few caseating foci.

The changes in the clinical picture produced by various complications, such as pleurisy, pulmonary tuberculosis, perforation of the peritonitic exudate into the intestine or through the skin, and other conditions, cannot be described in detail. One point, however, is of special importance—viz., that the disease sometimes shows a tendency to remissions and may become perfectly quiescent without any treatment whatsoever. This remarkable fact was first noticed by Vierordt, and I can confirm it from my own experience. The fever stops, the appetite returns, the patients gain in strength, the amount of exudate decreases —in fact, even the inflammatory tumors, as I know from direct observation, may disappear. The patient, in other words, becomes a useful member of society. Such a remission may last as long as three years (Vierordt).

These remissions naturally raise the question whether tuberculosis of the peritoneum can be spontaneously cured. Although some of the older observers, as Bamberger, long ago believed that this was possible, the majority of more modern clinicians seemed inclined to the idea that the prognosis of this affection *quoad sanationem* was absolutely bad. This view was somewhat shaken in 1884 when König showed that the disease could be cured by laparotomy; and since then by the numerous published cases of recovery after surgical intervention. It follows that the cases of apparent recovery without surgical intervention acquire a new significance. Formerly, the objection could always be raised that the case was not really one of tuberculous peritonitis; but now, since the results of surgical cases and of experimental research prove that recovery can occur, there is no good reason to doubt that other cases diagnosed as tuberculous by competent clinicians—and numerous examples have been published in the last ten years—did not also recover spontaneously. The principle is, therefore, established that some cases of this disease, which were formerly believed to be invariably fatal, can recover spontaneously; it is another question whether recovery is possible in all the different anatomic forms.

This question of spontaneous cure is intimately connected with the surgical treatment of the disease, and as the only way to explain the former is to study the latter, the surgical treatment of the disease will be dealt with here. It is, of course, impossible to quote all the existing monographs on the subject, and the reader should refer to the works of other authors for the complete bibliography (Lindner, Philipps, Hinterberger, Valenta, Roersch, Teleky, U. Rose, and others).

The possibility of curing tuberculous peritonitis by operative interference has been established beyond any doubt by eminent surgeons and gynecologists (Hönig, Czerny, Hegar, Chrobak, Bumm, Israel, and others). The tuberculous nature of the disease was proved in the majority of these cases not only by the presence of tubercles, but also by the demonstration of giant-cells, and, in many instances, of tubercle bacilli. In some of the cases positive animal experiments were also made. The objection that in some of the cases bacilli were not found in the tubercles or in the exudate is not a valid argument, for it is a well-known fact that the bacilli cannot always be found, even when they are present. In fact, some are inclined to the view that the fibrous nodules found in some cases of peritonitis which are not ordinarily considered to be tuberculous are, in reality, healed tubercles.

The most surprising result is that a complete *restitutio in integrum*, a complete cure, of the pathologic process can be brought about in some of the cases; a good many cases are now on record in which not only the signs of inflammation, but even the tuberculous nodules and the adhesions disappeared, and the peritoneum became perfectly healthy and smooth.

Israel, in the course of a laparotomy, found fibrinous masses and tuberculous nodules the size of cherry-stones. Thirty-six days later the abdomen was again opened and no trace of tubercles could be found, and only a single nodule could be discovered on one loop of intestine, which, on microscopic examination, was shown to consist of fibrous tissue containing a drop of oil derived from an injection of iodoform oil. The patient recovered after the second operation.

In a patient of Chrobak's double salpingo-oöphorectomy was performed by the abdominal route for tuberculosis of the adnexa and of the peritoneum; the peritoneum was covered with nodules (histologically, tubercles) varying in size from that of a millet-seed to that of a hemp-seed; the parietal peritoneum was, moreover, adherent to the intestine by a pseudo-membrane, and the visceral peritoneum over the intestine in its turn was covered with similar nodules. Three years and a half later another laparotomy was performed for a ventral hernia; the peritoneum was then found to be perfectly smooth, there being no nodules, ascites, or adhesions. In a case of Hirschberg's, where laparotomy was successfully performed for tuberculous peritonitis, the patient died eight months later from pulmonary phthisis, and at the autopsy the peritoneum was perfectly smooth. Jordan has collected similar cases.

In other cases recovery occurs clinically while anatomic evidence of the morbid process persists. The patients are free from all the symptoms of tuberculosis of the peritoneum, but at a second laparotomy or at autopsy the peritoneum is found covered with nodules, which, however, no longer present the characteristics of tubercles. Thus in one case of Bumm's, where three liters of fluid were evacuated, the peritoneum was covered all over with nodules of the size of millet-seeds, which, on microscopic examination, were found to be true tubercles; eight weeks later a second laparotomy was performed, and again nodules were found, but they were much contracted by cicatricial change in the adjacent tissues, and contained neither giant-cells nor bacilli.

Although results are far from being equally favorable in all cases, those given above prove that a genuine cure of tuberculous peritonitis may actually occur, and as—I repeat it—all subjective and objective

signs of the disease may sometimes disappear without operation, and in face of the most indifferent therapeutic treatment, I do not hesitate to express my belief that a spontaneous cure of the disease may occasionally take place.

The question how the recovery is brought about is of the greatest interest. Investigations on human subjects, such as those made by Bumm in the case already quoted, seem to show that one method is obliteration of the tubercles by becoming surrounded in a capsule of connective tissue. Nothing remains in such cases but harmless nodules of cicatricial tissue and adhesions. A number of experimental investigations have given some further information, particularly those of Gatti, Stschelogeff, Varvilevsky, Nannoti, and Baciocchi. Gatti came to the conclusion that the retrograde metamorphosis of the tubercles was due neither to adhesions nor to obliteration of the tubercles by connective-tissue hyperplasia, but to a slow degeneration and dissolution of the epithelioid cells without proliferation of connective tissue. The process, as he describes it, is the following: a few days after the laparotomy a quantity of blood-serum enters the peritoneal cavity, which, by its bactericidal properties, reduces the vitality of the bacilli or destroys them; later, regeneration occurs, which eventually leads to complete recovery. In this way the complete disappearance of the nodules can be explained, as well as the fact that after a time the peritoneum becomes perfectly smooth.

Bumm believes that migrating cells appear, crowd the epithelioid cells apart, and thus destroy the giant-cells; then young blood-vessels penetrate into the center of the tubercle, and the constituents of the tubercle are replaced by young connective tissue. Stschelogeff, as well as Nannoti and Baciocchi, lays special stress on fibrous metamorphosis of the tubercles. The fact, however, that the peritoneum is so often found perfectly smooth shows that the destruction of the tubercles cannot be due to fibrous transformation of the nodules, but must consist in some process which actually causes their complete solution and disappearance; in the same way a complete disappearance of the cause of the inflammatory changes in the peritoneum may be followed by a complete disappearance of all inflammatory processes.

According to the observations of Borchgrevink, a fibrillar intercellular substance arises in the young tubercle, which consists, first, of epithelioid cells only and resembles granulation tissue. The fibrillar intercellular substance first surrounds the tubercle in the form of a connective-tissue capsule, but gradually penetrates into the interior. In tubercles consisting entirely of epithelioid cells, giant-cells being absent, necrotic changes are quite wanting and healing is rapid. The disappearance of cells follows the formation of connective tissue. The rapidly shrinking tubercle soon forms a loose network of fine fibers; these are gradually absorbed, and the tubercle disappears, leaving no trace behind, in a short space of time.

The important question remains to be answered, What are the factors which determine the cure after an operation? A large number

of theories have been formulated, and Gatti has collected some twenty different factors which have been thought by different authors to be responsible for the good results of operative intervention. It is quite impossible to discuss all these factors here. The most probable and plausible explanation was or is that the operation acts as an irritant (or that some irritant is introduced when the abdomen is opened), which is capable of producing retrograde changes in the affected areas of the peritoneum. The cause of irritation is not necessarily always the same, but may depend on a number of physical or biologic factors, such as injury to the tissues *per se,* the entrance or the insufflation of atmospheric air, the entrance of light, of antiseptics, or of micro-organisms. Special stress has been laid upon the removal of the ascitic fluid. While this procedure is certainly of some importance, it must never be forgotten that tuberculous peritonitis without effusion may also be cured by laparotomy.

The above paragraphs give the prevalent view as held in 1898, when the first edition appeared. Since then a change has gradually taken place, and doubts arose as to the mystic curative effect of laparotomy. [Borchgrevink [1] and Saltykow,[2] from their experiments on animals, in whom the peritoneum had been infected with tubercle, came to the conclusion that the process of fibrosis and cure occurred in the same manner in animals left alone as in those operated upon. Saltykow insists on the absence of any rapid changes in peritoneal tuberculosis as a result of laparotomy.—Ed.] A. Frank (from Czerny's clinic) gives 38 per cent. of cures in 63 cases of operation, and 50 per cent. of cures in 8 not operated upon. Borchgrevink expresses himself distinctly on this point. Twenty-five of his cases had laparotomy performed, 22 not. Of the former, 3 must be deducted, so that the numbers are actually the same. Of the former (all with serous exudation), 14, or 63.6 per cent., were cured; of the latter (all with serous exudation), 19, or 81.8 per cent., so that cases not operated upon did much better.

[Much the same appears to be true in the case of children. Oehler [3] states that 50 per cent. of children with tuberculous peritonitis recover without operation. In 27 cases in children treated medically Sutherland [4] found that 22, or 81.3 per cent., recovered, 4, or 15 per cent. died, and 1, or 3.7 per cent., was unrelieved, while in 14 cases treated surgically, 7 recovered while 7 died. In 98 cases, chiefly in adults, on the other hand, Shattuck [5] found the ultimate mortality in the 52 cases treated surgically was 37.5 per cent., while in the remaining 46 cases treated medically it was 68 per cent.; but this higher mortality may be accounted for by the fact that the medical cases were nearly always complicated by tuberculosis in the lungs or in some other part of the body. Duckworth [6] speaks of tuberculous peritonitis as the form of tuberculosis in which the prognosis is most hopeful.—Ed.]

[1] Borchgrevink, *Mittheil. aus d. Grenzgeb. d. Med. u. Chir.,* 1900, vol. vi., p. 434.
[2] Saltykow, *Arch. de Méd. exper. et d'Anat. Path.,* 1903, vol. xv., p. 571.
[3] Oehler, *Münch. med. Wochenschr.,* December 25, 1901, p. 1823.
[4] G. A. Sutherland, *Clinical Jour.,* vol. xxi., p. 189.
[5] F. C. Shattuck, *Amer. Jour. Med. Sci.,* vol. cxxiv., p. 1.
[6] Sir Dyce Duckworth, *Clinical Jour.,* July 22, 1903, vol. xxii., p. 213.

Borchgrevink is convinced that tuberculous peritonitis, unaccompanied by much fever, runs a favorable course, and that it is more than probable that just those cases which gave laparotomy its importance would have been cured just as well without it. The tendency to spontaneous cure owes its origin to the fact that tubercles of the peritoneum are particularly poor in bacilli. Those cases only of laparotomy were cured in which the tubercles at the time of the operation were found far advanced in an effort at spontaneous cure; on the other hand, those cases of laparotomy met with absolutely no success in which, at the operation, this tendency was absent or extremely small.

U. Rose reports from Naunyn's clinic the result of cases not operated upon: Of 56 such, 34 died, 17 were cured, partially cured 2, not cured or slightly improved 3. Thus two-thirds of the cases died, one-third was cured, and among the cured were some extremely grave cases.

TREATMENT.

All the dietetic, internal, and external methods of treatment recommended in chronic exudative peritonitis of non-tuberculous origin have also been employed in the tuberculous form. Personally, I can confirm emphatically, in addition to rest in bed, Pribram's high opinion of the use of green soft soap. Though it is hard to decide whether its good effects are *post hoc* or *propter hoc,* I have seen both the exudate and the tumors disappear after its use. [Mercurial ointment as an application to the outside of the abdomen has long been employed, and has recently been highly spoken of by Duckworth.[1]—ED.]

[The importance of good hygienic conditions must be specially insisted on, and no patient should be kept in hospital if good food and better air can be obtained elsewhere (Shattuck[2]); the air of Margate, in England, is specially recommended for this and for other forms of tuberculosis.—ED.]

When the case does not recover spontaneously, or when all the ordinary external and internal remedies fail (and this presumably amounts to the same thing), surgical measures, particularly laparotomy, have been resorted to during the last few years. [Shattuck advises that if a patient does not improve in a month or six weeks under medical measures, surgical treatment should be adopted, and that this interval should be less if the patient loses ground.—ED.]

Since the first communications by Spencer Wells and König, a number of cases have been operated upon. The following results may be referred to, which have appeared in the voluminous literature on the subject: Hinterberger and Valenta, in Chrobak's clinic, operated on 38 cases (females) from 1887–96; of these, 21, or 55 per cent., were cured. Other reports are equally or even more favorable; thus, Mazzoni reports 35 cases, with 33 recoveries (94 per cent.); Dohrn, 12 with 11 (91 per cent.); Scheuer, 13 with 7 (53 per cent.); Frees' results are less favor-

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[1] Duckworth, *loc. cit.*
[2] F. C. Shattuck, *Amer. Jour. Med. Sci.,* July, 1902, vol. cxxiv., p. 1.

able—18 with 6 recoveries (33 per cent.). Roersch has collected a large number of cases—358 with 253 recoveries (71 per cent.); in König's earlier series there were 131 cases, with 84 recoveries (64 per cent.). Of all these cases, only a few ended unfavorably as a direct result of the operation, and in a considerable proportion, as stated above, recovery was shown to be complete histologically. In the majority of the cases, of course, this was impossible, but the latter could, at least, be regarded as clinically cured, inasmuch as all the symptoms and objective abdominal signs disappeared and recovery seemed to be permanent ; in one case no recurrence appeared for a period of eleven years (Czerny), during which the case remained under observation.

Some authors only consider cases fit for operation in which there is a fluid exudate, and particularly an encysted fluid exudate, while they regard cases in which the exudation is solid as unsuitable. Other writers again, and among them König, claim that all forms of the disease are suitable for operation—*i. e.*, the serous, the serofibrinous, and the purulent form, which is accompanied by the formation of thick fibrous layers of adhesive tissue.

So far as the operation itself is concerned, all authors seem to agree that incision is the proper method. When the abdomen is opened in this way the greater portion of the exudate is evacuated. Some authors—*e. g.*, Czerny—lay stress on the necessity of removing the primary focus in the abdomen (pyosalpinx, intestinal ulcers, mesenteric glands), whereas others do not consider this important; this is, however, a matter of surgical technic, and I shall not touch on this or on the other purely surgical questions of the operative treatment.

Simple puncture and removal of the exudate by aspiration, with or without the introduction of air, irrigation of the abdominal cavity with antiseptics, etc., are not followed by such good results as a laparotomy.

The operation is contraindicated probably only in cases of advanced tuberculosis of other organs, particularly of the lungs and intestine, and then only because death supervenes early in these cases in spite of the operation, for it cannot be said that the fatal issue is accelerated by the operation or that the disease tends to take an unfavorable turn after the laparotomy. Further, the operation is not contraindicated when there is fever or concomitant tuberculosis of the pleura or of the pericardium.

This view, generally adopted a few years ago, has been opposed in the last few years. Spontaneous cure of tuberculous peritonitis without any surgical intervention is constantly being reported, and Borchgrevink expresses himself very clearly against laparotomy. He has arrived at the following conclusion : " Cases with little or no fever run a favorable course if left alone ; laparotomy in these cases may be dispensed with ; in advancing cases, with constant fever, laparotomy is harmful. Laparotomy may, therefore, be discarded in all cases." Further observations can alone decide this question.

TUMORS OF THE PERITONEUM.

MALIGNANT NEW GROWTHS (Carcinoma; Sarcoma Peritonaei et Peritonitis Carcinomatosa).

PERITONEAL new growths are rare, but the malignant (carcinoma and sarcoma) are less infrequent than the innocent varieties. The disease is more often met with in the second half of life than in the first half. The opinion of the older writers that "carcinoma of the peritoneum" is commoner in women than in men is confirmed by recent statistics. Among Petrina's 40 cases 24 were women; in Mongird's (quoted by Courtois-Suffit) 28 cases, 18 were in women and 10 in men; in a series of 25 consecutive cases in my clinic there were only 2 men. The latter proportion is probably accidental, and the statistics available at present are not sufficiently numerous to justify any final conclusions. The preponderance of the female sex, however, is worthy of note.

The peritoneum is rarely the site of a primary growth, but metastatic tumors secondary to a malignant growth elsewhere are common. Thus among Bamberger's 14 cases there was only 1 case, and among Petrina's 40 cases only 14, of primary carcinoma (or sarcoma?) of the peritoneum. My personal experience coincides with these figures; in all the cases examined postmortem the peritoneum was secondarily involved, and nearly all the cases that left the clinic before autopsy presented clinical evidence that the peritoneal growths were secondary to a primary new growth in some other part of the body (mamma, stomach, intestine).

Carcinoma invades the peritoneum either by way of metastases from some distant organ or by direct extension from one of the viscera covered by peritoneum. The commonest situations for the primary growth are the stomach, pancreas, and colon, while the breast, rectum, and uterus are the starting-points in a number of cases; in several of my cases the gall-bladder was the organ primarily affected, but new growths in other situations may give rise to peritoneal metastases.

Leyden has found a peculiar form of rhizopod resembling an ameba in the fluid aspirated from the abdomen in a case of carcinomatous peritonitis; these bodies have been called Leydenia gemmipara by Schaudinn. It is not yet proved that these amebæ, which Schaudinn regards as undoubted, play any etiologic rôle in the genesis of cancer. In my clinic these amebæ have been found in a number of cases, and Lauenstein has found them in the ascitic fluid in carcinoma of the peritoneum.

Anatomy.—The malignant tumors of the peritoneum are the various histologic forms of sarcomata and carcinomata. Formerly, "carcinoma vulgare" was considered the most common form of primary tumor of the peritoneum; within the last decades the views on the subject have, however, changed somewhat. Thus, to quote Waldeyer, in 1872: "Whereas formerly—*i. e.*, before the appearance of Thiersch's work—primary carcinomata of the peritoneum and of the bones were described by reliable authors, no cases of this kind have been described within the last six years." This has held good ever since, and primary

carcinoma of the peritoneum is reported only in exceptional and rare instances, such as the cases published by Quincke (1875), Hervéon, and Hubl. Orth, in speaking of the latter description, however, says: "The characters of the tumor can hardly be considered conclusive evidence of its carcinomatous nature."

[The microscopic appearances of an endothelioma may be exactly the same as those of an ordinary carcinoma, and it may be possible to determine that the growth is an endothelioma only by cutting serial sections and thus seeing the origin of the tumor in or about lymphatics or other vessels. Thus, in 1890, the Morbid Growths Committee of the Pathological Society of London examined and described as carcinoma a primary tumor of the omentum with numerous metastatic growths in the abdominal cavity shown by West.[1] At the present time most primary malignant tumors of the peritoneum are regarded as endotheliomata.— Ed.]

The origin of carcinoma from the superficial endothelium of the peritoneum being doubtful, so-called endothelial tumors of the peritoneal lymph-vessels or lymph-spaces have been described. The exact histologic position of these "endotheliomata" is by no means clear ; they have been considered infective and have also been included among the carcinomata and sarcomata. According to Glockner, who has analyzed the whole literature on the subject up to date (1895), this form of tumor is by no means rare. I have myself seen of late years two cases in which the peritoneum was primarily and exclusively affected.

Though rare, sarcoma occasionally occurs as a primary growth of the peritoneum ; it may be a spindle-cell, round-cell, alveolar, or cystic myxosarcoma, lymphosarcoma, plexiform sarcoma, angiosarcoma, or fibrosarcoma. The so-called gelatinous carcinoma described by Rokitansky as a primary peritoneal tumor belongs to the group of sarcomata.

Primary sarcomata may reach an enormous size and form tumors as big as an adult's head. Hedenius has described a gigantic "gelatinous carcinoma" which filled the whole of the abdomen and pelvis, and was 75 cm. long, 46 cm. broad, and 124 cm. in circumference. These tumors most commonly start in the mesentery and omentum.

[Intraperitoneal tumors commonly start as retroperitoneal growths, and it is, therefore, necessary to draw attention to retroperitoneal sarcomata, endotheliomata, etc. Retroperitoneal sarcoma is a rare disease, as shown by the fact that among 894 sarcomata out of 14,630 tumors examined at Vienna, there was only one sarcoma of the retroperitoneal space (Gurlt). Dutton Steele[2] has analyzed 61 cases of retroperitoneal sarcoma, and finds that it occurs most often in the first, fourth, and sixth decades of life. Males are rather more often (60 per cent.) affected than females (40 per cent.). In 57 per cent. of Steele's cases the growth arose laterally in the lumbar region, the right side of the body being more often affected than the left, while in 41 per cent. the growth arose in the middle line from the posterior wall of the abdo-

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[1] S. West, *Trans. Path. Soc.*, vol. xli., p. 95.
[2] J. Dutton Steele, *Amer. Jour. Med. Sci.*, March, 1900, vol. cxix., p. 311.

men near the attachment of the mesentery, and in 2 per cent. from the pelvis. The tumors may start from the retroperitoneal lymphatic glands, connective tissue around the vessels, and from the remains of the Wolffian body,[1] and may be so close to the pancreas as to suggest a pancreatic origin, as in Watkins Pitchford's [2] case. The growths, which are usually lobulated, are very prone to degeneration, usually combined with hemorrhage, and in 35 per cent. of Steele's cases went on to the formation of pseudocysts. In a perirenal retroperitoneal sarcoma described by Rolleston and Turner [3] the pseudocyst opened into the second part of the duodenum. Retroperitoneal tumors must, of course, be distinguished from tumors definitely arising from the suprarenals and kidneys.—Ed.]

In acute general carcinomatosis the growth occasionally appears in the form of miliary nodules, scattered in great numbers all over the peritoneum, and at first sight greatly resembling miliary tubercles. The carcinomatous nodules are, however, as a rule, rather larger, not so gray and translucent, but of a more whitish hue, and show no tendency to caseation. As in acute miliary tuberculosis, there is no inflammation of the peritoneum.

In the great majority of the cases carcinomatosis of the peritoneum is accompanied by evidence of inflammation (carcinomatous peritonitis); this inflammation in many respects resembles that in tuberculous peritonitis; in both forms there are definite tumors, a tendency on the part of the mesentery and the omentum to retract and become adherent, encapsulation of coils of intestine and the formation of an exudate which is often hemorrhagic.

The peritoneum may either be locally injected around the small nodules, or more commonly diffusely inflamed and covered with flakes of fibrin and numerous hemorrhagic spots. In addition, there are widely disseminated hard white nodules, varying in size from that of a pin's head to that of a pea, or even of a walnut; some of the smaller nodules also show a depression in the center, resembling the umbilication of a small-pox pustule. The peritoneum that does not cover the nodules presents a peculiar reticulated appearance. In other cases the carcinomatous masses form flat plaques, either thin or thick; or, again, they may form tumors as large as a man's fist, with a nodular surface. In advanced cases these tumors cover the whole parietal and visceral peritoneum, and form hard masses on the surface of the liver, the spleen, and the intestine, and produce considerable deformity of these viscera. The tumors have a special tendency, as in tuberculosis, to develop in the mesentery, which becomes retracted and shortened, and anchors the intestine to the spinal column. The omentum is similarly affected, and is often converted into a thick, round or flat tumor with a number of projecting nodules of varying size. The mesenteric and retroperitoneal glands also become infiltrated.

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[1] See F. Craven Moore.
[2] W. Watkins Pitchford, *Brit. Med. Jour.*, 1902, vol. i., p. 1087.
[3] H. D. Rolleston and G. R. Turner, *Lancet*, 1901, vol. i., p. 1273.

In some cases the lymph-vessels are filled with a white, opaque growth and stand out prominently on the peritoneum; the material filling the lymph-channels consists of epithelial cells imbedded in lymphatic (adenoid) tissue (Waldeyer's "carcinoma lymphaticum").

In other cases, again, the peritoneum is uniformly thickened and opaque over large continuous areas. The resulting adhesions between the viscera and the parietal peritoneum are, of course, subject to considerable variation.

This form of peritonitis is rarely dry and adhesive, and without any trace of exudate. The exudate is usually encysted, seldom free, and often consists of clear yellowish serum containing a few flakes of fibrin; it is rarely purulent. In other cases—and more frequently than in tuberculous peritonitis—it is hemorrhagic; occasionally it is slightly brown in color, at other times decidedly blood-stained. A peculiarity that is specially common in carcinomatosis of the peritoneum is the chylous or chyliform character of the ascitic fluid; in these cases the milky white and non-translucent appearance of the exudate is due to the presence of fat-globules. Quincke, Brieger, and others have called attention to the fact that this form of ascites may occur in carcinoma of the peritoneum, and I have seen this form of ascites which may be either genuinely chylous or merely "chyliform or fatty" in carcinomatous peritonitis (the details of this form of ascites were given in the section on Ascites).

Clinical Features.—In describing the clinical aspects of carcinomatous peritonitis it is unnecessary to go into detail, as this would entail the repetition of practically all that has already been said about the clinical features of tuberculous peritonitis. There is a great clinical resemblance between the two affections as regards the physical signs, the symptoms, and the characters of the tumors. The two diseases may be so much alike that it is often impossible to make a differential diagnosis between them. Pain, usually slight or completely absent, vomiting, intestinal disturbance, hiccup, and meteorism may be exactly the same in both. This also applies to the exudate, which may be absent, very scanty, or very profuse, may be freely movable or encysted, and may show Thomayer's sign—viz., a tympanitic note on percussion which is more widely distributed over the right side of the abdomen than over the left. Even the quality of the exudate is generally the same in both diseases, with possibly this difference, that in carcinomatous peritonitis the milky (chylous, fatty) and hemorrhagic exudates are somewhat more common than in the tuberculous form. The similarity in the clinical features is also shared by every detail of the tumors, such as the impression they give on palpation, their form, and their distribution; thus in both conditions the omentum is often felt as a thick, round, hard cord, running obliquely or transversely across the upper part of the abdomen. It is true that in carcinoma the tumors are more numerous, and, as a rule, larger in size. On the other hand, there may be no palpable tumors, either because they are too small or because they are hidden deep down in the abdomen. Lastly, all the

sequelæ which may involve the intestine, the common bile-duct, and other organs, from the formation of fibrinous deposits or from adhesions (compression and kinking of the bowel, icterus, etc.), or that may follow destruction of tissues (perforation), are seen as frequently in the one disease as in the other.

As the two diseases are so much alike, it is unnecessary to go over again all the clinical details already given in the section on Peritoneal Tuberculosis, and I shall, therefore, confine my remarks here to a few points in which these two diseases differ.

Tubercle bacilli and carcinomatous cells respectively are occasionally found in the ascitic fluid removed by aspiration in these two conditions. Quincke says that these cells may be either single or in groups, in a state of activity or degenerated. They are often vacuolated, and may be converted into large hyaline spheres consisting of a watery, albuminoid substance (hydropic degeneration—Quincke), or show granular or even fatty metamorphosis; when a large number of the cells contain fat, the exudate becomes milky. The cancer-cells can usually be distinguished from endothelial cells of the peritoneum by their size, number, and by the fact that they are arranged in spheric groups (also glycogen reaction?), and the diagnosis can, therefore, be occasionally made by the microscopic examination of the ascitic fluid.

[In discussing the diagnostic value of the character of the cells in effusions into serous cavities due to carcinoma, Dock [1] pointed out that while absolute reliance cannot be attached to their characters, there are more cells showing mitosis than in simple or tuberculous effusions, and that the mitoses are often atypical.—ED.]

There are also some differences in the temperature, though they are not fundamental or constant; in malignant disease the temperature is normal or even subnormal, while there is usually some degree of fever in tuberculosis. Fever, however, may sometimes occur in malignant disease of the peritoneum, and the temperature may be as high as 104° F.; this may occur not only in acute miliary carcinomatosis, but also when the peritoneum is secondarily involved by direct extension of the growth from some adjacent organ. The cachexia, due to the primary disease, is usually profound. When the course of the disease is prolonged, secondary complications in other organs sometimes develop, especially malignant growths of the pleura, which comparatively often involves both sides.

The clinical manifestations of primary peritoneal sarcoma (or endothelioma) are vague for a long time. Gradually the indefinite initial signs and symptoms—usually gastric or intestinal—are succeeded by more definite evidence that the peritoneum is affected, but it is often as difficult to make out that disease of the peritoneum is malignant as it is in secondary carcinoma in which the seat of the primary cancer is latent; or, again, there may be only one tumor, which may easily be mistaken for an innocent growth.

General acute carcinomatosis of the peritoneum (I have never seen

[1] G. Dock, *Amer. Jour. Med. Sci.*, June, 1897.

such a case) is said to imitate an acute infection with more or less fever, delirium, coma, and absence of local peritoneal symptoms ; for death is stated to occur before inflammatory changes in the peritoneum have had time to develop, since nothing is found postmortem but an eruption of miliary cancer nodules.

The **diagnosis** is possible when peritoneal symptoms appear in an individual suffering from cancer of some organ, in whom there is no other cause—*e. g.*, tuberculosis—for the peritoneal manifestations. The diagnosis, however, is difficult when no primary focus can be found. Sometimes indurated glands in the neck or in the inguinal region furnish a clue ; when no glands are palpable, the course of the disease sometimes settles the diagnosis. Fever and remissions in the course of the disease are in favor of tuberculosis. Aspiration of some of the fluid sometimes decides the diagnosis by showing the presence of tubercle bacilli or of cancer-cells. The exudate is more often hemorrhagic or milky, and collects again more rapidly in malignant disease than in tuberculosis, and the appearance of cachexia, when the other organs are healthy, is of itself in favor of malignant disease.

Except in acute miliary carcinomatosis, the course of malignant peritonitis, whether it is—as it usually is—secondary or is one of the rare cases of primary sarcomatosis, is, as a rule, chronic, with an insidious onset and indefinite symptoms. In exceptional instances the disease comes on rapidly with fever ; this occurred in a case under my care of cancer of the stomach without perforation in which the disease spread all over the peritoneum ; the spread of the disease in this instance could be followed by the increase of the pain and the formation of a fluid exudate. There are no remissions and no arrest in the course of the disease, as in tuberculosis ; the process goes on without interruption until death occurs. No case of recovery is known, all cases ending fatally.

[Ten years ago the late Professor Greig Smith[1] described cases of "the (so-called) spontaneous disappearance of solid abdominal tumors which presented the following leading features : first, the presence of a visible and palpable solid tumor of the abdomen ; secondly, total absence of signs of inflammation, local or general ; thirdly, diagnosis of malignancy after inspection during exploratory operation ; and, fourthly, complete disappearance of the tumor, with restoration of the patient's health." It is possible that these tumors may have been due to the presence in the peritoneal cavity of foreign bodies which have worked their way out of the alimentary canal (Bland Sutton[2]). Shattock[3] described a lamellar fibroma, $2\frac{1}{2}$ inches in diameter, which lay between the bladder and rectum, in the subperitoneal tissue, and so loosely connected with the outer surface of the latter that it might readily have been torn off ; it contained an eroded piece of iron.—ED.]

The **treatment** is symptomatic, since nothing can be done for the

[1] J. Greig Smith, *Medico-Chir. Trans.*, 1894, vol. lxxvii., p. 139.
[2] J. Bland Sutton, *Lancet*, 1903, vol. ii., p. 1148.
[3] S. G. Shattock, *Trans. Path. Soc.*, 1893, vol. xliv., p. 151.

disease itself, and consists in compresses, morphin for the pain, and, when necessary, regulation of the bowels. When the ascites is distressing and interferes with respiration the abdomen should be tapped, and this may be repeated as often as is necessary, especially when the exudate is serous; but when it is hemorrhagic, it is advisable not to tap too frequently. Surgical intervention is useless. It need hardly be pointed out that the diet should be generous and appropriate.

The only form of the disease that can be treated surgically, at least as far as the prolongation of life is concerned, is when there is a single sarcomatous growth of the peritoneum. Cases reported by Czerny, Braun and others show that occasionally the immediate effect of removal of omental tumors may be favorable; recurrence, however, always occurs. [Pearce Gould [1] removed a hemorrhagic spindle-celled sarcoma weighing 21 pounds from the gastrohepatic omentum; the patient was in good health four years later.—ED.] The majority of surgeons absolutely condemn surgical interference in sarcoma of the mesentery.

INNOCENT TUMORS.

A large number of innocent tumors of the peritoneum have been recorded, although they are, absolutely speaking, rare. Lipomata, fibromata, myxomata, and pseudomyxomata, angiomata, lymphomata, neuromata, teratomata, and cysts of various kinds have been observed. In addition, parasitic cysts, especially hydatid and cysticercus cellulosæ, have been reported. Goebell gives the literature of the subject.

Anatomy.—Innocent tumors occur in any part of the peritoneum, but more especially, as is well known, in the omentum and mesentery. They may be either single or multiple, and in the latter case may be widely distributed.

The omental tumors are only pseudo-omental—*i. e.*, although situated intra-omentally, they originate, in reality, in some other organ. Tumors of the stomach and transverse colon, and especially myomata, insert themselves between the layers of the omentum. They (myomata) grow to the size of a child's or a man's head, and are connected with their seat of origin by a very thin stalk. In an account of a case Borrmann has dealt fully with the literature.

Hydatid disease is relatively rare as a primary lesion of the peritoneum. In these rare cases there is a single cyst which is usually in the omentum or mesentery. Hydatid infection of the peritoneum is far more often secondary, and is then multiple; there are often enormous numbers of cysts scattered over the whole omentum, mesentery, and the visceral peritoneum covering the stomach, intestine, diaphragm, and pelvic organs.

[Potherat [1] has put forward the view that multiple hydatid cysts in the peritoneum are primary, and not due to rupture of a large hydatid cyst in the liver or elsewhere. But there is ample proof that infection

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[1] A. Pearce Gould, *Medico-Chir. Trans.*, vol. lxxxiii., p. 257.
[2] Potherat, *Bull. et Mem. Soc. de Chirurg.*, 1900.

of the peritoneal cavity with secondary cysts does occur after rupture of a large primary cyst; it appears to take about two years before the secondary cysts are sufficiently large to give rise to signs and symptoms; the omentum and pelvis are the most frequent sites of the secondary cysts. It is a somewhat remarkable fact that secondary infection of the peritoneum may occur when the original cyst is in free communication with a bile-duct, and that scolices will continue to grow in equal parts of hydatid fluid and bile (Dévé[1]). *Pseudotuberculosis of the peritoneum* (Dévé[2]) after rupture or leakage of a hydatid cyst consists in small granulomata, covered by the endothelium of the peritoneum and containing pieces of hydatid membrane or hooklets. Histologically, they resemble tubercles in the presence of giant-, endothelioid, and small round-cells, but there are no tubercle bacilli. The process may be regarded as an attempt to absorb the fragments of hydatid membrane and hooklets.—ED.]

The **Cysticercus cellulosæ** is rare; it is found in the subserous tissues, particularly in the mesentery, and sometimes in enormous numbers. These cysts are very small, and produce hardly any clinical symptoms.

The **lipomata** may be either pure fatty tumors or fibrolipomata; some of the cases show calcareous infiltration, and others the characters of myxolipomata, and then approach the malignant tumors, with a tendency to metastases; they have been repeatedly described in the subperitoneal tissues of the anterior abdominal wall; they usually project into the abdominal cavity, and vary in size from that of an orange to such enormous dimensions that they imitate ascites (Canvy). Large and small lipomata may also be present in the omentum, and when in the mesentery are of some clinical interest. According to Terrilon, most of the mesenteric lipomata start from the iliac fossa or the renal region, usually on the right side. They may grow to an enormous size and fill the whole of the abdomen; tumors 60 cm. long and 40 cm. broad and weighing $31\frac{1}{2}$ kilos have been described (Waldeyer, "myxoma lipomatosum"). When on the right side, the ascending colon is usually carried in front of them, and on the left, the descending colon; they may grow around and inclose the transverse colon.

[Adami[3] collected 42 of these retroperitoneal lipomata in 1897, and found that the female sex is most often affected, and that the disease usually occurs in middle or late life. In a third of the cases the lipoma arises from the fat in the perirenal region. The rate of growth is slow, and symptoms do not appear for a long time, but from fluctuation, ascites is very likely to be diagnosed; later on edema of the legs, dyspnea, and emaciation appear, and may suggest malignant disease. In most of the recorded cases the weight is over 20 pounds. In Cooper Forster's[4] case it weighed 55 pounds. Of Adami's 42 cases, the tumor was removed partially or wholly in 26, with recovery in 12—a per-

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[1] Dévé, *Soc. de Biol.*, January 17, 1903. [2] *Ibid., Rev. de Chir.*, July, 1902, p. 79.
[3] J. G. Adami, *Montreal Med. Jour.*, January and February, 1897.
[4] Cooper Forster, *Trans. Path. Soc.*, vol. xix., p. 246.

centage of 46.1, but in one of these it recurred. Anderson [1] divided the abdominal lipomata into three groups : (1) The retroperitoneal, which may become mesenteric, mesocolic, omental, or parametric. Treves [2] described a lipoma of the broad ligament, but was only able to refer to one other case—Peyrot's [3]). (2) Hernial, through inguinal, femoral, or obturator openings, through the linea alba, and in other situations. (3) Intraperitoneal, after the manner of appendices epiploicæ.

Adami and Gardner [4] have reported a remarkable case with two retroperitoneal tumors, one a lipoma myxomatodes, the other a chondro-myxofibroma.—ED.]

Fibromata of the peritoneum are rare ; they must be distinguished from so-called fibrous peritonitis, in which the peritoneum is uniformly covered with fibrous tissue overgrowths. Numerous small fibrous tumors may develop on the intestinal or parietal peritoneum, without any inflammation. The larger fibromata may be as big as an adult fist or bigger ; they may start from the omentum, the mesentery, and very commonly from the pelvic organs.

[Lexer [5] removed a fibroma weighing 5 pounds from the mesentery of a man aged forty-one, who had suffered from colic for some weeks. The combination of fibrous and fatty tissues in fibrolipomata has been already referred to ; fibromyomata may occur. Anderson [6] quotes a remarkable case of Professor Saneyosthi's, in which there were 21 fibromyomata, the largest weighing over 7 pounds ; one weighing at least 9 pounds was successfully removed by Sheild,[7] which may have arisen from the root of the mesentery (Eve [8]). It is, of course, possible that fibromyomata of the broad ligament, or pedunculated fibromyomata of the uterus may become loose in the peritoneal cavity and contract secondary adhesions elsewhere.

Intraperitoneal fibrous tumors must be distinguished from the remarkable *fibrous desmoid tumors of the abdominal wall* which arise from the sheath of the rectus, possibly from tendons (Doran [9]), and, though it is not very probable, from the round ligament. In 100 connective-tissue tumors of the abdominal wall collected by Staveley,[10] at least 79 occurred in women ; the condition has, therefore, chiefly come under the notice of gynecologists. Olshausen,[11] who has met with 22 cases in twelve years, states that after removal, other tumors may spring up in the abdominal wall, usually at a distance from the site of the first. This is not an indication that they are malignant, though they often have been erroneously diagnosed as sarcomata, but merely a manifestation of the tendency which innocent tumors have to be multiple.—ED.]

[1] W. Anderson, *Brit. Med. Jour.*, 1896, vol. ii., p. 1087.
[2] F. Treves, *Trans. Clin. Soc.*, 1893, vol. xxvi., p. 101.
[3] Peyrot, *Bull. Soc. Anat.*, Paris, 1875, p. 178.
[4] Adami and Gardner, *Montreal Med. Jour.*, June, 1900, p. 417.
[5] Lexer, *Berlin. klin. Wochenschr.*, January 1, 1900.   [6] W. Anderson, *loc. cit.*
[7] A. M. Sheild, *Medico-Chir. Trans.*, vol. lxxx., p. 205 ; *Brit. Med. Jour.*, 1897, vol. i., p. 655.   [8] F. Eve, *Brit. Med. Jour.*, 1897, vol. i., p. 655.
[9] A. Doran, *Trans. Med. Soc.*, London, vol. xiv., p. 337.
[10] Staveley, *Phila. Med. Jour.*, March 17, 1900, vol. v., p. 638.
[11] Olshausen, *Zeitschr. f. Geb. u. Gynäk.*, vol. xli.

Cystic tumors of the peritoneum, apart from hydatids, are relatively common. Their starting-point, mode of origin, and situation may vary. Occasionally they are found in the omentum, but most frequently in the mesentery (according to Augagneur, 30 out of 90 mesenteric tumors were cystic); these mesenteric cysts have been the subject of a good deal of operative and literary work in the last decades; in addition to a number of recorded cases, the following authors have written specially important papers: Augagneur, Bramann, Hahn, Frentzel, Hochenegg, Pagenstecher, and others.

Hahn's classification of mesenteric cysts according to their contents is into—serous, chylous, hemorrhagic, and hydatid cysts. In addition, dermoid cysts are found not only in the omentum, but also in the mesentery—this was formerly doubted.

[Dowd,[1] who has collected 145 cases of mesenteric cysts, groups them into three classes: (1) Embryonic cysts, including dermoid, chylous, and serous cysts; (2) hydatid; (3) cystic malignant disease.—ED.]

The most interesting are the *chylous cysts.* The contents are milky or creamy, sometimes snow-white, at other times grayish, either thin or pultaceous, and resembling coagulated milk or a suspension of chalk. By chemic and microscopic examination they are shown to contain albumin, fat, margarin, and cholesterin crystals. The contents are generally agreed to be chylous. The wall of the cystoma is characterized by the absence of endothelium, but in exceptional cases some parts of the cyst-wall have been described as having been lined with endothelium; Weichselbaum, for instance, recorded a case in which the tumor contained several communicating cavities, in the smallest of which some endothelium was found. These chylous cysts are probably formed by retention of chyle in the lacteals or in the receptaculum chyli. Rokitansky's view, which Virchow considered as possible, was that they originated from lymph-glands; Pagenstecher has recently described a case in which he regarded the process, that eventually led to the conversion of a lymph-gland into a cyst, as a chronic inflammatory hyperplasia with hyaline degeneration and softening, presumably tuberculous in character.

[Dowd and Fawcett[2] regard chylous cysts as embryonic. The cyst-wall in Fawcett's case, like one of Eve's,[3] contained unstriped muscular fibers, and he regards these cysts as sequestrations from the intestine or its neighborhood, some from mesoblastic tissue with fibrous and muscular walls or both; others from the hypoblast as well, and showing epithelial structures.—ED.]

These chylous cysts are usually found in the mesentery, but they may occur in the mesocolon or mesosigmoid.

Blood cysts usually originate from traumatic hematomata, and contain a brownish or brown-red fluid; in other cases they are chylous cysts,

[1] C. Dowd, *Annals of Surgery*, October, 1900, vol. xxxii., p. 515.
[2] J. Fawcett, *Trans. Path. Soc.*, vol. liii., p. 406.
[3] F. Eve, *Medico-Chir. Trans.*, vol. lxxxi., p. 52.

containing sanguinolent material, or they may possibly (Virchow) be derived from lipomata by a process of softening.

[T. Fisher [1] has collected a number of cases of sanguineous peritoneal cysts ; they are allied to some peripancreatic cysts.—ED.]

The *serous cysts* with greenish contents have received the least attention, and little can, therefore, be said about their pathogenesis. Possibly some of them were originally true hydatid cysts. A case of Tuffier's (quoted by Pagenstecher) would appear to show that occasionally serous cysts may develop from chylous cysts, as the cyst was divided into three compartments, in two of which there was chylous fluid, while the third contained serous fluid. It is also conceivable that an encysted intraperitoneal abscess may become transformed into a cyst (Quainson).

[*Multiple Cystic Disease of the Omentum.*—Two exceptional cases of this remarkable condition have been described by Harrison Cripps,[2] and J. Berry.[3] In both cases there were large numbers of cysts containing clear fluid, which were conjectured to be due to lymphatic obstruction. A cyst attached by a delicate pedicle to the cecum was regarded by MacDonald [4] as due to degenerative changes in one of the appendices epiploicæ.—ED.]

Dermoid cysts containing hair have been described. König's case was in the mesentery, while the one recorded by Bonfigli was in the omentum, and contained mucilaginous material, a few hairs, and a piece of bone with two teeth. [Launay [5] removed a dermoid cyst from the transverse colon of a man aged thirty-six years. Multiple dermoid cysts in the abdominal cavity have been recorded by Kolaczek,[6] Fraenkel,[7] Hulke,[8] and Latham.[9] The multiplicity may be due to dermoid cysts, or embryomata, which, as Wilms [10] has shown, are really teratomata, becoming malignant. Montgomery [11] has collected 10 cases of malignant teratomata. On the other hand, multiple abdominal "dermoids" may be innocent implantations of fragments of a ruptured ovarian "dermoid" on the peritoneum, in the same way that secondary peritoneal implantations occur in ovarian papillomata ; this interpretation may be applied to Hulke's and Latham's cases.—ED.]

Heinrichs has described a cyst in the ascending mesocolon with a remarkable origin, being an encysted Treitz hernia of the subcecal fossa ; this cyst was lined with endothelium over the whole of its internal surface, and in this respect differed from all the other recorded cases.

[F. Craven Moore [12] has written a valuable paper on " Peritoneal

[1] T. Fisher, *Guy's Hosp. Rep.*, vol. xlix.
[2] W. Harrison Cripps, *Trans. Path. Soc.*, vol. xlviii., p. 85.
[3] James Berry, *ibid.*, vol. xlviii., p. 105.
[4] G. C. MacDonald, *American Medicine*, April 20, 1901, p. 123.
[5] Launay and Potherat, *Bull. et Mem. Soc. Chir.*, Paris, October 14, 1903, p. 923.
[6] Kolaczek, *Virchow's Arch.*, vol. lxxv., p. 39.
[7] Fraenkel, *Wien. med. Wochenschr.*, 1883.
[8] J. W. Hulke, *Trans. Path. Soc.*, vol. xxiv., p. 157.
[9] A. Latham, *ibid.*, vol. l., p. 232.
[10] Wilms, *Deutsch. Arch. f. klin. Med.*, vol. lv., p. 289.
[11] D. W. Montgomery, *Jour. Exper. Med.*, May, 1898, vol. iii., p. 259.
[12] F. Craven Moore, *Medical Chronicle*, February, 1903.

Multilocular Cystadenomata of Retroperitoneal Origin." They are the rarest of the cysts commonly included under the general headings of mesenteric or omental cysts, and only about five are on record. Structurally, they resemble the ordinary multilocular cystadenoma of the ovary. The cases recorded by Moore and by Brown and Brady were in men. The cyst may present comparatively simple anatomic relations and no very great complexity of structure, consisting of a single, large, thick-walled cyst with a variable number of smaller and thin-walled cysts attached to its inner surface (cases of MacDonald [1] and Dowd [2]). On the other hand, there may be a much more complex structure and a capacity for progressive new growth which endow it with the features of malignancy. In Brown and Brady's [3] case, described as gelatinous carcinoma of the peritoneum, there were metastatic growths in the left lung and sternum. In Moore's case the growth had remained limited to the intra-abdominal subserous tissues. The probable origin of these tumors is from some portion of the Wolffian body which has become displaced during the process of development.—ED.]

The size of the mesenteric and omental cysts varies within wide limits; they may not be larger than an orange, or may contain several liters of fluid; the majority contain between $\frac{1}{2}$ and 3 liters, and are of moderate size.

Myxomata are very rare. Werth has described cysts under the name of pseudomyxomata of the peritoneum, in which the abdominal cavity was filled with gelatinous masses attached to the peritoneum and traversed by blood-vessels and strands of connective tissue, so that the whole mass seemed to consist of small cavities. He regarded this condition as the result of peritonitis produced by foreign bodies, and caused by the entrance of jelly-like masses into the peritoneum after spontaneous rupture of an ovarian cyst.

Angiomata and **enchondromata** are rarities.

[Arbuthnot Lane [4] successfully removed an extensive nevus from the peritoneum of a boy aged seven years.—ED.]

All these tumors exert mechanical pressure on neighboring parts, and may thus lead to the formation of chronic inflammatory adhesions between them and the intestine, omentum, etc. These adhesions are occasionally very dense, and must, from a pathologic point of view, be regarded as the result of "mechanical" peritonitis. (See Wieland's Experiments, p. 729.)

Clinical Features.—The clinical picture varies according to the size, anatomic structure, and position of the tumors, and, of course, is different when there is only a single tumor or when there are several. The clinical manifestations may, of course, vary greatly, and some idea may be obtained of the difficulty of diagnosis from the fact that mesenteric cysts have been regarded as carcinoma, ovarian cyst, floating kid-

[1] MacDonald, *Edinburgh Med. Jour.*, 1885, vol. xxx., p. 1074.
[2] C. Dowd, *Annals of Surgery*, October, 1900, vol, xxxii., p. 515.
[3] Brown and Brady, *New York Med. Jour.*, January 4, 1902, p. 9.
[4] W. Arbuthnot Lane, *Trans. Clin. Soc.*, vol. xxvi., p. 5.

ney, intestinal obstruction, and intussusception ; and that ascites has been diagnosed when the actual condition was a subperitoneal lipoma.

Occasionally there are indefinite symptoms, such as vomiting, constipation, and pain, before the diagnosis is settled by the discovery of a tumor. The diagnosis must hinge on the examination and interpretation of the tumor. The tumor may be firm and solid (fibroma, lipoma, myxoma, carcinoma) or soft and fluctuating (cyst). It must be remembered, however, that lipomata and myxomata, and even soft carcinomata, occasionally fluctuate, and that, conversely, cysts are sometimes hard and solid to the touch, either because their capsule is tense or because the contents of the cyst, as in chylous cysts, are really pultaceous.

Attention has recently been mainly devoted to mesenteric cysts, and such a large number of cases are now on record that a definite clinical picture of the disease is possible, and has been given by Augagneur, Frentzel, Hochenegg, and others.

A mesenteric cyst usually appears as a tumor about as large as a child's head ; sometimes they are smaller and often bigger, and their size may be said to vary from that of an apple to that of an adult head. The tumor is generally round or globular, and perfectly smooth on the surface, which usually fluctuates or is tense and elastic, and in exceptional cases apparently hard and solid. It may either be tender on pressure or not. The position and the motility of the tumor are important : in the majority of cases it is well to the right of and a little below the umbilicus, and is, therefore, in the right lower quadrant of the abdomen ; it is only in rare cases that the tumor extends beyond the middle line to the left or above the level of the umbilicus in an upward direction. This position corresponds to the most common situation of the cysts in the mesentery of the small intestine. In rare cases, however, where the cyst is in the mesocolon of the sigmoid flexure, the tumor is found in the lower left half of the abdomen ; or when it arises in the mesentery of the jejunum near the duodenum, the tumor lies in the epigastrium (Rotter).

[Morton [1] has described a cyst of the mesentery which developed in a hernial sac and led to an irreducible condition of the hernia.—Ed.]

The tumor is usually extremely movable, though it may only move very slightly or not at all with respiration. But the tumor is apt to change its position with every movement of the patient, and to occupy a different position in the abdomen when the patient is standing up, lying down, or leaning forward. The tumor can also be readily displaced and moved about by the hand ; it can be forced downward nearly to the symphysis pubis, or upward under the margin of the ribs until it disappears ; it can be pushed to the left or to the right, but cannot, like a floating kidney, be displaced into the loin. Sometimes, however, the tumor is movable only in one direction,—for example, vertically,—while tumors in the mesocolon and large cysts of the duodenal mesentery may not be movable at all. The excessive motility of most mesenteric cysts distinguishes them from pancreatic cysts, retroperitoneal

[1] C. A. Morton, *Lancet*, 1896, vol. ii., p. 1678.

tumors, and tumors of the uterus and uterine appendages; lastly, the fact that they cannot be pushed into the loin distinguishes them from floating kidney. Brentano has called attention to the fact that cysts in the mesentery of the small intestine are surrounded by the colon, and that this can be shown by inflating the colon with air. The percussion-note over the cysts varies in intensity and character, and depends entirely on the degree of flatulent distention and on the amount of solid material in the small intestine.

In addition to these physical signs, there are a number of symptoms, of which the most important is pain. This is rarely continuous, but almost invariably colicky in character (sometimes spoken of as "cramps in the stomach"), and occasionally of "terrific" violence. The pain may be accompanied by vomiting, constipation, meteorism, and may come on suddenly while the patient is apparently in perfect health, and may, therefore, imitate intestinal obstruction, which has, in fact, been diagnosed on several occasions. In other cases the intensity and the frequency of the attacks of pain gradually increase, and when the abdomen is examined in these cases, a tumor will often be felt. The cause of this pain has been explained in different ways. Frentzel is inclined to attribute it to sudden intermittent enlargement of the tumor; Hochenegg believes that it is due to displacement of the heavy tumor and dragging, kinking, or twisting of the bowel and mesentery which are thus produced. The constant pain and tenderness on pressure are due either to the tension of the cyst-wall or to adhesive peritonitis. There is nothing characteristic about the constipation.

Witzel has given an account of the clinical manifestations of omental tumors which is surprisingly like the above description. There are the same peculiar attacks of pain radiating from the tumor, and due, according to him, to traction exerted by the heavy tumor on the omentum of the transverse colon and the stomach. The tumor is also in the neighborhood of the umbilicus, and when not too large, possesses considerable motility in all directions—least toward the pelvis. If the tumor is a cyst,—*e. g.*, a hydatid,—it may be impossible to differentiate it from a tumor of the mesentery, just as it is impossible to distinguish between a lipoma of the omentum and one of the mesentery. In fact, the attempt to diagnose a tumor of the omentum from one of the mesentery usually has to be abandoned.

All the diagnostic considerations given above fail in the case of a large tumor or when the tumor is fixed by peritoneal adhesions. It is then often impossible, in spite of the most minute care, to make out the origin or the nature of the doubtful tumor, and exploratory puncture has been advised. The majority of surgeons, however, are opposed to this procedure, particularly in small tumors, because there is always danger of injuring the bowel. An exploratory laparotomy may, however, be advisable, followed eventually by a further operation.

Primary malignant tumors of the peritoneum, whether omental or mesenteric, usually become fixed by peritoneal adhesions early in their course.

It is obvious that the **treatment** of all forms of peritoneal tumors can only be surgical.

BIBLIOGRAPHY.

Abercrombie, "Untersuchungen über die Krankheiten des Magens, des Darmcanals, u. s. w.," trans. by v. d. Busch, Bremen, 1843.

Bamberger, "Krankheiten des chylopoetischen Systems," Virchow's Handb. d. spec. Path., 1864, vol. vi., second ed.

Bauer, "Krankheiten des Peritoneum," v. Ziemssen's Handb. d. spec. Pathol. u. Therap., 1878, Leipsic, second ed., vol. viii.

Boas, Diagnostik und Therapie der Darmkrankheiten, Leipsic, 1899.

Courtois-Suffit, "Maladies de l'intestin et du peritoine," Traité de méd., Paris, 1892, vol. iii.

Damaschino, Maladies des voies digestives, Paris, 1880.

Einhorn, Die Krankheiten des Darms, Berlin, 1901.

Ewald, "Diseases of the Intestines," Twentieth Century Practice of Med., New York, vol. ix.

Ewald, Die Krankheiten des Darms und des Bauchfells, Berlin, 1902.

Habershon, Diseases of the Abdomen, London, 1878.

Hemmeter, Diseases of the Intestines, Philadelphia, 1901.

Henoch, Klinik der Unterleibskrankheiten, Berlin, 1863, third ed.

Leube, "Krankheiten des Darms," v. Ziemssen's Handb. d. spec. Pathol. Therap., Leipsic, 1878, second ed.

Liebermeister, Vorlesungen über die Krankheiten der Unterleibsorgane, Leipsic, 1894.

Mathieu, Traité des maladies de l'estomac et de l'intestin, Paris, 1901.

Nothnagel, Beiträge zur Physiologie und Pathologie des Darms, Berlin, 1884.

Pick, A., Vorlesungen über Magen- und Darmkrankheiten, Vienna, 1895.

Pribram, "Krankheiten des Darms," Ebstein and Schwalbe, Handb. der prak. Med., vol. ii.

Rosenheim, Pathologie und Therapie der Krankheiten des Darms, Leipsic, 1893.

Widerhofer, "Darmkrankheiten," Gerhardt's Handb. d. Kinderkrankh., Tübingen, 1880, vol. iv.

(See, also, the numerous handbooks on Practice of Medicine, etc.)

A

Abraham, "Ueber die Rosenbach'sche Urinfärbung," Berlin. klin. Wochenschr., 1890.

Achard and Broca, "Bactériologie de vingt cas d'appendicite suppurée," Bull. et mém. de la soc. méd. des hôp. de Paris, 1897.

Acland, Pathological Transactions, 1885.

Adenot, Revue de med., 1890.

Adrian, "Die Appendicitis als Folge einer Allgemeinerkrankung," Grenzgeb. d. Med. u. Chir., vol. vii.

Ahlfeld, "Beiträge zur Lehre vom Resorptionsfieber in der Geburt und im Wochenbett und von der Selbstinfection," Zeitschr. f. Geburtsh. u. Gynäk., 1893, vol. xxvii.

Akerlund, "Studien über Enteritis membranacea," Arch. f. Verdauungskr., vol. i.

Albers, Beobachtungen auf dem Gebiete der Pathologie und pathol. Anatomie, Bonn, 1838.

Albrecht, "Ueber arterio-mesenterialen Darmverschluss an der Duodeno-Jejunalgrenze," Virchow's Arch., vol. clvi.

Albrecht and Ghon, Ueber die Beulenpest im Bombay im Jahre 1897, Vienna, 1898, Part II. B.

Albu, Ueber die Autointoxicationen des Intestinaltractus, Berlin, 1895.

Alexander, "Thrombose der Pfortader und ihre Aeste," Berlin. klin. Wochenschr., 1866, No. 4.

Allingham, Diseases of the Rectum, New York, 1883.

Angerer, Verhandl. des XIII. Cong. f. innere Med. (Discussion über die Typhlitiden), Wiesbaden, 1895.

Angerer, "Ueber subcutane Darmrupturen und ihre operative Behandlung," Arch. f. klin. Chir., vol. lxi.

Anghel, "Etude sur la pathogénie de l'appendice," Thèse de Paris, 1897.

Apolant, "Ueber das gleichzeitige Vorkommen von Angina und Perityphlitis," Therap. Monatsh., 1897, No. 2.

D'Arcy-Power, "On the Pathology and Surgery of Intussusception," Lancet, 1897.

D'Arcy-Power, after Virchow-Hirsch's Jahresbericht, 1886.

Arnd, "Ueber die Durchlässigkeit der Darmwand eingeklemmter Brüche für Mikroorganismen," Mittheil. aus Klin., etc., der Schweiz, 1893, Series 1, No. 4.

Arnold and Steele, "Masses of Fibrin from the Intestine," Proc. Path. Soc. of Philadelphia, November 1, 1898.

Askanazy, "Ueber acute Leukämie und ihre Beziehungen zu geschwürigen Processen im Verdauungscanal," Virchow's Arch., vol. cxxxvii.

Askanazy, M., "Ueber das Verhalten der Darmganglien bei Peritonitis," Verhandl. der deutsch. path. Gesellschaft, 1900, No. 3.

Aufrecht, "Amyloidgeschwüre des Darmcanals," Berlin. klin. Wochenschr., 1869, No. 30.

Aufrecht, "Zur Pathologie und Therapie der Paratyphlitis," Therap. Monatsh., 1895, No. 5.

Augagneur, "Tumeurs du mesentère," Thèse de Paris, 1884.

Austerlitz and Landsteiner, "Bakteriendichtigkeit der Darmwand," Sitzungsb. der kais. Akad. der Wissenschaft, Vienna, January, 1898.

B

Bäumler, "Syphilis," v. Ziemssen's Handb. d. spec. Pathol. u. Therap., vol. iii.

Bäumler, "Klinische Erfahrungen über Behandlung der Perityphlitis," Deutsch. Arch. f. klin. Med., vol. lxxiii.

Baginsky, A., "Ueber Gährungsvorgänge im kindlichen Darmcanal," Deutsch. med. Wochenschr., 1888, Nos. 20, 21.

Baginsky, A., Lehrbuch der Kinderkrankh., Berlin, 1889, third ed.

Baldy, "Salines in Peritonitis Following Abdominal Section," New York Med. Rec., November 15, 1887.

Baltzer, Arch. f. klin. Chir., vol. xliv.

Bamberger, "Krankheiten des chylopoetischen Systems," Virchow's spec. Pathol. u. Therap., Würzburg, 1864.

Bamberger, Oesterreichische Zeitschr. f. prak. Heilk., 1857.

Bamberger, "Die Entzündungen der rechten Fossa iliaca," Wien. med. Wochenschr., 1853.

Barbacci, "Sull' eziologia e patogenesi della peritonite de perforazione," Lo sperimentale, 1893, No. 4.

Bardeleben, "Ueber die Lage des Blinddarms beim Menschen," Virchow's Arch., vol. ii.

Bardeleben, Lehrbuch der Chirurgie, Berlin, 1875, vol. iii., seventh ed.

Bardenheuer, Arch. f. klin. Chir., vol. xli.

Bargebuhr, "Ueber Ascites chylosus und chyliformis," Deutsch. Arch. f. klin. Med., 1893, vol. li.

Basly, "The Effusion of Chyle, etc., into the Serous Cavities," Amer. Jour. Med. Sci., 1889, vol. xcvi.

Bauer, "Die Krankheiten des Peritoneum," v. Ziemssen's Handb. d. spec. Pathol. u. Therap., 1875, vol. viii., first ed.; 1878, second ed., Leipsic.

Baumgarten, "Ueber latente Tuberculose," Volkmann's Samml. klin. Vorträge, No. 218.

Baumgarten, Die Histogenese des tuberculösen Processes, Berlin, 1885.

Baumgarten, Centralbl. f. klin. Med., 1884, No. 2.

Baumgarten, Zeitschr. f. klin. Med., 1886, vol. x.

Bayer, Arch. f. Heilk., 1870, p. 399.

Bayer, "Charakteristischer Meteorismus bei Volvulus des S romanum," Arch. f. klin. Chir., vol. lvii.

Bayer, "Zur Pathologie und Therapie der Darmstenose," Würzburger Abhandlungen, Würzburg, 1902, vol. ii., No. 6.

Bayliss and Starling, "The Movements and Innervation of the Small Intestine," Jour. Physiol., vol. xxiv.

Beard, Die Nervenschwäche, u. s. w., trans. by Neisser, Leipsic, 1883.

Beaussenat, "Appendicites expérimentales," Bull. de la soc. anat. de Paris, 1897, vol. xi.

Beck, "Ueber die Aufsaugung feinvertheilter Körper aus den serösen Höhlen," Wien. klin. Wochenschr., 1893, No. 46.

Beck, C., "Appendicitis," Samml. klin. Vorträge, 1898, new series, No. 221.

Beck v., Deutsch. Zeitschr. f. Chir., vols. xi. and xv.

Bellfrage and Hedenius, cited from Virchow-Hirsch's Jahresbericht für 1876.

Bellon, "Des symptomes de l'étranglement interne," Thèse de Paris, 1878.

Berggrün and Katz, "Beitrag zur Kenntniss der chronisch-tuberculösen Peritonitis des Kindesalters," Wien. klin. Wochenschr., 1891, No. 4.

Beschorner, "Ueber chronisch-essentielle fibrinöse Bronchitis," Samml. klin. Vorträge, new series, No. 73.

Besnier, "Des étranglements internes de l'intestin," Paris, 1860.

Bessel-Hagen, "Ein ulceröses Sarkom des Jejunum bei einem Kinde," Virchow's Arch., vol. xcix., p. 99.

Biedert, Jahrbuch f. Kinderheilk., 1878, 1879, and 1881.

Bieganski, ref. in Virchow-Hirsch's Jahresbericht, 1892.

Bieganski, "Beitrag zur Differentialdiagnose der subphrenischen Abscesse," Virchow's Jahrb., 1893, vol. vi., p. 174.

Biermann, "Die diagnostische Bedeutung des Nachweises der Tuberkelbacillen in den Stuhlentleerungen," Inaug. Diss., Berlin, 1896–97.

Biesalski, Die Entstehungsweise der verschiedenen Formen von Peritonitis, Berlin, 1895.

Billroth, "Ueber Duodenalgeschwüre bei Septikämie," Wien. med. Wochenschr., 1867, No. 45.

Billroth, "Fall von Cöcumtuberculose," Wien. klin. Wochenschr., 1891, No. 10.

Birch-Hirschfeld, Lehrbuch der pathol. Anat., Leipsic, 1887.

Blaschko, "Erkrankung der sympathischen Geflechte der Darmwand," Virchow's Arch., vol. xciv.

Blavel, "Ueber Sarkome der Ileocöcalgegend," ibid., vol. clxii.

Boas, Deutsch. med. Wochenschr., 1893, No. 41.

Boas, "Ueber Duodenalstenosen," Berlin. klin. Wochenschr., 1891, p. 949.

Boas, "Ueber die Stenose des Duodenum," Deutsch. med. Wochenschr., 1891, No. 28.

Boas, ibid., 1900, No. 36.

Boas, "Ernährungstherapie bei functionellen Erkrankungen der Speiseröhre, des Magens und Darms," Leyden's Handb. d. Ernährungstherap., Leipsic, 1898.

Boas, "Symptomatologie und Diagnose der Colitis membranacea," XIII. Cong. internat. de méd., Paris, 1900.

Boettcher, "Einige Bemerkungen über Darmmyome," Virchow's Arch., 1886, vol. civ.

Bönnecken, "Ueber Bakterien des Bruchwassers eingeklemmter Hernien und deren Beziehungen zur peritonealen Sepsis," ibid., vol. cxx.

Böhm, "Die Indicationen zur chir. Behandlung der Perityphlitis," Fortsch. d. Med., 1902, No. 20.

Boilo, Baumgarten's Jahresbericht f. 1891. Gazetta med. di Torino, 1891.

Bokai, "Experimentelle Beiträge zur Kenntniss der Darmbewegungen," Arch. f. exper. Path. u. Pharmakol., vol. xxiii.

Bollinger, "Ueber Tuberkelbacillen im Euter einer tuberculösen Kuh," Münch. ärztlich. Intelligenzbl., 1883, No. 16.

Bolt, "Ueber Darmeinklemmungen durch das Diverticulum Meckelii," Dissertation, Marburg, 1891.

Bonfigli, "Un caso di cisti dermoide nella cavita degli epiplon," Rivista clin. di Bologna, 1875.

Borchardt, "Die Behandlung der Appendicitis," Mittheil. aus den Grenzgeb. der Med. u. Chir., vol. ii.

Borchgrevink, "Zur Kritik der Laparotomie bei der serösen Bauchfelltuberculose," Grenzgeb. d. Med. u. Chir., vol. vi.

Borgen, "Ein Fall von habituellem Volvulus," ref. in Arch. f. Verdauungskr., vol. v.

Borrmann, "Ueber Netz- und Pseudonetztumoren, etc.," Grenzgeb. d. Med. u. Chir., vol. vi.

Borsack, "Ein Fall von zweifacher Verletzung des Mastdarms und des S romanum," Virchow's Jahresbericht für 1893, vol. ii., p. 475.

Borschke, "Pathogenese der Peritonitis tuberculosa," Virchow's Arch., vol. cxxvii.

Borst, "Fibrinöse Exsudation und fibrinoide Degeneration," Berlin. klin. Wochenschr., 1897, No. 10.

Bossard, '' Ueber die Verschwärung und Durchbohrung des Wurmfortsatzes,'' Inaug. Diss., Zurich, 1896.

Bouchanan, Glasgow Journal, March–April, 1892.

Bouchard, Leçons sur les auto-intoxications dans les maladies, Paris, 1887.

Bouveret, Die Neurasthenie, von Dornblüth, Leipsic and Vienna, 1893.

Braam-Houkgeest, '' Untersuchungen über die Peristaltik des Magens und Darmcanals,'' Pflüger's Arch., 1872, vol. vii.

Braun, '' Ueber entzündliche Geschwülste des Netzes,'' Arch. f. klin. Chir., vol. lxiii.

Brentano, '' Ueber Mesenterialcysten,'' Berlin. klin. Wochenschr., 1895, No. 18.

Brieger, '' Beiträge zur Lehre der fibrösen Hepatitis,'' Virchow's Arch., vol. lxxv.

Brinton, On Intestinal Obstruction, London, 1867.

Bristowe, Reynold's System of Medicine, London, 1871.

Broesicke, '' Ueber Intraabdominale Hernien und Bauchfelltaschen,'' Berlin, 1891.

Brosche, '' Zur Aetiologie der Carcinome des Verdauungstractus,'' Wien. med. Wochenschr., 1895, No. 40.

Brunner, W., '' Ueber die häutige Gedärmentzündung,'' Virchow-Hirsch's Jahresbericht, 1892.

Brunton and Cash, St. Barthol. Hosp. Reports, 1887, vol. xxii.

Bryant, Annals of Surgery, February, 1893.

Bumm, '' Die puerperale Wundinfection,'' Centralbl. f. Bakteriol., 1887, vol. ii., p. 343.

Bumm, '' Zur Aetiologie der septischen Peritonitis,'' Münch. med. Wochenschr., 1889.

Bumm, '' Ueber die Heilungsvorgänge nach dem Bauchschnitt bei bacillärer Bauchfelltuberculose,'' Sitzungsbericht der Würzburger Gesellschaft, 1893, No. 1.

Bunge, Lehrbuch der physiol. und pathol. Chemie, Leipsic, 1887.

Burckhardt, '' Ueber acute fortschreitende Peritonitis bei Epityphlitis und ihre chirurgische Behandlung,'' Deutsch. Zeitschr. f. Chir., 1899, 1901, and Württemburgisches med. Correspondenzbl., 1901.

Burginsky, '' Ueber die pathogene Wirkung des Staphylococcus aureus auf einige Thiere,'' Baumgarten's Jahresbericht für 1891.

C

Cahn, '' Ueber die Diagnose der Verengerung des unteren Theiles des Duodenum, etc.,'' Berlin. klin. Wochenschr., 1886, No. 22.

Cannon, '' The Movements of the Intestines Studied by Means of the Roentgen Rays,'' Amer. Jour. Physiol., 1901, vol. vi.

Carter, Edinburgh Med. Jour., 1858.

Castel, du, '' Cancer de l'iléon,'' Arch. gén. de méd., 1882, vol. ii.

Cathelin, Presse médicale, June 21, 1899.

Charcot, Leçons sur les maladies du foie et des reins, Paris, 1877.

Charrin and Veillon, Comp. rend. de la Soc. de Biol., 1893, p. 1057.

Chelichowski, '' Zur Diagnose des subdiaphragmatischen Abscesses,'' Virchow's Jahrb. für 1893, vol. ii., p. 173.

Cherchevsky, '' Contribution à la pathologie des névroses intestinales,'' Rév. de méd., 1883.

Chevalier, Contribution à l'étude de la lithiase intestinale, Paris, 1898.

Chvostek, '' Das einfache Duodenalgeschwür,'' Wien. med. Jahrb., 1883.

Citron, '' Zur klin. Würdigung des Eiweissgehaltes und des specifischen Gewichtes pathol. Flüssigkeiten,'' Deutsch. Arch. f. klin. Med., vol. xlvi.

Clairmont and Haberer, '' Ueber das Verhalten des gesunden und veränderten thierischen Peritoneums,'' Wien. klin. Wochenschr., 1902, No. 45.

Cohnheim, Vorlesungen über allgemeine Pathologie, Berlin, 1880.

Colberg, '' Beiträge zur normalen und patholog. Anatomie der Lungen,'' Deutsch. Arch. f. klin. Med., 1867, vol. ii.

Collin, '' Étude sur l'ulcère simple du duodenum,'' Thèse de Paris, 1894.

Condio, '' Nervöse Diarrhöe in der Gravidität,'' Il Morgagni, 1896.

Conrath, '' Ueber die locale chronische Cöcumtuberculose und ihre chir. Behandlung,'' Beitr. zur klin. Chir., 1898, vol. xxi.

Cornil, Bull. de l'acad. de méd., 1888, No. 32.

Cossy, Arch. gén. de méd., 1879, vol. ii.

Da Costa, Amer. Jour. Med. Sci., 1871.

Da Costa, '' The Clinical Value of Blood-examination in Appendicitis,'' ibid., November, 1901.

Courtois-Suffit, '' Maladies du péritoine,'' Traité de médecine, publié par Charcot, Bouchard, Brissaud, Paris, 1892, vol. iii.

Criegern, v., "Die Feststellung kleiner Mengen freier Flüssigkeit in der Bauchhöhle von den Leistenringen aus," Berlin. klin. Wochenschr., 1901, No. 19.

Cruveilhier, Traité d'anatomie pathologique générale, 1849.

Csokor, "Ueber das Wurmaneurysma des Pferdes," Wien. klin. Wochenschr., 1893, No. 50.

Curling, Med.-Chir. Trans., 1842, vol. xxv.

Curschmann, "Die Pocken," v. Ziemssen's Handb. d. spec. Pathol. u. Therap., Leipsic, 1877, vol. ii., second ed.

Curschmann, "Die Behandlung des Ileus," Cong. f. innere Med., Wiesbaden, 1889.

Curschmann, "Topographisch-klinische Studien," Deutsch. Arch. f. klin. Med., vol. liii.

Curschmann, "Zur Differentialdiagnostik der mit Ascites verbundenen Erkrankungen der Leber und des Pfortadersystems," Deutsch. med. Wochenschr., 1884, No. 35.

Curschmann, "Zur diagn. Beurtheilung der vom Blinddarm und Wurmfortsatz ausgehenden entzündl. Processe," Münch. med. Wochenschr., 1901, Nos. 48, 49.

Czerny, "Ueber die chirurgischen Erfolge bei intraperitonealer Tuberculose," Beitr. zur klin. Chir., von Bruns, 1890, vol. vi.

Czerny, "Perforationsperitonitis nach Magengeschwür," Centralbl. f. Chir., 1887.

Czerny and Heddaeus, "Beitrag zur Pathol. u. Ther. der Wurmfortsatzentzündung," Beitr. zur klin. Chir., vol. xxi.

D

Dauber, "Ueber primäre Typhlitis als Ursache recidivirender Appendicitisattaquen," Grenzgeb. d. Med. u. Chir., vol. iv.

Daver, "Ueber einige seltenere Varietäten der Appendicitis," Wien. med. Blätter.

Deaver, "Remarks upon the Differential Diagnosis, etc., of Appendicitis," Annals of Surgery, 1898, vol. xxvii.

Deckart, "Ueber Thrombose und Embolie der Mesenterialgefässe," Grenzgeb. d. Med. u. Chir., 1900, vol. v.

Demme, Jahresbericht des Jenner'schen Kinderspitals in Bern, 12, 15–18, and 21 Bericht.

Desnos, "Société méd. des hôp. de Paris," Wien. med. Presse, 1891, No. 51.

Devic and Rocca, "Ulcère chronique du duodénum," Province méd., 1896, Nos. 44–47.

Dickinson, Trans. Path. Soc., London, 1866–67, vol. xviii., 1879, vol. xxix.

Dieulafoy, "La lithiase intestinale et la gravelle de l'intestin," Bull. de l'acad. de méd., March 9, 1897, and Presse méd., March 10.

Dobroklonski, "De la pénétration des bacilles tuberculeux dans l'organisme à travers la muqueuse intestinale," Arch. de méd. expér. et d'anat. path., 1890, vol. ii.

Dohrn, Deutsch. med. Wochenschr., 1893.

Dunin, "Ueber habituelle Stuhlverstopfung, deren Ursachen und Behandlung," Berlin. Klinik, Berlin, 1894, No. 34.

E

Ebstein, "Ueber die acute Leukämie und Pseudoleukämie," Deutsch. Arch. f. klin. Med., vol. xliv.

Ebstein, "Krankheiten des Harnapparates," v. Ziemssen's Handb. d. spec. Path. u. Therap., Leipsic, 1878, vol. ix.

Ebstein, "Klinisches und Kritisches zur Lehre von der Perforationsperitonitis," Zeitschr. f. klin. Med., vol. ix.

Ebstein, "Zur Aetiologie der acut sich entwickelnden Bauchtympanie Hysterischer," Neurolog. Centralbl., 1883, No. 2.

Ebstein, Die chronische Stuhlverstopfung, Stuttgart, 1901.

Edel, "Ueber erworbene Darmdivertikel," Virchow's Arch., vol. cxxxviii.

Edlefsen, "Ueber febris ex obstipatione," Klin.-therap. Wochenschr., 1900, No. 46.

Edwards, "Membranous Enteritis," Amer. Jour. Med. Sci., April, 1888.

Ehrmann, Wien. med. Jahrb., 1885.

Eichhorst, "Ueber Darmgries," Deutsch. Arch. f. klin. Med., vol. lxviii.

Einhorn, "Ueber Perforationen des Processus vermiformis und des Cöcum," Inaug. Diss., Munich, 1891

Einhorn, "Verstopfung und Diarrhöe als Folgezustände mancher Magenkrankheiten," Arch. f. Verdauungskr., vol. iii.

Einhorn, "Die membranöse Enteritis und ihre Behandlung," ibid., vol. iv.

Einhorn, "Scheinbare Tumoren des Abdomens," Berlin. klin. Wochenschr., 1901, No. 43.

Einhorn, "Bemerkung über Enteroptose," Deutsche Praxis, Zeitschr. f. prakt. Aerzte, 1901, Nos. 7, 8.

Eisenlohr, "Ein Fall von hinter dem Colon gelegenen Abscess," Deutsch. med. Wochenschr., 1890.

Eisenlohr, "Zur Thrombose der Mesenterialvenen," Jahrb. d. Hamburger Staatskrankenanstalten, 1890, No. 2.

Ekehorn, Virchow-Gurlt Jahresbericht für 1893.

Elliot, Annals of Surgery, 1895.

Emminghaus, "Einiges über pathologisch-anatomische Befunde bei Innervationsstörungen des Darms," Münch. med. Wochenschr., 1894, Nos. 5, 6.

Enderlen and Hess, "Ueber Antiperistaltik," Deutsch. Zeitschr. f. Chir., vol. lix.

Engelmann, Pflüger's Arch., 1869, vol. ii., and 1871, vol. iv.

Englisch, "Ueber Albuminurie bei eingeklemmten Eingeweidebrüchen," Oesterreichische med. Jahrb., 1884, Nos. 2, 3.

Engstroem, "Zur Kenntniss des Dünndarmsarkoms," ref. in Arch. f. Verdauungskr., vol. iv.

Erskine, Maron, Amer. Jour. Med. Sci., January, 1873.

Esau, "Ueber Achsendrehung des Darms," Deutsch. Arch. f. klin. Med., vol. xvi.

Esmarch, "Die Krankheiten des Mastdarms und des Afters," Pitha-Billroth's Handbuch, vol. iii.

Eulenburg and Guttmann, Die Pathologie des Sympathicus auf physiologischer Grundlage, Berlin, 1873.

Ewald, Klinik der Verdauungskrankheiten, Berlin.

Ewald, "Ueber Enteroptose und Wanderniere," Berlin. klin. Wochenschr., 1890, Nos. 12, 13.

Ewald, Ibid., 1889.

Ewald, Deutsch. med. Wochenschr., 1893, No. 41.

Ewald, "Discussion über die Typhlitiden," Verhandl. d. XIII. Cong. f. innere Med., Wiesbaden, 1895.

Ewald, "Darmstenose," Encyklopädische Jahrb., vol. i.

Ewald, "Diseases of the Intestines," Twentieth Century Practice of Med., New York, 1897, vol. ix.

Ewald, "Ueber Appendicitis larvata," Arch. f. klin. Chir., vol. lx.

Ewald, "Anæmia gravis als Folge versteckter Hämorrhoidalblutungen," Therap. d. Gegenwart, November, 1899.

Ewald, "Ernährungstherapie bei Darmkrankheiten," Leyden's Handb. der Ernährungstherap., Leipsic, 1898, vol. ii.

Exner, S., "Zur Mechanik der peristaltischen Bewegung," Pflüger's Arch., 1884, vol. xxxiv.

F

Faber, "Die Embolie der Arteria mesenterica superior," Deutsch. Arch. f. klin. Med., vol. xvi.

Faber, "Ueber Darmdyspepsie," Arch. f. Verdauungskr., vol. viii.

Faber and Bloch, "Ueber die patholog. Veränderungen am Digestionstractus bei der perniciösen Anämie und über die sogenannte Darmatrophie," Zeitschr. f. klin. Med., vol. xl.

Faisans, "Sur la nature et le traitement de l'appendicite," Semaine méd., 1899, No. 14.

Federn, "Ueber partielle Darmatonie," etc., Wien. Klinik, Vienna, 1891.

Federn, Blutdruck und Darmatonie, Vienna, 1894.

Fehling, Die Physiologie und Pathologie des Wochenbettes, second ed., Stuttgart, 1897.

Feigel, "Przeglad lekarski," ref. in Virchow-Hirsch's Jahresbericht, 1877.

Fellner, Pflüger's Arch., vol. lvi.

Fenwick, On the Atrophy of the Stomach, etc., London, 1880.

Fenwick and Dodwell, "Perforation of the Intestines in Phthisis," Lancet, July, 1892.

Ferguson, Amer. Jour. Med. Sci., January, 1891.

Feyat, De la constipation et des phénomènes toxiques qu'elle provoque, 1890.

Ficket and Malvon, Arch. de méd. exper., 1891, vol. iii.

Fiedler, "Gibt es eine Peritonitis chronica exsudativa idiopathica?" Jahresber. der Gesellschaft für Natur- und Heilkunde zu Dresden, 1885–86.

Finney, "Surgical Treatment of Perforating Typhoid Ulcer," Annals of Surgery, 1897.

Finney and Hamburger, "The Relation of Appendicitis to Infectious Diseases," Amer. Medicine, December 14, 1901.

Fischer, H., "Bericht über die 1862–64 in der Klinik Traube's vorgekommenen Puerperalerkrankungen," Charité-Annalen, Berlin, 1865.

Fischer, J., "Zur Kenntniss der Darmaffectionen bei Nephritis und Urämie," Virchow's Arch., vol. cxxxiv.

Fischl, Prager Vierteljahrsschr. f. Heilk., 1878.

Fischl, "Ein Beitrag zur Casuistik der nervösen Diarrhöen," Prager Wochenschr., 1891, No. 47.

Fitz, Boston Med. and Surg. Jour., 1890, vol. cxxii. ; New York Med. Jour., 1888, vol. xlvii. ; Amer. Jour. Med. Sci., 1886.

Fleiner, "Ueber Behandlung d. Constipation, u. s. w.," Berlin. klin. Wochenschr., 1893, Nos. 3, 4.

Fleiner, "Zwei Fälle von Darmgeschwülsten mit Invagination," Virchow's Arch., 1885, vol. ci.

Fleischer, Lehrbuch der inneren Med., Wiesbaden, 1896.

Florand, "Sur la nature et le traitement de l'appendicite," Semaine méd., 1899, No. 14.

Förster, Handbuch der pathol. Anat., Leipsic, 1854.

Foges, Casuistische Beiträge zur Klinik der Appendicitis simplex, Vienna, 1896.

le Fort, Gaz. hebdom., Paris, February, 1865.

Fowler, George R., Ueber Appendicitis, trans. by Steinthal, Berlin, 1896.

Fraenkel, Alb., Zeitschr. f. klin. Med., vol. iii.

Fraenkel, Albert, "Discussion über den subphrenischen Abscess," Berlin. klin. Wochenschr., 1892, No. 46.

Fraenkel, "Ueber idiopathische acut und chronisch verlaufende Peritonitis," Charité-Annalen, Berlin, vol. xii.

Fraenkel, "Ueber puerperale Peritonitis," Verhandl. d. Vereines f. innere Med. zu Berlin, Berlin, 1883–84, vol. iii.

Fraenkel, Alexander, "Ueber peritoneale Infection," Wien. klin. Wochenschr., 1891, Nos. 13, 14, and 15.

Fraenkel, "Zur Chirurgie des Gallensystems," Centralbl. f. Chir., 1892, No. 35.

Fraenkel, Eugen, "Zur Aetiologie der Peritonitis," Münch. med. Wochenschr., 1890, No. 2.

Frank, "Ueber Albuminurie bei Darmeinklemmung in Brüchen," Berlin. klin. Wochenschr., 1887, No. 38.

Frank, Adolph, "Die Erfolge der operativen Behandlung der chronischen Bauchfell-tuberculose," Grenzgeb. d. Med. u. Chir., vol. vi.

Franke, Felix, "Ueber einige chir. wichtige Complicationen der Influenza," ibid., vol. v.

Franke, F., "Heilung eines Falles von Colica mucosa durch Anlegung eines künstlichen Afters," ibid., vol. i.

Frankenhaeuser, Petersburger med. Wochenschr., 1894.

Frankfurter, "Zur Pathologie und Therapie der Perityphlitis und Appendicitis," Inaug. Diss., Strassburg O. E., 1893.

Frankl-Hochwart, v. and Fröhlich, "Ueber Tonus und Innervation der Sphinkteren des Anus," Arch. f. ges. Physiol., vol. lxxxi.

Frankl-Hochwart, v. and Fröhlich, Wien. klin. Rundschau, 1901, No. 41.

Frentzel, "Zur Semiotik und Therapie mesenterialer Cysten," Deutsch. Zeitschr. f. Chir., vol. xxxiii.

Frerichs, Die Bright'sche Nierenkrankheit, Braunschweig, 1851.

Frerichs, "Verdauung," Wagner's Handwörterb. d. Physiol.

Frerichs, "Klinik der Leberkrankheiten, Braunschweig, 1861.

Friedreich, Deutsch. Arch. f. klin. Med., vol. ix.

Friedreich, "Krankheiten des Pankreas," v. Ziemssen's Handb. d. spec. Pathol. u. Therap., Leipsic, 1878, vol. viii.

Friedreich, "Ueber eine besondere Form chronischer hämorrhagischer Peritonitis," Virchow's Arch., vol. lviii.

Friedreich, "Einige Fälle von ausgedehnter amyloider Entartung," ibid., vol. xi.

Friedrich, "Zur bakteriellen Aetiologie und zur Behandlung der diffusen Peritonitis," Arch. f. klin. Chir., vol. lxviii.

Frohmann, "Zur Kenntniss der primären Sarkome des Darms," Festschr. f. M. Jaffe, Braunschweig, 1901.

Fürbringer, "Discussion über den subphrenischen Abscess," Verhandl. d. Vereines f. innere Med., Berlin, 1889, No. 8, p. 278.

Fürbringer, Deutsch. med. Wochenschr., 1882, No. 10.

Fürbringer, "Die Gallensteinkrankheiten," Verhandl. d. Cong. f. innere Med., Wiesbaden, 1891.

Fürbringer, "Deutsch. med. Wochenschr., 1891, p. 299.

Fürbringer, "Zur Kenntniss der traumatischen Perityphlitis," Aerztl. Sachverständigen-Zeitung, 1900, No. 9.

G

Gad, "Darmperistaltik," Eulenburg's Realencyklopädie d. ges. Heilk.

Gärtner, Correspondenzbl. d. allgem. Thüringer Aerztevereines, 1888.

Galvagni, "Sulla peritonite ad essudato sieroso e siero fibrinoso," Rivista clinica di Bologna, 1869, Nos. 6, 7, and 12.

Garré, "Bakteriologische Untersuchungen des Bruchwassers eingeklemmter Hernien," Fortschr. d. Med., 1886, No. 8.

Gatti, "Ueber die feineren histologischen Vorgänge bei der Rückbildung der Bauchfell-tuberculose nach einfachem Bauchschnitt," Arch. f. klin. Chir., vol. liii.

Genser, v. "Eingeweidewürmer bei Appendicitis," Wien. med. Wochenschr., 1901, No. 19.

Geoffroy, "La contracture du gros intestin, son rôle dans la pathogénie de l'entérite glaireuse," XIII. Cong. Internat. de méd., Paris, 1900.

Georgiewski, ref. in Arch. f. Verdauungskrankh., vol. ii.

Gerhardi, "Pankreaskrankheiten und Ileus," Virchow's Arch., vol. vi.

Gerhardt, "Zur Lehre von der Gelbsucht," Zeitschr. f. klin. Med., vol. vi.

Gerhardt, "Discussion in der Gesellschaft der Charité-Aerzte zu Berlin," Berlin. klin. Wochenschr., 1891, No. 4.

Gerhardt, "Embolie der Arteriæ mesentericæ," Würzburger med. Zeitschr., 1863, vol. iv.

Gerlach, Wold., "Kritische Bemerkungen zur gegenwärtigen Lehre von der Darmatrophie," Deutsch. Arch. f. klin. Med., vol. lvii.

Gersuny, "Wann operiren wir bei Appendicitis?" Wien. med. Presse, 1896, No. 46.

Gersuny, "Ueber eine typische peritoneale Adhäsion," Arch. f. klin. Chir., vol. lix.

Gersuny, "Ueber ein Symptom bei Kothtumoren," Wien. klin. Wochenschr., 1896, No. 40.

Gesselewitsch and Wanach, "Die Perforationsperitonitis beim Abdominaltyphus und ihr operativer Erfolg," Grenzgeb. d. Med. u. Chir., 1897, vol. ii.

de Giacomi, Fortschr. d. Med., vol. i., No. 5.

Glaeser, Czerny, Heddaeus and die Behandlung der Appendicitis, Hamburg, 1899.

Glénard, De l'enteroptose, 1889.

Glinski, "Zur Kenntniss der Dickdarm-Lymphosarkome," Virchow's Arch., vol. clxvii.

Glockner, "Ueber den sogenannten Endothelkrebs der serösen Häute," Zeitschr. f. Heilk., 1897, vol. xviii.

Gluzinski, "Ein Beitrag zur Symptomatologie der Darmperforation," Virchow's Jahresbericht, 1895, vol. ii., p. 198.

Gockel, "Ueber die traumatische Entstehung des Carcinoms mit besonderer Berücksichtigung des Intestinaltractus," Arch. f. Verdauungskr., vol. ii.

Goebell, "Zur Kenntniss der lateral-retroperitonealen Tumoren," Deutsch. Zeitschr. f. Chir., vol. lxi.

Gölder, v., "Ueber acute Psychosen bei Koprostase," Klin. Jahrb. f. Psychiatrie u. Neurol., 1898.

Goeppert, "Fall von angeborener Abknickung des Dickdarms, etc.," Arch. f. Verdauungskrankh., vol. v.

Goldberger and Weiss, "Die Jodreaction im Blute und ihre diagnostische Verwerthung in der Chirurgie," Wien. klin. Wochenschr., 1897, No. 20.

Goluboff, "Die Appendicitis als eine epidemische infectiöse Erkrankung," Berlin. klin. Wochenschr., 1897, No. 1.

Good, "Beiträge zur Kenntniss der Divertikelbildungen und inneren Incarcerationen des Darmtractes," Mittheil. aus Klin. und med. Instituten der Schweiz, Basle, 1895.

Gottsacker, "Zur Histogenese der tuberculösen Darmgeschwüre," Inaug. Diss., Bonn, 1880.

Graser, 'Untersuchungen über die feineren Vorgänge bei der Verwachsung peritonealer Blätter," Deutsch. Zeitschr. f. Chir., vol. xxvii.

Graser, "Behandlung der Darmverengerung und des Darmverschlusses," Penzoldt's und Stintzing's Handbuch der speciellen Therapie innerer Krankheiten, Jena, 1896.

Graser, "Das falsche Darmdivertikel," Arch. f. klin. Chir., vol. lix.
Grassberger, "Ein Fall von multipler Divertikelbildung des Darmtractes, etc.," Wien. klin. Wochenschr., 1897, No. 6.
Grawitz, "Statistischer und experimentell pathologischer Beitrag zur Kenntniss der Peritonitis," Charité-Annalen, Berlin, 1886, No. 11.
Grawitz, "Beitrag zur Theorie der Eiterungen," Virchow's Arch., vol. cxvi.
Grawitz, "Ein Fall von Perforation eines perityphlitischen Abscesses, u. s. w.," Berlin. klin. Wochenschr., 1889, No. 32.
Grawitz, "Ein Fall von Embolie der Arteria meseraica superior," Virchow's Arch., vol. cx.
Greenwood, Lancet, August 21, 1880.
Grisolle, Arch. gén. de méd., 1839, new series, vols. iii., iv.
Grohé, B., Pathologie und Therapie der Typhlitiden. Eine historische Studie, Greifswald, 1896. (Complete literature to 1896.)
Gross, Alf., "Ein Beitrag zur Kenntniss der pseudo-chylösen Ergüsse," Arch. f. exper. Path. u. Pharmak., vol. xliv.
Grosser, "Ueber Zwerchfellhernien," Wien. klin. Wochenschr., 1899, No. 24.
Grützner, "Zur Physiologie der Darmbewegung," Deutsch. med. Wochenschr., 1894, No. 48.
Grützner, "Ueber die Bewegungen des Darminhaltes," Arch. f. d. ges. Physiol., 1898, vol. lxxi.
Guttmann, P., "Discussion über den subphrenischen Abscess," Verhandl. des Vereines f. innere Med., Berlin, 1889, No. 8, p. 278.
Guttmann, P., "Discussion über Peritonitis," ibid., Berlin, 1883–84, No. 3, p. 301.

H

Habel, "Ueber Darmatrophie," Virchow's Arch., vol. cliii.
Hagenbach, "Ueber complicirte Pankreaskrankheiten und deren chirurgische Behandlung," Deutsch. Zeitschr. f. Chir., vol. xxvii.
Hahn, "Ueber Mesenterialcysten," Berlin. klin. Wochenschr., 1887, No. 23.
Hamburger, "Ueber die Regelung der osmotischen Spannkraft von Bauch- und von Pericardialhöhle," Du Bois-Reymond's Arch. f. Physiol., 1895.
Hampeln, Zeitschr. f. klin. Med., 1884, vol. viii.
Hanan, "Bemerkungen zu der Mittheilung von Hansemann: Ueber die Entstehung falscher Darmdivertikel," Virchow's Arch., vol. cxlv.
Hansemann, "Ueber die Enstehung falscher Darmdivertikel," ibid., vol. cxliv.
Harbitz, cited after Krogius.
Hartley, "Appendicitis," New York Med. Rec., 1890.
Hauser, "Das Cylinderepithel-Carcinom des Magens und des Dickdarms," Jena, 1890.
Hauser, "Ueber Polyposis intestinalis adenomatosa," Deutsch. Arch. f. klin. Med., 1895, vol. lv.
Hayem, Gazette méd. de Paris, 1866, vol. xxi.
Hedenius, Virchow-Hirsch's Jahresbericht für 1875, vol. i., p. 358.
Hegar, "Embolie der Lungenarterie und der Arteria meseraica inferior," Virchow's Arch., vol. xlviii.
Heidenhain, Bericht des 26. Deutschen Chirurgencongresses, 1897.
Heineke, "Experimentelle Untersuchungen über die Todesursache bei Perforationsperitonitis," Deutsch. Arch. f. klin. Med., vol. lxix.
Heinrich, "Beitrag zur Lehre von den Mesenterialcysten," Berlin. klin. Wochenschr., 1895, No. 36.
Hektoen, cited from Virchow-Hirsch's Jahresbericht, 1894, vol. ii., p. 213.
Helferich, "Ueber Perityphlitis," Verhandl. d. XIII. Cong. f. innere Med., Wiesbaden, 1895.
Hemmeter, "Beiträge zur Antiperistaltik des Darms," Arch. f. Verdauungskr., vol. viii.
Hennige, "Die Indicanausscheidung in Krankheiten," Deutsch. Arch. f. klin. Med., vol. xxiii.
Henoch, Klinik der Unterleibskrankheiten, third ed., Berlin, 1863.
Henoch, Berlin. klin. Wochenschr., 1888, No. 29.
Henoch, "Ueber Peritonitis Chronica," ibid., 1874, No. 20.
Henro, "Des pseudo-étranglements que l'on peut rapporter à la paralysie de l'intestin," Thèse de Paris, 1865.
Herczel, "Experimentelle und histologische Untersuchungen über compensatorische Muskelhypertrophie bei Darmstenosen," Zeitschr. f. klin. Med., 1886, vol. xi.

Herff, v., "Ueber schwere Darm- und Magenlähmungen, insbesondere nach Operationen," Zeitschr. f. Geburtsh. u. Gynäk., vol. xliv.

Hermes, "Casuistische Beiträge zur Chirurgie der Leber und Gallenwege," Deutsch. Zeitschr. f. Chir., vol. xli.

Herrlich, "Ueber subphrenische Abscesse," Deutsch. med. Wochenschr., 1886, p. 139.

Herrmann, Pflüger's Arch., 1890, vol. xlvi.

Hervéon, "Cancer primitif du péritoine," Thèse de Paris, 1877.

Herxheimer, Deutsch. med. Wochenschr., 1885, No. 52.

Herzfelder, Ueber die Perforation des Blinddarms und Wurmfortsatzes, Berlin, 1896.

Heubner, "Dysenterie," v. Ziemssen's Handb. d. spec. Pathol. u. Therap., vol. ii.

Heubner, "Ueber das Verhalten des Darmepithels bei Darmkrankheiten der Säuglinge," Zeitschr. f. klin. Med., vol. xxix.

Heyl, Hedwig, Krankenkost, Berlin.

Hiller, "Ueber Darmlipome," Beiträge z. klin. Chir., 1899, vol. xxiv.

Hinterberger, "Neunzehn Fälle von Bauchfelltuberculose," Wien. klin. Wochenschr., 1893, Nos. 38, 39.

Hirsch, "Ueber Enteritis membranacea et mucosa," Inaug. Diss., Berlin, 1891, 1892, No. 17.

Hirsch, C., "Zur klinischen Diagnose der Zwerchfellhernie," Münch. med. Wochenschr., 1900, No. 29.

Hirschler, "Experimentelle Beiträge zur urämischen Diarrhöe," Pester med.-chir. Presse, 1891, No. 30.

Hirschsprung, cited by Goeppert, and referred to in Arch. f. Verdauungskr., vol. vii.

Hlava, "Sur les lésions dites urémiques de l'intestin," Sbornik lékarsky, vol. iv.

Hochenegg, "Ueber cytische Mesenterialtumoren," Wien. klin. Rundschau.

Hochenegg, "Chirurgische Eingriffe bei Blinddarmerkrankungen," Wien. klin. Wochenschr., 1895, Nos. 16, 17, 18, and 20.

Hochenegg, "Ueber eine neue typische Form des acuten Darmverschlusses," ibid., 1897, No. 51.

Hochhaus, "Ueber Magenerweiterung nach Duodenalstenose," Berlin. klin. Wochenschr.

Hochstätter, v., "Colica processus vermiformis," Beiträgen z. Chir., Festchr. für Billroth, Stuttgart, 1892.

Hodenpyl, "On the Etiology of Appendicitis," New York Med. Jour., 1893.

Hoefele, "Häufigkeitstabellen, nach 630 systematischen Fäcesanalysen aufgestellt," Med. Blätter, Vienna, 1902, No. 5.

Hoefele, Handbuch der physiologisch-pathologisch-chemischen Analyse, Berlin, 1883, fifth ed.

Hoffmann, F. A., "Ueber den Eiweissgehalt der Ascitesflüssigkeiten," Virchow's Arch., vol. lxxviii.

Honigmann, "Ueber die Behandlung des Ileus mit Belladonnapräparaten," Centralbl. f. d. Grenzgeb. d. Med. u. Chir., vol. v., No. 7.

Hoppe-Seyler, Physiol. Chem., Berlin, 1878, part ii.

Horvath, Centralbl. f. d. med. Wissensch., 1873, Nos. 38–42.

Huber, Deutsch. Arch. f. klin. Med., vol. vii.

Huber, "Ueber Irrwege bei der Diagnose der Perityphlitis," Correspondenzbl. f. Schweiz. Aerzte, 1901, No. 15.

Hubl, "Primäres Carcinom des Peritoneum," Wien. med. Wochenschr., 1879, No. 52.

Hübler, Sr., "Ein Fall von chronischer Perihepatitis hyperplastica," Berlin. klin. Wochenschr., 1897, No. 51.

Hünerfauth, Ueber die habituelle Obstipation und ihre Behandlung, Wiesbaden, 1885.

Hüter, Grundriss der Chirurgie, second half, Leipsic, 1882.

Huismans, "Ein Fall von tödtlicher parenchymatöser Colonblutung," Deutsch. med. Wochenschr., 1900, No. 47.

Hulsebosch. See van Ledden-Hulsebosch.

Humbert, "Etude sur la septicémie intestinale," Thèse de Paris, 1873.

Hunter, W., Pathol. Trans., 1890.

I

Illoway, Constipation in Adults and Children, New York, 1897.

Immermann, "Aus der med. Klinik in Basel," Deutsch. Arch. f. klin. Med., vol. xii., p. 173.

Israel, "Erfahrungen über operative Behandlung der Bauchfelltuberculose," Deutsch. med. Wochenschr., 1896, No. 1.

Israel, "Einige Beobachtungen an Ileusfällen," Berlin. klin. Wochenschr., 1892, No. 1.

J

Jacusiel, Berlin. klin. Wochenschr., 1887, p. 253.
Jagic, "Zur Histologie der Enteritis membranacea und des Dickdarmkatarrhs," Wien. klin. Rundschau, 1901, No. 41.
Jaksch, v., Klin. Diagnostik innerer Krankheiten, fifth ed., Vienna, 1901.
Johnston, W. W., Amer. Jour. Med. Sci., July, 1888.
Jolly, "Hysterie," v. Ziemssen's Handb. d. spec. Pathol. u. Therap., vol. xii.
Jones, Allan, "Chronic Diarrhea Associated with Achylia Gastrica," Jour. Amer. Med. Assoc., July 30, 1898.
Jones, H., Lancet, June, 1878.
Jonesco, Hernies int. retro-peritonéales ou hernies formées dans les fossettes normales du péritoine, Paris, 1890.
Josué, "Appendicite expérimentale par infection sanguine," Sem. méd., 1897, No. 12.
Journet, "Étude sur le cancer de l'intestin grêle," Thèse de Paris, 1883.
Joy and Wright, "Leukocytosis as a Point of Prognosis in Appendicitis," New York Med. News, April, 1902.
Jürgens, "Atrophia gastro-intestinalis progressiva," Berlin. klin. Wochenschr., 1882, Nos. 23 and 28.

K

Kader, "Ein experimenteller Beitrag zur Frage des localen Meteorismus bei Darm-occlusion," Inaug. Diss., Dorpat, 1891.
Kaiser, "Ueber die operative Behandlung der Bauchempyeme," Deutsch. Arch. f. klin. Med., vol. xvii.
Kassowitz, Beiträge z. Kinderheilk., Vienna, 1892.
Kast and Baas, "Zur diagnostischen Verwerthung der Aetherschwefelsäureauscheidung im Harn," Münch. med. Wochenschr., 1888, No. 4.
Kaufmann, "Ueber den Verschluss der Arteria meseraica superior durch Embolie," Virchow's Arch., vol. cxvi.
Kaulich, "Ueber Peritonealtuberculose," Prager Vierteljahrsschr., 1871.
Keen, American Surgery, 1891, vol. xiii.
Kelling, "Zeitschr. f. klin. Med., vol. xxix.
Kelling, Physikalische Untersuchungen über die Druckverhältnisse in der Bauch-höhle," Samml. klin. Vorträge, 1896, N. F., No. 144.
Kelly, "Pathogenesis of Appendicitis," Phila. Med. Jour., November, 1899.
Kelsch, Arch. de physiol. normale et pathol., 1877.
Kelynack, A Contribution to the Pathology of the Vermiform Appendix, London, 1893.
Kiär, "Ein Fall von einem Darmstein in einem Cöcalbruch," ref. in Arch. f. Ver-dauungskr., vol. ii.
Kijanitzin, "Zur Frage nach der Ursache des Todes bei ausgedehnten Hautverbren-nungen," Virchow's Arch., vol. cxxxi., p. 436.
Kijewski, "Ueber subphrenische Abscesse," Virchow's Jahresber. f. 1893, vol. ii., p. 173.
Kirstein, "Experimentelles zur Pathologie des Ileus," Deutsch med. Wochenschr., 1889, No. 49.
Kirstein, "Zur Casuistik der subcutanen Darmverletzungen," Deutsch. Zeitschr. f. Chir., vol. lvii.
Kissel, "Ueber die Diagnose der tuberculösen Peritonitis bei Kindern," Arch. f. klin. Chir., vol. lxv.
Kitagawa, "Beiträge zur Diagnostik der Darmkrankheiten," Inaug. Diss., Würzburg, 1889.
Kitagawa, "Beiträge zur Kenntniss der Enteritis membranacea," Zeitschr. f. klin. Med., vol. xviii.
Klapp, "Ueber Bauchfellresorption," Mittheil. aus den Grenzgeb. d. Med. u. Chir., vol. x.
Klebs, Arch. f. exper. Pathol. u. Pharmak., 1873, vol. i.
Klebs, Handb. d. pathol. Anat., Berlin, 1868.
Klecki, "Contribution à la pathogénie de l'appendicite," Annales de l'Institut Pasteur, 1899, vol. xiii.
Klecki, "Recherches sur la pathogénie de la péritonite," etc., ibid., 1895.
Klein, E., The Anatomy of the Lymphatic System, London, 1873.
Klob, Wien. med. Wochenschr., 1863.
Kobler, "Beitrag zur Kenntniss der Nierenerscheinungen bei acuten Darmaffec-tionen," etc., Wien. klin. Wochenschr., 1890, Nos. 28–31.

Kobler, "Ueber Nierenerscheinungen bei Obstipation und **Darmkoliken**," ibid., 1898, No. 20.

Kocher, Deutsch. Zeitschr. f. Chir., vol. viii.

Kocher, "Ueber Ileus," Grenzgeb. d. Med. u. Chir., vol. iv.

König, "Die peritoneale Tuberculose und ihre Heilung durch den Bauchschnitt," Centralbl. f. Chir., 1890, No. 35.

König, Deutsch. Zeitschr. f. Chirurgie, 1892, vol. xxxiv.

König, "Ueber diffuse Tuberculose," Centralbl. f. Chir., 1884, No. 6.

Körte, "Ueber chirurgische Behandlung der Perityphlitis," Berlin. klin. Wochenschr., 1891, Nos. 26, 27.

Körte, "Weiterer Bericht über die chirurgischen Erfolge der diffusen eitrigen Bauchfellentzündung," Grenzgeb. d. Med. u. Chir., 1897, vol. ii.

Koester, "Drei Fälle von Venenthrombose der Bauchhöhle," Deutsch. med. Wochenschr., 1898.

Kortum, "Ueber Enterophthise," Inaug. Diss., Berlin, 1879.

Krafft, "Ueber die frühzeitige operative Behandlung der durch Perforation des Wurmfortsatzes hervorgerufenen Perityphlitis stercoralis," Volkmann's Samml. klin. Vorträge, 1889, No. 331.

Kraft, L., "Experimentell-patholog. Studie über acute Peritonitis," Virchow-Hirsch's Jahresbericht, 1891.

Kraske, "Erfahrungen über den **Mastdarmkrebs**," Volkmann's Samml. klin. Vorträge, 1883–84.

Kraus, "Ueber den gegenwärtigen Stand der Lehre von der Enteroptose," Wien. klin. Rundschau, 1900, Nos. 25, 26.

Kraus, F., Prager Wochenschr., 1889, Nos. 6, 7.

Kraus, J., Das perforirende Geschwür im Duodenum, Berlin, 1865.

Kraussold, "Ueber die Krankheiten des Processus vermiformis und des Cöcum," Volkmann's Samml. klin. Vorträge, No. 191.

Kretz, "Phlegmone des Processus vermiformis im Gefolge einer Angina tonsillaris," Wien. klin. Wochenschr., 1900, No. 49.

Krogius, Ueber die vom Processus vermiformis ausgehende diffuse eitrige Peritonitis, Jena, 1901.

Krueger, "Die primären Bindegewebsgeschwülste des Magendarmcanals," Inaug. Diss., Berlin, 1894.

Krupezky, "Ein Fall von syphilitischer Peritonitis," Wien. med. Blätter, 1895, Nos. 40, 41.

Krysinsky, Ueber Enteritis membranacea, Jena, 1884.

Kümmel, Ueber Perityphlitis, Leipsic, 1896.

Küttner, Ueber innere Incarcerationen, Virchow's Arch., 1868, vol. xliii.

Küttner, "Multiple carcinomatöse Darmstricturen durch Peritoneal-Metastasen," Beiträge zur klin. Chir., vol. xxiii.

Kukula, L'étiologie des enterrhagies consécutives à l'étranglement des hernies, Brussels, 1900.

Kukula, "Etiologie des entérrhagies suivantes l'étranglement des hernies," Arch. bohèmes de méd. clin., 1899, p. 247.

Kukula, "Untersuchungen über Autointoxicationen bei Darmocclusionen," Arch. f. klin. Chir., vol. lxiii.

Kundrat (-Widerhofer), Gerhardt's Handbuch der Kinderkrankheiten, Tübingen, 1880, vol. iv., section 2.

Kundrat, "Ueber eine seltene Form der inneren Incarcerationen," Wien. med. Wochenschr., 1891.

Kussmaul, "Die peristaltische Unruhe des Magens," Volkmann's Samml. klin. Vorträge, 1880, No. 181.

Kussmaul, "Zur Embolie der Arteriæ mesentericæ," Würzburger med. Zeitschr., 1864, vol. v.

Kussmaul and Maier, "Aneurysma verminosum hominis, Periarteritis nodosa," Deutsch. Arch. f. klin. Med., 1866, vol. i., pp. 125, 484.

Kussmaul-Cahn, "Heilung von Ileus durch Magenausspülung," Berlin. klin. Wochenschr., 1884, Nos. 42, 43.

Kyber, "Weitere Untersuchungen über die amyloide Degeneration," Virchow's Arch., vol. lxxxi.

L

Lacher, "Ueber Zwerchfellshernien," Deutsch. Arch. f. klin. Med., vol. xxvii.

Laehr, "Ueber subcutane Ruptur der Leber, u. s. w.," Inaug. Diss., Munich, 1890.

Lambl, "Mikroskopische Untersuchungen der Darmexcrete," Prager Vierteljahrsschr., 1859, vol. i.

Lampe, "Ueber subphrenische Abscesse," Münch. med. Wochenschr., 1895.

Landau, "Ueber den frühen Nachweis von freier Bauchwassersucht," Centralbl. f. Gynak., 1900, No. 45.

de Langenhagen, Semaine médicale, 1898, No. 1.

de Langenhagen, "Six cents cas d'entéro-colite muco-membraneuse," XIII. Internat. Cong. Med., Paris, 1900.

Langley and Anderson, Jour. Physiol., vols. xii., xvi., xvii., xviii., xix., xx.

Langmann, "On Antiperistaltic Movement," Festschr. in honor of Jacobi, New York, 1900.

Lapponi, Virchow-Hirsch's Jahresbericht, 1895.

Larcher, Arch. de méd., 1864–65.

Laruelle, "Etude bactériologique sur les péritonites par perforation," La Cellule, 1889, vol. v.

Lauenstein, "Zur Pathologie der Leukämie," Deutsch. Arch. f. klin. Med., vol. xviii.

Lauenstein, "Verwachsungen und Netzstränge im Leibe als Ursache andauernder schwerer Koliken," Arch. f. klin. Chir., 1892, vol. xlv.

Ledden-Hulsebosch, van, Makro- und mikroskopische Diagnostik der menschlichen Excremente, Berlin, 1899.

Leichtenstern, "Die Charcot-Robin'schen Krystalle in den Fäces," Deutsch. med. Wochenschr., 1892, No. 25.

Leichtenstern, "Der Ileus und seine Behandlung," Verhandl. d. Cong. f. innere Med., Wiesbaden, 1889.

Leichtenstern, "Verengerungen, Verschliessungen, und Lageveränderungen des Darms," v. Ziemssen's Handb. d. spec. Pathol u. Therap., Leipsic, 1878, vol. vii., second half.

Leichtenstern, Prager Vierteljahrsschr., vols. cxviii. to cxxi.

Lennander, Ueber Appendicitis, Vienna, 1895.

Lennander, "Ueber die Appendicitis und ihre Complicationen," Samml. klin. Vorträge, N. F., Leipsic, 1893, No. 75.

Lennander, "Ueber Operationen der Gallenwege und Adhärenzbildungen im oberen Theile des Bauches," Wien. klin. Wochenschr., 1893, Nos. 37, 38, 39.

Lennander, "Ueber die Sensibilität der Bauchhöhle, u. s. w.," Centralbl. f. Chir., 1901, No. 8.

Lennander, "Beobachtungen über die Sensibilität in der Bauchhöhle," Grenzgeb. d. Med. u. Chir., vol. x.

Lennander, "Acute eitrige Peritonitis," Deutsch. Zeitschr. f. Chir., vol. lxiii.

Lenzmann, Die entzündlichen Erkrankungen des Darms in der Regio ileo-cöcalis, Berlin, 1901.

Leo, "Ausscheidung von Salol-Concrementen durch die Fäces," Sitzungsber. d. Niederrhein. Gesellsch. f. Natur- und Heilk. in Bonn, 1899.

Letcheff, "De la colite muco-membraneuse chez les utérines," Thèse de Paris, 1895.

Leube, "Ueber Darmschwindel," Deutsch. Arch. f. klin. Med., 1885, vol. xxxvi.

Leube, "Behandlung des Magengeschwürs," Grenzgeb. d. Med. u. Chir., 1897, vol. ii.

Leube, Specielle Diagnose der inneren Krankheiten, Leipsic, 1898.

Leube, Verhandl. deutscher Naturforscher und Aerzte in Düsseldorf, 1898.

Leube and Salkowski, Die Lehre vom Harn, Berlin, 1882.

Leubuscher, Experimentelle Beiträge zur Aetiologie der Darminvagination," Virchow's Arch., vol. lxxxv.

Leyden, v., "Ueber Pyopneumothorax subphrenicus," Zeitschr. f. klin. Med., 1880, vol. i.

Leyden, v., "Ueber einen Fall von Perityphlitis durch Perforation des Processus vermiformis," Berlin. klin. Wochenschr., 1889, No. 31.

Leyden, v., Verhandlungen des Vereines für innere Medicin in Berlin, 1882.

Leyden, v., "Ueber spontane Peritonitis," ibid., Berlin, 1883–84, vol. iii.

Leyden, v., and Renvers, "Ueber Pyopneumothorax subphrenicus und dessen Behandlung (mit Discussion)," Berlin. klin. Wochenschr., 1892, No. 46.

Leyden, v., and Schaudin, "Leydenia gemmipara," Sitzungsber. d. königl. preussischen Akad. d. Wissensch. zu Berlin, 1896, vol. xxxix.

Lewin, "Der heisse Umschlag als diagnostisches Hilfsmittel," Blätter f. klin. Hydrotherapie, 1900.

Libmann, "Ueber Dünndarmsarkome," Grenzgeb. d. Med. u. Chir., vol. vii.

Lichtenstein, Wien. med. Blätter, 1894, No. 3.

Lichtheim, "Zur diagnostischen Verwerthung der Tuberkelbacillen," Fortschr. d. Med., vol. i., No. 1.

Liebermeister, Vorlesungen über die Krankheiten der Unterleibsorgane, Leipsic, 1894.

Lindner, "Ueber die operative Behandlung der Bauchfelltuberculose," Deutsch. Zeitschr. f. klin. Chir., 1892, vol. xxxiv.

Link, "Mannsfaustgrosses Lipom im Colon desc.," Wien. klin. Wochenschr., 1890, No. 13.

Litten, "Enteritis membranacea," Berlin. klin. Wochenschr., 1888, No. 29.

Litten, "Discussion über Peritonitis," Verhandl. d. Vereines f. innere Med. z. Berlin, 1883–84, p. 309, No. 3.

Litten, Deutsch. med. Wochenschr., 1887, No. 27.

Litten, "Zur Diagnostik der Flüssigkeitsansammlung im Cavum abdominale," etc., Zeitschr. f. klin. Med., 1881, vol. ii.

Litten, "Ueber die Folgen des Verschlusses der Arteria meseraica superior," Virchow's Arch., vol. lxiii.

Litten, "Ueber circumscripte gitterförmige Endarteritis," Deutsch. med. Wochenschr., 1889, No. 8.

Lode, "Ein subseröses Myom des Ileum," Wien. klin. Wochenschr., 1894, Nos. 21, 22.

Lonart, "Le cancer du gros intestin (rectum excepté) dans la jeunesse," Thèse de Paris, 1900.

Lop, "Typhlite suppurée sans lésion de l'appendice," Rev. de méd., 1897, p. 648.

Lorenz, H., "Beitrag zur Kenntniss der multiplen degenerativen Neuritis," Zeitschr. f. klin. Med., vol. xviii., Nos. 5, 6.

Lorenz, H., "Untersuchungen über Acetonurie, etc.," Zeitschr. f. klin. Med., 1891, vol. xix.

Louyer-Villermay, Arch. gén. de méd., 1824, vol. v.

Lower, "Spontaneous Rupture of the Bowel," Med. Record, October 5, 1895.

Lubarsch, "Ueber den primären Krebs des Ileum, etc.," Virchow's Arch., vol. cxi.

Luederitz, Verhandlungen des X. internationalen medicinischen Congresses, second sect., Berlin, 1891.

Luederitz, "Experimentelle Untersuchungen über das Verhalten der Darmbewegungen bei herabgesetzter Körpertemperatur," Virchow's Arch., vol. cxvi.

Luschka, "Ueber die peritoneale Umhüllung des Blinddarms," ibid., vol. xxi.

Luschka, "Ueber polypöse Vegetationen der gesammten Dickdarmschleimhaut," ibid., 1861, vol. xx.

Lynch, "Coprologia," Tesis, Buenos Ayres, 1896.

M

McBurney, "Reihe von Aufsätzen über Appendicitis," New York Med. Jour., 1888–91, in New York Med. Rec., 1892, in Annals of Surgery, 1891 and 1894.

Madelung, Centralbl. f. Chir., 1892, No. 30.

Mainzer, "Histol. Beiträge zur Entstehung und Structur der Hämorrhoidalknoten," Inaug. Diss., Würzburg, 1894.

Mall, Johns Hopkins Hospital Reports, 1889, vol. i.

Malvoz, "Le bacterium coli commune comme agens habituel des péritonites d'origine intestinale," Arch. de méd. expér., 1891, vol. iii.

Maly, Hermann's Handbuch der Physiologie, 1880, vol. v.

Mannaberg, "Ueber Accentuirung des zweiten Pulmonaltones bei Perityphlitis," Centralbl. f. innere Med., 1894, No. 10.

Mannaberg, "Pathogenese und pathologische Anatomie der Colitis membranacea," XIII. Internat. Cong. Med., Paris, 1900.

Mannaberg, "Diagnostische Bemerkungen zur einigen Unterleibskrankheiten," Wien. med. Wochenschr., 1902, Nos. 13, 14.

Maragliano, "Eine besondere Form von geschwüriger Darmentzündung," Berlin. klin. Wochenschr., 1894, No. 13.

Marchand, "Ueber eigenthümlich verzweigte Gerinnsel in den Darmausleerungen," ibid., 1877.

Marcus, "Ueber die Resorption von Bakterien aus dem Darm," Zeitschr. f. Heilk., vol. xx.

Marfan, cited by Goeppert.

Markwald, "Drei Fälle von Darmblutung beim Typhus," Inaug. Diss., Berlin, 1869.

Martius, Achylia gastrica, ihre Ursachen und Folgen, Leipsic and Vienna, 1897.

Mathieu, "Traitement de colite muco-membraneuse," XIII. Internat. Cong. Med., Paris, 1900.

Matterstock, "Perityphlitis," Gerhardt's Handb. d. Kinderkrankh., Tübingen, 1880, vol. iv., second sec.

Mauclaire, Bull. méd., October, 1899.

Maurin, "Essai sur l'appendicite et la péritonite appendiculaire," Thèse de Paris, 1890.

Maydl, Ueber subphrenische Abscesse, Vienna, 1894.

Maydl, Ueber den Darmkrebs, Vienna, 1883.

Mayer, S., and Basch, v., Sitzungsberichte der Wiener Akademie, 1870, vol. lxii.

Mayor, "Quelques mots sur une variété d'entérite iliaque," Rev. méd. de Suisse romande, 1893.

Mazzoni, Il Policlinico, 1895.

Meinert, "Ueber Enteroptose," Jahresber. d. Gesell. f. Natur- und Heilk. zu Dresden, 1891–92.

Mêlier, Arch. gén. de méd., 1828, vol. xvii.

Meltzer, "On Subphrenic Abscess," New York Med. Jour., June 24, 1893.

Meltzer and Adler, "Experimental Contribution to the Study of the Path by which Fluids are Carried from the Peritoneal Cavity into the Circulation," Jour. Exper. Med., 1896, vol. i., No. 3.

Meltzing, "Enteroptose und intraabdominaler Druck," Arch. f. Verdauungskrankh., vol. iv.

Menge, "Ueber die gonorrhoische Erkrankung der Tuben und des Bauchfells," Zeitschr. f. Geburtsh. u. Gynäk., 1891, vol. xxi.

Mertens, "Falsche Divertikel der Flexura sigmoidea und des Proc. vermiformis," Grenzgeb. d. Med. u. Chir., vol. ix.

Meschede, "Zur Casuistik der Lungen- und Darmsyphilis," Virchow's Arch., vol. xxxvii.

Metichnikow, Gaz. hebd. de méd. et de chir., 1901, No. 22.

Meusser, "Ueber Appendicitis und Typhlitis mit cachirtem und ungewöhnlichem Verlauf," Mittheil. aus den Grenzgeb. d. Med. u. Chir., vol. ii.

Meyer, Ernst, "Anatomische Beiträge zur Lehre von der Darmatrophie," Inaug. Diss., Bonn, 1900.

Meyer, Willy, "When Shall we Operate for Appendicitis?" New York Med. Rec., February 29, 1896.

Micheli and Matticolo, Rivista critica di clinica, 1900, No. 4.

Mikulicz, "Weitere Erfahrungen über die operative Behandlung der Perforationsperitonitis," Arch. f. klin. Chir., 1889, vol. xxxix.

Mikulicz, "Die chirurgische Behandlung des chronischen Magengeschwürs," Grenzgeb. d. Med. u. Chir., 1897, vol. ii.

Minkowski, Naunyn's Mittheil. aus der med. Klinik in Königsberg, p. 59, Leipsic, 1888.

Minkowski, "Ueber die Synthese des Fettes aus Fettsäuren im Organismus des Menschen," Arch. f. exper. Pathol. u. Pharmak., vol. xxi.

Möbius, Erlenmeyer's Centralbl. f. Nervenheilk., 1884, seventh year, No. 1.

Möbius, Dieses Handbuch, vol. xii.

Moisejew, "Zur Pathologie und Aetiologie der Enteritis phlegmonosa acuta idiopathica," ref. im Arch. f. Verdauungskrankh., 1900, vol. vi.

Molinari, "Esiste una peritonite cronica essudativa idiopatica?" Riforma med., August, 1889.

Moos, "Beitrag zur Casuistik der embolischen Gefässkrankheiten," Virchow's Arch., vol. xli.

Moritz, "Beitrag zur Lehre von den exsudaten und Transsudaten," Inaug. Diss., Munich, 1886.

Moritz, "Ueber den durch Essigsäure fällbaren Eiweisskörper in Exsudaten," Münch. med. Wochenschr., 1902, No. 42.

Moroux, "Des rapports de la cirrhose du foie avec la péritonite tuberculeuse," Thèse de Paris, 1883.

Moser, "Ueber Peritonitis sero-fibrinosa bei Typhus abdominalis," Grenzgeb. d. Med. u. Chir., vol. viii.

Moskowicz, "Ueber Perityphlitis acuta," ibid., vol. x.

Mouisset, "Ulcérations intestinales chez les arteriosscléreux," Lyon méd., December, 1900.

Le Moyne, "Contributions à l'étude de l'occlusion intestinale," Thèse de Paris, 1878.

Mühsam, "Experimentelles zur Frage der Antiperistaltik," Grenzgeb. d. Med. u. Chir., vol. vi.

Mühsam, "Ueber Appendicitisexperimente," Deutsch. Zeitschr. f. Chir., vol. lv.

Mühsam, " Beiträge zur Differentialdiagnose der Appendicitis," Berlin. klin. Wochenschr., 1899, No. 31.

Müller, Mittheilungen aus der med. Klinik zu Würzburg, 1886.

Müller, " Einige Beobachtungen aus dem Percussionscurs," Berlin. klin. Wochenschr., 1895, No. 13.

Müller, Fr., " Untersuchungen über Icterus," Zeitschr. f. klin. Med., 1887, vol. xii.

Müller, H. F., Ueber die Beulenpest in Bombay im Jahre 1897, Vienna, 1898, Pt. i.

Müller, Max, " Beiträge zur Kenntniss der Metastasenbildung maligner Tumoren," Inaug. Diss., Bern, 1892.

Münstermann, " Ueber Bauchfelltuberculose," Inaug. Diss., Munich, 1890.

Muron, Gaz. méd. de Paris, 1873, No. 11.

McMurtry, Medical News, 1891.

Muscatello, " Ueber den Bau und des Aufsaugungsvermögen des Peritoneum," Virchow's Arch., vol. cxlii.

N

Nannoti and Bacciocili, " Esperimenti sugli effetti della laparotomia nelle peritoniti tuberculare," Il Policlinico, 1894.

Nasse, Beiträge zur Physiologie der Darmbewegung, Leipsic, 1866.

Naunyn, Klinik der Cholelithiasis, Leipsic, 1892.

Naunyn, " Ueber Ileus," Mittheil. aus den Grenzgeb. d. Med. u. Chir., 1896, vol. x., No. 1.

Nepveu, Présence des bacteries et des cercomonas intestinales dans la sérosité péritonéale de la hernie étranglée et de l'occlusion intestinale, Paris, 1883.

Neugebauer, " Zur Casuistik der Darmverletzungen," Wien. klin. Wochenschr., 1899, No. 3.

Neumann, E., " Zur Kenntniss der fibrinoiden Degeneration des Bindegewebes bei Entzündungen," Virchow's Arch., vol. cxliv.

Neumann, " Darmdivertikel und persistirende Dottergefässe als Ursache für Darmincarcerationen. Internationale Beiträge zur wissenschaftlichen Medicin," Virchow's Festzeitschr., Berlin, 1891, vol. ii.

Neumann, Arch. d. Heilk., 1868, vol. ix.

Neumann, Alfred Egon, " Zur Frage der operativen Behandlung des Ascites bei Lebercirrhose," Deutsch. med. Wochenschr., 1899, No. 26.

Neusser, E., " Zur Casuistik des Pyopneumothorax subphrenicus," Wien. med. Wochenschr., 1884, Nos. 44–47.

Nickel, " Ueber die sogenannten syphilitischen Mastdarmgeschwüre," Virchow's Arch., vol. cxxvii.

Noack, J., " Peritoneale Verwachsungen nach schweren Bauchquetschungen," Grenzgeb. d. Med. u. Chir., vol. iv.

Nobel, Le, " Ein Fall von Fettstuhlgang mit gleichzeitiger Glykosurie," Deutsch. Arch. f. klin. Med., vol. xliii.

Noeggerath, Die latente Gonorrhœ beim Weibe, Bonn, 1872.

Noorden, v., " Ueber die Behandlung der Colica mucosa," Zeitschr. f. prakt. Aerzte, 1898, No. 1.

Noorden, v., " Das Physostigmin gegen Erschlaffung des Darms," Berlin. klin. Wochenschr., 1901, No. 42.

Nothnagel, " Ueber compensatorische Muskelhypertrophie," Zeitschr. f. klin. Med., 1886, vol. x., p. 208 ff.

Nothnagel, " Zur Klinik der Darmblutungen," Festschr. f. M. Jaffe, Braunschweig, 1901.

Nothnagel, " Ueber idiopathische Peritonitis," Wien. klin. Rundschau, 1901, No. 23.

Nothnagel, " Pseudoperityphlitis," Wien. klin. Wochenschr., 1899, No. 15.

Nothnagel and Rossbach, Handb. der Arzneimittellehre, Berlin, 1887, sixth ed., Literatur on p. 590 ff. and on p. 899.

O

Obermayer and Schnitzler, " Ueber die Durchlässigkeit der lebenden Darm- und Gallenblasenwand für Gase," Centralbl. f. d. med. Wissensch., 1894, No. 29.

Obrastzoff, " Zur Diagnose des Blinddarmcarcinoms, der Blinddarmtuberculose, etc.," Arch. f. Verdauungskrankh., vols. iv., vi.

Oeder, " Ueber Lagerungsbehandlung bei Hämorrhoiden," Zeitschr. f. physik. u. diätet. Therap., vol. iv.

Oesterlein, Mittheil. aus d. med. Klinik in Würzburg, 1885, vol. i.

Oettingen, R. v., Enterostomie und Laparotomie bei acuter innerer Darmocclusion, bedingt durch Volvulus, Strangulation, und Inflexion, Dorpat, 1888.

Opitz, "Durchgängigkeit von Darm und Nieren für Bakterien," Zeitschr. f. Hyg. u. Infectionskrankh., vol. xxix.

Oppel, Experimentelle acute Mikrobenperitonitis, Wratsch, 1902, No. 11.

Oppenheimer, "Das Ulcus pepticum duodenale," Inaug. Diss., Würzburg, 1891.

Oppler, Deutsch. med. Wochenschr., 1896, No. 82.

Oppolzer, Allge. Wien. med. Zeitschr., 1866.

Orlow, Pflüger's Arch. f. Physiol., vol. lix.

Orth, "Experimentelle Untersuchungen über Fütterungstuberculose," Virchow's Arch., 1879, vol. lxxvi.

Orth, "Experimentelles über Peritonitis," Internat. klin. Rundschau, 1890, No. 41.

Ortweiler, Mittheilungen aus der med. Klinik zu Würzburg, Wiesbaden, 1886, vol. ii.

Oser, Die Erkrankungen des Pankreas, Vienna, 1898, vol. xviii. of this handbook. In vol. on Diseases of Pancreas, Liver, and Suprarenal Glands. English edition.

Oser, Arch. f. Dermatol. u. Syphilis, 1871.

Oser, Wien. med. Blätter, 1884, No. 48.

Ott, Prager Zeitschr. f. Heilk., 1896, vol. xvii.

P

Pagenstecher, E., "Zwei Fälle von Cystenbildung im Mesenterium," Berlin. klin. Wochenschr., 1895, No. 42.

Paijkull, ref. i. Jahresber. f. Thierchemie, 1892.

Pal, "Ueber den Einfluss der Temperatur auf die Erregbarkeit des Darms," Wien. klin. Wochenschr., 1893, No. 2.

Pal, "Primäre submucöse circumscripte Colitis," ibid., 1897, Nos. 18, 19.

Pal, "Ueber den motorischen Einfluss des Splanchnicus auf den Dünndarm," Arch. f. Verdauungskrankh., vol. v.

Pal, "Ueber Beziehungen zwischen Circulation, Motilität und Tonus des Darms," Wien. med. Presse, 1901, No. 44.

Parenski, Wien. med. Jahrbücher, 1876, No. 3.

Pariser, Deutsch. med. Wochenschr., 1893, No. 41.

Pascheles, "Ueber den Zuckergehalt pathologischer Flüssigkeiten," Wien. klin. Wochenschr., 1896, No. 17.

Pawlowsky, "Zur Lehre von der Aetiologie der Entstehungsweise und der Formen der acuten Peritonitis," Virchow's Arch., vol. cxvii.

Pel, "Ueber ein eigenthümliches Vorkommen von weissen thonartigen Stühlen," Centralbl. f. klin. Med., 1887, vol. viii., No. 17.

Pennington, Jour. Amer. Med. Assoc., 1900, vol. xxxv.

Penzoldt, "Behandlung der Erkrankungen des Darms," Penzoldt's and Stintzing's Handb. d. spec. Therap. innerer Krankh., Jena, 1896.

Périer, Gaz. des hôp., 1891, No. 101.

Perry and Shaw, Guy's Hospital Reports, 1894.

Petitbien, "Des ulcérations intestinales dans l'érisipèle," Thèse de Paris, 1883.

Petrina, "Ueber Carcinoma peritonei," Prager Vierteljahrsschr., 1872, vol. ii.

Petrow, "Polyposis gastro-intestinalis adenomatosa," ref. in Arch. f. Verdauungskrankh., vol. ii.

Peyer, Die chronische nervöse oder reflectorische Diarrhöe, Basel, 1884.

Peyrot, "De l'intervention chirurgique dans l'obstruction intestinale," Thèse de Paris, 1880.

Pfeufer, Zeitschr. für rationelle Medicin, 1851, N. F., vol. i.

Pflüger, Ueber das Hemmungs- und Nervensystem für die peristaltischen Bewegungen der Gedärme, Berlin, 1857.

Pfuhl, "Ein oberhalb der Leber gelegenes peritonitisches Exsudat, etc.," Berlin. klin. Wochenschr., 1877, No. 5.

Pfungen, R. v., "Ueber Darmfäulniss bei Obstipation," Zeitschr. f. klin. Med., 1892, vol. xxi.

Phillips, "Die Resultate der operativen Behandlung der Bauchfelltuberculose," Gekrönte Preisschr., Göttingen, 1890.

Pic, "Contribution à l'étude du cancer primitif du duodénum," Rev. de méd., 1894, No. 12, and 1895, No. 1.

Pick, Friedel, "Ueber chronische, unter dem Bilde der Lebercirrhose verlaufende Pericarditis (Pericarditische Pseudolebercirrhose)," Zeitschr. f. klin. Med., vol. xxix.

Pickardt, "Zur Kenntniss der Chemie pathologischer Ergüsse," Berlin. klin. Wochenschr., 1897, No. 39.

Pilliet, "Thrombose des veines mesaraiques," Progrès méd., 1890, No. 25.

Pilz, Jahrbuch für Kinderheilk., N. F., vol. iii., No. 1.

Pincus, "Ueber die Constipatio myogenita mulierum chronica," Arch. f. Gynäkol., vol. liii.

Pölchen, "Zur Aetiologie der stricturirenden Mastdarmgeschwüre," Virchow's Arch., vol. cxxvii.

Pohl, Prager med. Wochenschr., 1883, No. 21.

Poland, Guy's Hospital Reports, 1859, ser. 3, vol. iv.

Ponfick, "Ueber die plötzlichen Todesfälle nach schweren Verbrennungen," Berlin. klin. Wochenschr., 1877, No. 47.

Ponfick, "Ueber die Wechselwirkung zwischen örtlicher und allgemeiner Tuberculose," ibid., 1890, No. 40.

Ponfick, "Anatomische Studien über den Typhus recurrens," Virchow's Arch., vol. lx.

Ponfick, "Zur Casuistik der Embolie der Arteria mesenterica superior," ibid., vol. l.

Posner and Lewin, Internation. Centralbl. f. Krankh. der Harn. und Geschlechtsorgane, 1896.

Pouzet, "Des rétrécissements fibreux de l'intestin grêle," Thèse de Paris, 1877.

Predöhl, "Untersuchungen zur Aetiologie der Peritonitis," Münch. med. Wochenschr., 1890.

Preindlsberger, "Ueber Darmblutungen nach Reposition incarcerirter Hernien," Wien. klin. Wochenschr., 1901, No. 14.

Pribram, "Der acute Gelenkrheumatismus," this handbook, vol. v.

Pribram, "Ueber die Therapie der Bauchfelltuberculose," Prager med. Wochenschr., 1887, No. 35.

Puchelt, System der Medicin, Heidelberg, 1829.

Q

Quincke, "Ueber fetthaltige Transsudate," Deutsch. Arch. f. klin. Med., 1875, vol. xvi.

Quincke, "Ueber die geformten Bestandtheile in Transsudaten," Deutsch. Arch. f. klin. Med., vol. xxx.

Quincke, "Ueber Ascites," ibid., 1882, vol. xxx.

Quincke, "Krankheiten der Gefässe," v. Ziemssen's Handb. d. spec. Pathol. u. Therap., Leipsic, 1879, vol. vi.

Quincke, "Ueber die Farbe der Fäces," Münch. med. Wochenschr., 1896, No. 36.

R

Radziejewski, Reichert's and Du Bois-Reymond's Arch. f. Anat. u. Physiol., 1870.

Rählmann, Duetsch. med. Wochenschr., 1887, No. 27.

Rafinesque, "Étude sur les invaginations intestinales chroniques," Thèse de Paris, 1878.

Raiser, "Beiträge zur Kenntniss der Darmbewegungen," Diss., Giessen, 1895.

Ranschoff, Trans. Amer. Surg. Assoc., 1890, vol. viii.

Raschkow, "Histologische und klinische Beiträge zur örtlichen Behandlung der Hämorrhoiden," Inaug. Diss., Breslau, 1898.

Rauenbusch, "Beiträge zur Localisation und Verbreitungsweise der eitrigen Peritonitis," Grenzgeb. d. Med. u. Chir., vol. x.

Reach, "Die multiplen Darmstricturen," Centralbl. f. d. Grenzgeb. d. Med. u. Chir., 1900, vol. iii.

Recklinghausen, v., "Ueber retrograde Strömung in Lymphgefässen," Virchow's Arch., vol. c.

Recklinghausen, v., Handbuch der Allgemeinen Pathologie, Stuttgart, 1883.

Recklinghausen, v., "Zur Fettresorption," Virchow's Arch., vol. xxvi.

Reckmann, "Ueber Ulcus duodenale und seine Diagnose," Inaug. Diss., Berlin, 1893.

Rehn, "Die Erkrankungen des Bauchfells," Handb. d. Kinderkrankh., vol. iv., pub. by Gerhardt, Tübingen, 1880.

Rehn, "Gallensteinileus," Arch. f. klin. Chir., vol. lx.

Reichel, Beiträge zur Aetiologie und chirurgischen Therapie der septischen Peritonitis, Leipsic, 1889.

Reichel, "Zur Pathologie des Ileus und Pseudo-Ileus," Sitzungsber. d. Würzburger med. Gesell., 1892, No. 7.

Reichmann, ref. i. Arch. f. Verdauungskrankh., vol. vii., p. 113.

Reinbach, "Pathol. Anat. und klin. Beiträge zur Lehre von den Hämorrhoiden," Beiträge z. klin. Chir., vol. xix.

Reinhardt, Charité-Annalen, Berlin, 1851, No. 1.

Remak, Diagnostische und pathogenetische Untersuchungen, Berlin, 1845.

Renvers, "Zur Pathologie und Therapie der Perityphlitis," Deutsch. med. Wochenschr., 1891.

Renvers, "Ueber Blinddarmentzündung und ihre Behandlung," Festschr. des Friedrich-Wilhelm Institutes, Berlin, 1895.

Reuss, "Beiträge zur klin. Beurtheilung von Exsudaten und Transsudaten," Deutsch. Arch. f. klin. Med., vols. xxiv., xxviii.

Ribbert, "Beiträge zur normalen und pathologischen Anatomie des Wurmfortsatzes," Virchow's Arch., vol. cxxxii.

Riedel, "Ileus, bedingt durch Schrumpfung des Mesenterium von Cöcum sammt unterem Ileumende, desgleichen vom Mesenterium, S romanum," Mittheil. aus den Grenzgeb., vol. ii.

Riedel, "Die Entfernungen von Narbensträngen und Verwachsungen, entstanden durch entzündliche Processe in der Gallenblase und in den weiblichen Genitalien," Correspondenzbl. d. allge. ärztl. Vereines von Thüringen, Weimar, 1891.

Riedel, "Ueber Adhäsiventzündungen in der Bauchhöhle," Langenbeck's Arch., vol. xlvii.

Riedel, "Ueber Peritonitis chronica non-tuberculosa und ihre Folgen : Verengerung des Darms und Dislocation der rechten Niere," Arch. f. klin. Chir., vol. lvii.

Riedel, "Ein Fall von chronischer, idiopathischer, exsudat. Peritonitis," Münch. med. Wochenschr., 1892.

Riedel, "Vorbedingungen und letzte Ursachen des plötzlichen Anfalls von Wurmfortsatzentzündung," Arch. f. klin. Chir., vol. lxvi.

Riegel, Deutsch. med. Wochenschr., 1889, No. 30.

Riesman, "Hemorrhagic Infarction of the Mesentery and Intestine," Proc. Path. Soc. Phila., February 1, 1899.

Robinson, "Rheumatism as a Cause of Appendicitis," Med. Record, September 14, 1895.

Röersch, Rev. de chir., 1893.

Roger and Josué, "Recherches expérimentales sur l'appendicite," Rev. de méd., 1896.

Rokitansky, Handb. d. pathol. Anat., Vienna, vol. iii.

Rokitansky, "Ueber Darmeinschnürungen," Oesterreichische med. Jahrb., 1836, vol. x.

Roloff, "Ueber die Rolle des Pleuroperitonealendothels bei der Entstehung bindegewebiger Adhäsionen," Arbeiten aus dem pathol.-anat. Institute zu Tübingen, Braunschweig, 1896, vol. ii.

Romberg, Lehrbuch der Nervenkrankheiten, Berlin.

Romberg and Paessler, "Exper. Untersuchungen über die allge. Pathol. der Kreislaufsstärung bei acuten Infectionskrankh.," Deutsch. Arch. f. klin. Med., vol. lxiv.

Rose, Edm., "Die offene Behandlung der Bauchhöhle bei der Entzündung des Wurmfortsatzes," Deutsch. Zeitschr. f. Chir., vols. lvii., lviii., lix.

Rose, Ulr., "Ueber den Verlauf und die Heilbarkeit der Bauchfelltuberculose ohne Laparotomie," Grenzgeb. d. Med. u. Chir., vol. viii.

Rosenbach, "Ueber das antagonistische Verhalten der Jod- und Salicylpräparate bezüglich der Ausscheidung in Gelenke, Exsudate und Transsudate," Berlin. klin. Wochenschr., 1890, No. 36, and Münch. med. Wochenschr., 1901, No. 14.

Rosenbach, "Die Perihepatitis simplex nebst Bemerkungen über Peristaltik, Plethora abdominalis und hämorrhoidale Zustände," Arch. f. Verdauungskrankh., vol. i.

Rosenbach, "Ueber eine eigenthümliche Farbstoffbildung bei schweren Darmleiden," Berlin. klin. Wochenschr., 1889–90.

Rosenbach, "Die Erkrankungen des Brustfells," this handbook, vol. xiv. In vol. on Diseases of Lungs and Pleura, English edition.

Rosenbach, "Zur Symptomatologie und Therapie der Darminsufficienz," Berlin. klin. Wochenschr., 1889, Nos. 13, 14.

Rosenfeld, "Ueber das Pigment der Hämochromatose des Darms," Arch. f. exper. Pathol., vol. xlv.

Rosenheim, "Ueber Tanocol," Berlin. klin. Wochenschr., 1899, No. 22.

Rosenstein, "Eine Beobachtung von anfallsweisen Kotherbrechen," ibid., 1882, No. 34.

Rosenstein, Die Pathologie und Therapie der Nierenkrankheiten, second ed., Berlin, 1870.

Roser, cited by Goeppert.

Rossbach, Nothnagel and Rossbach, Handbuch der Arzneimittellehre.

Rossi, "Sulla diagnosi della peritonite idiopatica cronica," Rivista clinica e terapeutica, June, 1886.

Roth, "Ueber Divertikelbildung am Duodenum," Virchow's Arch., vol. lvi.

Rothmann, M., "Beitrag zur Pathologie der Enteritis membranacea," Zeitschr. f. klin. Med., vol. xxiii.

Rothmann, O., "Enteritis membranacea," Verhandl. d. Vereins f. innere Med. zu Berlin, seventh year.

Rotter, "Polyposis recti—adenoma malignum—Spontanheilung," Arch. f. klin. Chir., vol. lviii.

Roux, Congrès français de chir., Lyon, 1894.

Rubner, "Physiologie der Nahrung und der Ernährung," v. Leyden's Handb. d. Ernährungstherapie, Leipsic, 1897, vol. i.

Ruepp, "Ueber den Darmkrebs," Inaug. Diss., Zurich, 1895.

Rumpel and Mester, "Bedeutung und Ursache der sogenannten Rosenbach'schen Reaction," Centralbl. f. klin. Med., 1891.

Rumpf, H., "Ueber die Zuckergussleber," Deutsch. Arch. f. klin. Med., vol. lv.

Runeberg, "Von der diagnostischen Bedeutung des Eiweissgehaltes in pathologischen Trans- und Exsudaten," Berlin. klin. Wochenschr., 1897, No. 33.

Runeberg, "Klinische Studien über Transsudationsprocesse," Deutsch. Arch. f. klin. Med., vols. xxxiv., xxxv.

Rydygier, "Zur Behandlung der Darminvaginationen," Verhandl. d. deutschen Gesellschaft f. Chir., 1895, vol. xxiv.

S

Sachs, "Der subphrenische Abscess," Langenbeck's Arch., vol. l.

Sachs, Deutsch. med. Wochenschr., 1892.

Sahli, "Ueber das Wesen und die Behandlung der Perityphlitiden," Correspondenzbl. f. Schweizer Aerzte, Basel, 1892.

Sahli, "Ueber die Pathologie und Therapie der Perityphlitis," Verhandl. d. XIII. Cong. f. innere Med., Wiesbaden, 1895.

Salkowski, "Eigenthümliche Farbstoffbildung bei schweren Darmleiden," Berlin. klin. Wochenschr., 1889.

Salzer, "Ueber peritoneale Verwachsungen," Wien. klin. Wochenschr., 1893, No. 23.

Salzer, "Beiträge zur Pathologie und chirurgischen Therapie chronischer Cöcumerkrankungen," Langenbeck's Arch., vol. xliii., No. 1.

Samson, v., "Einiges über den Darm, etc.," Arch. f. klin. Chir., vol. xliv.

Sasaki, "Veränderungen in den nervösen Apparaten der Darmwand bei perniciöser Anämie, etc.," Virchow's Arch., vol. xcvi.

Scheimpflug, "Beitrag zur pathologischen Histologie des Darms," Zeitschr. f. klin. Med., vol. ix.

Schiller, "Beitrag zur pathologischen Bedeutung der Darmparasiten, besonders für die Perityphlitis," Beiträge z. klin. Chir., 1902, vol. xxxiv.

Schlaefke, "Beitrag zur Casuistik der Perityphlitis," Münch. med. Wochenschr., 1895, No. 32.

Schlange, "Ueber den Ileus," Volkmann's Samml. klin. Vorträge, 1894, No. 101.

Schlange, "Zur Ileusfrage," Arch. f. klin. Chir., 1889, vol. xxxix.

Schleiffarth, "Ueber die Entzündung der serösen Organbedeckungen und der Gehirnhäute," Virchow's Arch., vol. cxxix.

Schloffer, "Ueber Ileus bei Hysterie," Beiträge z. klin. Chir., vol. xxiv.

Schloffer, "Ueber traumatische Darmverengerungen," Grenzgeb. d. Med. u. Chir., vol. vii.

Schmey, "Von der Perityphlitis (Apotyphlitis)," Reichs-Medicinal-Anzeiger, 1901, No. 23.

Schmidt, A., "Ueber Hydrobilirubinbildung im Organismus," Verhandl. d. Cong. f. innere Med., Wiesbaden, 1895.

Schmidt, A., "Experimentelle und klin. Untersuchungen über Functionsprüfung des Darms," Deutsch. Arch. f. klin. Med., vol. lxi.

Schmidt, A., "Ueber Schleim im Stuhlgang," Zeitschr. f. klin. Med., vol. xxxii.

Schmidt, A., "Ueber Darmgährung, Meteorismus und Blähungen," Therap. Monatsh., January, 1899.

Schmidt, A., "Beiträge zur Diätotherapie bei Magen- und Darmkrankheiten," Münch. med. Wochenschr., 1902, Nos. 6, 7.

Schmidt, A., and Strasburger, J., Die Fäces des Menschen im normalen und krankhaften Zustande, Berlin, 1901.

Schmidt, B., "Die Unterleibsbrüche," Pitha-Billroth's Handb. d. allgem. u. spec. Chir., Stuttgart, 1882, vol. iii.

Schmidt, Meinhardt, "Meckel'sches Divertikel und Ileus," Deutsch. Zeitschr. f. Chir., vol. liv.

Schmidt, Rudolf, "Beitrag zur Lymphosarkomatosis des Dünndarms," Wien. klin. Wochenschr., 1898, No. 21.

Schmitt, "Ueber Verletzung des Unterleibs durch stumpfe Gewalt," Münch. med. Wochenschr., 1898, Nos. 28, 29.

Schnitzler, J., "Ueber Darmblutungen nach Reposition incarcerirter Hernien," Internat. klin. Rundschau, 1894, No. 14.

Schnitzler, J., "Ueber mesenteriale Darmincarceration," Wien. klin. Rundschau, 1895, No. 37.

Schnitzler, J., "Zur Symptomatologie des Darmarterienverschlusses," Wien. med. Wochenschr., 1901, Nos. 11, 12.

Schnitzler, J., "Phlegmone des Wurmfortsatzes im Gefolge von Angina," Mittheil. d. Gesellschaft f. innere Med. in Wien, first year, 1902, No. 8.

Schnitzler, J., "Ueber die Verwerthung der mikroskopischen Blutuntersuchung zur Diagnostik und Indicationstellung bei intraabdominalen Eiterungen," Wien. klin. Rundschau, 1902, Nos. 10, 11.

Schnitzler, J., "Ueber Epiploitis im Anschluss an Operationen," ibid., 1900, Nos. 1–3.

Schnitzler and Ewald, "Zur Kenntniss der peritonealen Resorption," Deutsch. Zeitschr. f. Chir.

Schöning, ibid., 1885, vol. xxii., p. 36.

Scholz, "Ein Fall von Magencolonfistel," Wien. med. Blätter, 1896, No. 16.

Schorlemmer, "Ueber den Nachweis von Gallenfarbstoff in den Fäces," Arch. f. Verdauungskrankh., vol. vi., p. 263.

Schottmueller, "Epityphlitis traumatica," Grenzgeb. d. Med. u. Chir., vol. vi.

Schuberg, "Beiträge zur Kenntniss der Kothsteine," Virchow's Arch., vol. xc.

Schüle, "Duodenalstenose durch einen Gallenstein," Berlin. klin. Wochenschr., 1894, No. 45.

Schüller, "Gallensteine als Ursache der Darmobstruction," Inaug. Diss., Strassburg i. E., 1891.

Schüppel, "Die Krankheiten der Leber," v. Ziemssen's Handb. d. spec. Pathol. u. Therap., Leipsic, 1880, vol. viii.

Schuetz, "Schleimkolik und membranoser Dickdarmkatarrh," Münch. med. Wochenschr., 1900, No. 17.

Schutz, R., "Ueber chronische dyspeptische Diarrhöen und ihre Behandlung," Samml. klin. Vorträge; edit. by v. Bergmann, Müller, v. Winckel, 1901, No. 318.

Schweiger and Dogiel, "Ueber die Peritonealhöhle, u. s. w.," Arbeiten aus der physiologischen Anstalt zu Leipsic, 1867.

Schwerdt, "Enteroptose und intraabdominaler Druck," Deutsch. med. Wochenschr., 1896.

Sée, G., Bulletin de l'académie, Paris, 1893, No. 51.

Seiler, "Ascites im kindlichen Alter," Berlin. klin. Wochenschr., 1881, No. 26.

Seitz, J., "Pilze und Pilzgifte im Hirn und Rückenmark," Virchow's Arch., vol. cl.

Senator, "Ascites chylosus," Charité-Annalen, Berlin, 1885, vol. x., and 1895, vol. xx.

Senator, Die Albuminurie, second edition, Berlin, 1890.

Senator, "Hydrothionämie and über Selbstinfection durch abnorme Verdauungsvorgänge," Berlin. klin. Wochenschr., 1868, No. 24.

Senator, "Nierenkrankheiten," this handbook, vol. xix., p. 21. In vol. on Diseases of the Kidneys. English edition.

Siegel, "Die Appendicitis und ihre Complicationen," Mittheil. aus den Grenzgeb. d. Med. u. Chir., 1896, vol. i.

Siegel, "Ueber das primäre Sarkom des Dünndarms," Berlin. klin. Wochenschr., 1899, No. 35.

Siegel, Beiträge z. klin. Chir., vol. xxi.

Siegert, "Ueber die Zuckergussleber (Curschmann) und die pericarditische Pseudolebercirrhose (Pick)," Virchow's Arch., vol. cliii.

Sieur, "De l'intervention chirurgicale dans les contusions graves de l'abdomen," Arch. gén. de méd., 1893.

Silberschmidt, "Experimentelle Untersuchungen über die bei der Entstehung der Perforationsperitonitis wirksamen Factoren des Darminhaltes," Mittheil. aus Klin., etc., der Schweiz. 1894, series I., No. 5.

Singer, Aetiologie und Klinik des acuten Gelenkrheumatismus, Vienna, 1898.

Singer, "Transitorische Albuminurie bei acutem Darmkatarrh," Prager med. Wochenschr., 1887, No. 2.

Siredey, Union médicale, 1869.

Sklodowski, "Ueber chronische Verengerung des Dünndarms," Grenzgeb. der Med. u. Chir., vol. v.

Smidt, "Fall von chylösem Ascites," Zeitschr. f. klin. Med., vol. ii.

Smoler, "Zur Kenntniss der primären Darmsarkome," Prager med. Wochenschr., 1898, Nos. 13, 14.

Sonnenburg, "Verbrennungen und Erfrierungen," Deutsch. Chir. von Billroth und Lücke, 1879, fourteenth edition.

Sonnenburg, Pathologie und Therapie der Perityphlitis, Leipsic, 1900, fourth edition.

Sprengel, "Versuch einer Sammelforschung zur Frage der Frühoperation bei acuter Appendicitis und persönliche Erfahrungen," Arch. f. klin. Chir., vol. lxviii.

Stadelmann, "Natur der Fettkrystalle in den Fäces," Deutsch. Arch. f. klin. Med., 1887, vol. xl.

Staehelin, "Ueber den durch Essigsäure fällbaren Eiweisskörper der Exsudate und des Urins, ' Münch. med. Wochenschr., 1902, No. 34.

Starling and Tobby, Jour. Physiol., vol. xvi (see also Bayliss and Starling).

Stein, E., "Darmblutungen bei Lebercirrhose," Arch. f. Verdauungskrankh., 1899, vol. v.

Steinach, "Ueber die motorische Innervation des Darmtractus," Lotos, 1893, N. F., vol. xiv.

Stern, Ueber traumatische Entstehung innerer Krankheiten, Jena, 1900.

Stern, S., "Ueber operative Behandlung des durch Hepatitis interstitialis bedingten Ascites," ref. in Arch. f. Verdauungskrankh., vol. vii.

Still, Brit. Med. Jour., April 15, 1899.

Stiller, "Ueber chronische Peritonitis," Deutsch. Arch. f. klin. Med., 1875, vol. xvi.

Stiller, Wien. med. Wochenschr., 1880, Nos. 18, 19; also the same for 1901, No. 6.

Stiller, "Die stigmatische Bedeutung der Costa X. fluctuans," Arch. f. Verdauungskrankh., vols. ii. and vii.

Stilling, "Ein Fall von Neuritis der Nn. splanchnici," Deutsch. Arch. f. klin. Med., vol. lxxiii.

Stooss, F., "Die Pneumokokkenperitonitis im Kindesalter," Jahrb. f. Kinderheilk., N. F., vol. lvi.

Strasburger, J., "Die Sarkome des Dickdarms," Inaug. Diss., Bonn, 1894.

Strasburger, J., see Schmidt and Strasburger.

Straus, "Sur un cas d'ascite chyleuse," Arch. de physiol. normale et pathol., third series, 1886, vol. vii.

Strauss, "Hysteria virilis unter dem Bilde der chronischen Darmstenose," Berlin. klin. Wochenschr., 1898, No. 38.

Struppler, "Diagnose der Zwerchfellshernie," Deutsch. Arch. f. klin. Med., vol. lxx., p. 1.

Stschelogeff, "Recherches expérimentales sur l'influence de la laparotomie sur la péritonite tuberculeuse," Arch. de méd. expér., etc., vol. vi.

Stuparich, "Zur physiologischen und pathologischen Anatomie des Wurmfortsatzes," Wien. med. Presse, 1899, No. 35.

Sucksdorff, Arch. f. Hyg., 1885, vol. iv.

Sudsuki, "Beiträge zur normalen und pathologischen Anatomie des Wurmfortsatzes," Grenzgeb. d. Med. u. Chir., vol. vii.

Sutherland, "Appendicitis and Rheumatism," Lancet, August 24, 1895.

M'Sviney, "Rupture of Biliary Duct," Dublin Jour. Med. Sci., November, 1866.

Szydlowski, "Beiträge zur Mikroskopie der Fäces," Inaug. Diss., Breslau, 1879.

T

Tacke, "Ueber die Bedeutung der brennbaren Gase im thierischen Organismus," Inaug. Diss., Berlin, 1884.

Talamon, Appendicite et Perityphlite, Paris, 1892.

Talma, "Zur Kenntniss des Leidens des Bauchsympathicus," Deutsch. Arch. f. klin. Med., vol. xlix.

Talma, "Untersuchungen über Ulcus ventriculi simplex, Gastromalacie und Ileus," Zeitschr. f. klin. Med., 1890, vol. xvii.

Talma, "Zur Kenntniss der Tympanitis," Berlin. klin. Wochenschr., 1886, No. 23, and for 1902, No. 5.

Talma, "Chirurgische Oeffnung neuer Seitenbahnen für das Blut der Vena portae," ibid., 1898, No. 38.

Tavel and Lanz, "Ueber die Aetiologie der Peritonitis," Mittheil. aus Klin. u. In-ti-
tuten der Schweiz, Basel, 1893.
Teleky, "Die Bauchfelltuberculose und ihre Behandlung," Centralbl. f. d. Grenzgeb.
d. Med. u. Chir., 1899.
Terillon, "Lipomes en mesentère," Arch. gén. de méd., 1886.
Thierfelder, "Die Krankheiten der Leber," v. Ziemssen's Handb. d. spec. Pathol. u.
Therap., Leipsic, 1880, vol. viii.
Thiersch, "Zerreissung eines grossen Gallenganges," Berlin. klin. Wochenschr., 1879,
No. 23.
Thoma, "Vier Fälle von Hernia diaphragmatica," Virchow's Arch., vol. lxxxviii.
Thomayer, "Beitrag zur Diagnose der tuberculösen and carcinomatösen Erkrankungen
des Bauchfells," Zeitschr. f. klin. Med., 1887, vol. vii.
Tietze, "Die chirurgische Behandlung der acuten Peritonitis," Grenzgeb. d. Med. u.
Chir., vol. v.
Tilmann, "Ueber die chirurgische Behandlung des Ascites," Deutsch. med. Wochen-
schr., 1899, No. 18.
Tournier, "De la périgastrite antérieure suppurée dans le cancer de l'estomac," Lyon
med., 1897.
Traube, Die Symptome der Krankheiten des Respirations- und Circulationsapparates,
Berlin, 1867.
Traube, "Zwei Fälle von Perforationsperitonitis," Gesammelte Beiträge, Berlin, 1871,
vol. ii., p. 776.
Traube, "Ueber den Einfluss starker und anhaltender Diarrhöen auf die Gestaltung
peritonitischer Adhäsionen," Gesammelte Beiträge zur Physiol. u. Pathol.,
Berlin, 1878, vol. iii.
Traube, ibid., Berlin, 1871, vol. ii., p. 1031.
Treitz, Prager Vierteljahrsschr., 1859.
Treves, "On Peritonitis," Brit. Med. Jour., 1894.
Treves, Darmobstruction: Ihre Arten sowie ihre Pathologie, Diagnose und Therapie,
trans. by A. Pollack, Leipsic, 1886.
Trousseau, "Medicinische Klinik," Deutsch von P. Niemeyer, Würzburg, 1868.
Tschudnowsky, "Ein neues diagnostisches Merkmal der Darmperforation," Berlin.
klin. Wochenschr., 1869, Nos. 20, 21.
Tuffier, Semaine médicale, 1894, p. 285.
Turner, G. J., "Zur Anatomie des Blinddarms und Wurmfortsatzes," ref. in Cen-
tralbl. f. Chir., 1892, No. 41.

U

Uffelmann, "Verhalten der Fäces natürlich ernährter Säuglinge," Deutsch. Arch. f.
klin. Med., 1881, vol. xxviii.
Uhde, "Chirurgische Behandlung innerer Einklemmungen," Pitha-Billroth's Handb.
d. allge. u. spec. Chir., Stuttgart, 1882, vol. iii.
Umber, "Ueber autolytische Vorgänge in Exsudaten," Münch. med. Wochenschr.,
1902, No. 28.

V

Valenta v. Marchthurn, "Weitere neunzehn mittelst Laparotomie behandelte Fälle
von Bauchfelltuberculose," Wien. klin. Wochenschr., 1897, No. 9.
Valentiner, Die Hysterie und ihre Heilung, Erlangen, 1852.
Vanni, "L'enteria muco-membranosa," Rivista clinica, 1888, No. 4.
Varvilevsky, "Études anatomo-pathologiques sur l'influence de la laparotomie sur la
péritonite tuberculeuse," Arch. d. sci. biol., St. Petersburg, vol. iv.
Veillon and Zuber, "Recherches sur quelques microbes strictement anérobes et leur
rôle en pathologie," Arch. de méd. expér., 1898.
Vierordt, H., Die einfache chronische Exsudativ-Peritonitis, Tübingen, 1884.
Vierordt, O., "Weitere Beiträge zur Kenntniss der chronischen, insbesondere tubercu-
lösen Peritonitis," Deutsch. Arch. f. klin. Med., 1893, vol. lii.
Vierordt, O., "Ueber die Tuberculose der serösen Häute," Zeitschr. f. klin. Med.,
1888, vol. xiii.
Vierordt, O., "Ueber die Peritonealtuberculose," Deutsch. Arch. f. klin. Med., 1890,
vol. xlvi.
Virchow, "Ueber katarrhalische Geschwüre," Berlin. klin. Wochenschr., 1893, Nos.
8, 9.
Virchow, "Ueber die Reform der pathologischen und therapeutischen Anschauungen
durch die mikroskopischen Untersuchungen," Virchow's Arch., vol. i., p. 249.

Virchow, " Ueber Epithelialdesquamation im Darm," Virchow's Arch., vol. xc.
Virchow, "Historisches, Kritisches und Positives zur Lehre der Unterleibsaffectionen," ibid., 1853, vol. v.
Virchow, Gesammelte Abhandlungen, Hamm, 1862.
Virchow, " Ueber den Gang der amyloiden Degeneration," Virchow's Arch., vol. viii.
Virchow, " Neue Beobachtungen über amyloide Degeneration," ibid., vol. xi.
Vötsch, Die Koprostase, Erlangen, 1874.
Volkmann, " Ueber den Mastdarmkrebs," Volkmann's Samml. klin. Vorträge, No. 131.
Volz, Ad., Die durch Kothsteine bedingte Perforation des Wurmfortsatzes, etc., Carlsruhe, 1846.
de Vries, " Ueber das Indican im Harn und seine diagnostische Bedeutung," Inaug. Diss., Kiel, 1877.

W

Wagner, E., Arch. f. physiol. Heilk., 1858.
Wagner, " Zur Diagnostik und Therapie der perforativen Peritonitis," Deutsch. Arch. f. klin. Med., vol. xxxix.
Wagner, Arch. d. Heilk., 1861, No. 2.
Wagner, " Beiträge zur Pathologie und pathologischen Anatomie der Leber," Deutsch. Arch. f. klin. Med., vol. xxxiv.
v. Wahl, " Die Laparotomie bei Achsendrehungen des Dünndarms," Langenbeck's Arch., vol. xxxviii.
v. Wahl, " Ueber die klinische Diagnose der Darmocclusion durch Strangulation und Achsendrehungen," Centralbl. f. Chir., 1889, No. 9.
Walbaum, " Zur Histologie der acuten eitrigen Peritonitis," Virchow's Arch., vol. clxii.
Waldeyer, " Grosses Lipo-Myxom des Mesenteriums," ibid., vol. xxxii.
Waldeyer, " Die Entwicklung der Carcinome," ibid., vol. lv.
Wallerstein, " Ueber Cylindrurie und Albuminurie bei künstlich erzeugter Koprostase," Berlin. klin. Wochenschr., 1901, No. 21.
Wallmann, " Divertikelbildung," Virchow's Arch., vol. xiv.
Walter, " Ein Fall von Enteritis membranacea," Inaug. Diss., Halle, 1885.
Walthard, " Experimenteller Beitrag zur Kenntniss der Aetiologie der eitrigen Peritonitis nach Laparotomie," Arch. f. exper. Pathol., 1891.
Waterhouse, "Experimentelle Untersuchungen über Peritonitis," Virchow's Arch., vol. cxix.
Wechsberg, F., " Seltener Ausgang einer Darminvagination," Centralbl. f. allgem. Pathol. u. pathol. Anat., 1900, vol. xi., Nos. 6, 7.
Wechsberg, L., " Ueber einen Fall von Achsendrehung des Dünndarms," Prager Zeitschr. f. Heilk., 1902, vol. xxiii.
Wegele, Die diätetische Behandlung der Magen-Darmerkrankungen, Jena, 1896.
Wegeler, Jour. de méd., 1813, vol. xxviii.
Wegner, G., " Chirurgische Bemerkungen über die Peritonealhöhle, etc.," Langenbeck's Arch. f. klin. Chir., 1876, vol. xx.
Weichselbaum, " Eine seltene Geschwulstform des Mesenteriums," Virchow's Arch., vol. lxiv.
Weigert, " Ueber die Wege des Tuberkelgiftes zu den serösen Häuten," Deutsch. med. Wochenschr., 1883, Nos. 31, 32.
Weigert, " Onkologische Beiträge," Virchow's Arch., vol. lxvii., p. 513.
Weir, R. F., series of abstracts in New York Med. Rec., Med. News, Annals of Surgery, etc., for the years 1887-92.
Weir, New York Med. Rec., 1900, vol. lvii.
Weisker, " Bemerkungen über den sog. intraabdominalen Druck," Schmidt's Jahrbücher, 1888, vol. ccxix.
Weiss, Siegfried, " Intussusceptio intestini," summary in Centralbl. f. d. Grenzgeb. d. Med. u. Chir., 1899, vol. ii.
Werber, Berichte der Naturforscher-Gesellschaft zu Freiburg i. Br., Freiburg i. Br., 1865, vol. iii., No. 3.
Wernich, Verhandl. d. Vereines f. innere Med. in Berlin, 1882.
Werth, " Pseudomyxoma peritonei," Arch. f. Gynäk., vol. xxiv.
Werth, " Bemerkungen zur Laparotomie," Verhandl. der 71. deutsch. Naturforschervers. in München, 1899, Leipsic, 1900.
Wertheim, " Die ascendirende Gonorrhoe beim Weibe, etc.," Arch. f. Gynäk., 1892, vol. xlii.

Wesener, Kritische und experimentelle Beiträge zur Lehre der Fütterungstuberculose, Freiburg, 1885.
Westphalen, "Ueber die chronische Obstipation," Arch. f. Verdauungskrankh., vols. vi., vii.
Westphalen, "Ueber die sog. Enteritis membranacea," Berlin. klin. Wochenschr., 1901, Nos. 14–16.
White, Hale, "On Simple Ulcerative Colitis," Guy's Hosp. Rep., 1890, vol. xxx.
Whitties, Trans. Assoc. Amer. Phys., 1889.
Wick, "Ein Fall von nervöser Enteritis," Wien. med. Wochenschr., 1898, No. 40.
Wickhoff, "Ein Beitrag zur Gallenblasenchirurgie," Wien. klin. Wochenschr., 1893, No. 3.
Widenmann, "Zur Casuistik der Zwerchfellshernien beim Lebenden," Berlin. klin. Wochenschr., 1901, No. 11.
Wiel, Diätetisches Kochbuch für Gesunde und Kranke, seventh edition, Freiburg i. Br., 1895.
Wieland, "Experimentelle Untersuchungen über die Enstehung der circumscripten und diffusen Peritonitis," Mittheil. aus Klinik, etc., der Schweiz, 1895, second series, No. 7.
Willard, Phila. Med. Times, May 5, 1883.
Wilson, Ezra, On Appendicitis, by G. R. Fowler.
v. Winckel, Lehrbuch der Geburtshülfe, Leipsic, 1889.
v. Winckel, "Behandlung der von den weiblichen Genitalien ausgehenden Entzündungen des Bauchfells, etc.," Penzoldt-Stintzing's Handb. d. spec. Therap. innerer Krankh., Jena, 1897.
Windscheid, "Drei Fälle von Pericolitis," Deutsch. Arch. f. klin. Med., 1890, vol. xlv.
Witzel, "Beiträge zur Chirurgie der Bauchorgane," Deutsch. Zeitschr. f. Chir., vol. xxi.
Wölfler, Langenbeck's Arch., vol. xxi.
Wolbrecht, Ueber Pleuracomplicationen bei Typhlitis und Perityphlitis, Berlin, 1891.
Wolff, Alfred, "Transsudate und Exsudate, ihre Morphologie und Unterscheidung," Zeitschr. f. klin. Med., vol. xlii.
Wolkow and Delitzin, Die Wanderniere, Berlin, 1899.
Woodward, Med. and Surg. History of the War of Rebellion, vol. i., part ii.

Z

Zeidler, "Beiträge zur Pathologie und Therapie des acuten Darmverschlusses," Grenzgeb. der Med. u. Chir., vol. v.
v. Zeissl, M., "Péritonite causée chez l'homme par l'urétrite blennorrhagique," Annales des mal. des organes gènito-urinaires, July, 1893.
Ziegler, Studien über die intestinale Form der Peritonitis, Munich, 1893.
v. Ziemssen, Verhandl. d. VIII. Cong. f. innere Med., Wiesbaden, 1889.
v. Ziemssen, "Künstliche Gasaufblähung des Dickdarms," Deutsch. Arch. f. klin. Med., vol. xxxiii.
v. Ziemssen, Deutsch. med. Wochenschr., 1887, No. 35.
Zillner, "Ruptura flexurae sigmoideae neonati intra partum," Virchow's Arch., vol. xcvi.
Zöge von Manteuffel, Verhandl. d. Cong. f. innere Med., 1889.
Zöge von Manteuffel, "Volvulus Cœci," Samml. klin. Vorträge, 1899, N. F., No. 260.
Zuckerkandl, E., Ueber die Obliteration des Wurmfortsatzes beim Menschen, Wiesbaden, 1894.
Zunker, Deutsch. Zeitschr. f. prak. Med., 1878, No. 1.
Zuntz, Verhandlungen des Vereines für innere Medicin zu Berlin, 1884.
Zuppinger, "Der Darmkrebs im Kindesalter," Wien. klin. Wochenschr., 1900, No. 17.
Zweig, "Beiträge zur functionellen Diagnostik der Darmkrankheiten" Wien. klin. Rundschau, 1901, No. 41.

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